DISEASES OF THE NERVOUS SYSTEM

MODERN CLINICAL MEDICINE

DISEASES
OF THE
NERVOUS SYSTEM

EDITED BY

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EDITOR'S PREFACE

EVERY practitioner must have been frequently impressed with the inadequacy of the usual text book description of the various diseases with which he is brought into contact. Even books of reference do not always give the scope to special topics that are requisite to place the physician in a satisfactory position relative to any particular disease he may be called upon to treat. The articles on nervous diseases embraced within this volume meet this actual want in a manner so admirable that it is a satisfaction to have been associated with their production in English. To the beginning practitioner to some extent they supply the place of personal experience, and even to the specialist they furnish a most admirable résumé and guide.

The various subjects having been assigned to men of known experience, with large opportunities for their study, insured a presentation that is essentially practical and embraces the value of an authoritative personality. The manner in which the subject matter is presented also appeals to the medical reader, and while the statements are carefully made they are marshalled in an interesting and instructive fashion.

One is struck throughout the book with the fact that while Germany is supposed to be the land of therapeutic nihilism these various authors have found therapeutics of real help and afford the reader the benefit of their experience and judgment in this important branch of practice.

At first the editorial work was taken up with the expectation of making numerous addenda to the text, but when the proper scope of the work was realized it was deemed expedient to let each author stand responsible for the subject matter which he had personally presented and not to confuse the medical reader by discussions of disputed and unessential features.

ARCHIBALD CHURCH.

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The Macroscopic Anatomy of the Central Nervous System, with Special Reference to the Physiology of the Brain. By M. ROTHMANN, Berlin.

Normal and Pathological Histology of the Central Nervous System, with Special Reference to the Neuron Theory. By H. ROSIN, Berlin.

General Neurological Diagnosis, Including the Examination of Patients with Diseases of the Nervous System. By P. SCHUSTER, Berlin.

Modern Aids in the Diagnosis of Diseases of the Brain. By E. REDLICH, Vienna.

Lumbar Puncture. By H. QUINCKE, Kiel.

Cerebral Hemorrhage and Embolism. By R. GEIGEL, Würzburg.

The Symptom-Complex of Aphasia. By C. WERNICKE, Breslau.

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THE MACROSCOPIC ANATOMY OF THE CENTRAL NERVOUS SYSTEM

WITH SPECIAL REFERENCE TO THE PHYSIOLOGY OF THE BRAIN

By M. ROTHMANN, Berlin

At the beginning of the twentieth century the study of the macroscopic anatomy of the central nervous system is most necessary. As in many other departments of science which are apparently devoid of clinical interest, here also in the victorious progress of clinical research the necessity has arisen to reveal to the clinician such exact knowledge of the anatomical relations of the central nervous system as in preceding decades only the specialist in this branch of medicine was expected to acquire. Not merely because the enormous advance in the study of the physiology and pathology of the brain and spinal cord now enable us almost daily to localize exactly the manifold affections of the central nervous system, but surgery has very recently found its realm greatly enlarged in that it now searches for an abscess deep within the brain or locates a tumor of the brain or spinal cord by methods of diagnosis which are constantly becoming more certain; it successfully removes these, or even a cicatrix upon the surface of the brain which has been the cause of severe epileptic convulsions. In all of these diagnoses and operations accurate knowledge of the individual parts of the central nervous system, their blood supply, their position in the skull and vertebral column, is absolutely necessary. In addition, one of the greatest contributions to our clinical armamentarium, Quincke's method of *lumbar puncture,* the importance of which is constantly proven, in its various modifications can be practically utilized only when we fully understand the position of the spinal cord and its membranes within the canal of the vertebral column.

If the relations of the fully developed, human, central nervous system are to be correctly appreciated, its **embryologic structure** must be understood and the simpler forms in lower animals must be compared with these. The central nervous system is developed from the *ectoderm.* The *medullary plates* arising from the originally single-layered epithelium along the median line of the body grow toward each other, then close and form the embryonic *medullary tube,* which dorsally for some time reveals a space. Separated from this *embryonic medullary tube* is a ganglion crest, from which the ganglia of the head and spine arise, and a sympathetic layer, both of which subsequently develop spontaneously. In the lowest vertebrate animals known to us, the amphioxus, in which the brain is merely indicated by a slight thickening at the anterior end of the embryonic medullary tube, the central nervous system remains permanently in this stage. In all other vertebrates,

however, the medullary tube at its anterior portion has two ridges which form three spaces, the primary cerebral vesicles, the *anterior middle*, and the *posterior cerebral* vesicles, the latter of which passes immediately into the spinal cord. Soon afterward the embryonic medullary tube curves in such a way that the middle cerebral vesicle takes the uppermost position in the entire central nervous system. At about the fourth week of embryonic life the anterior brain shows a new development, the *secondary anterior brain*, which subsequently becomes the *cerebrum*, in contrast to the primary anterior brain

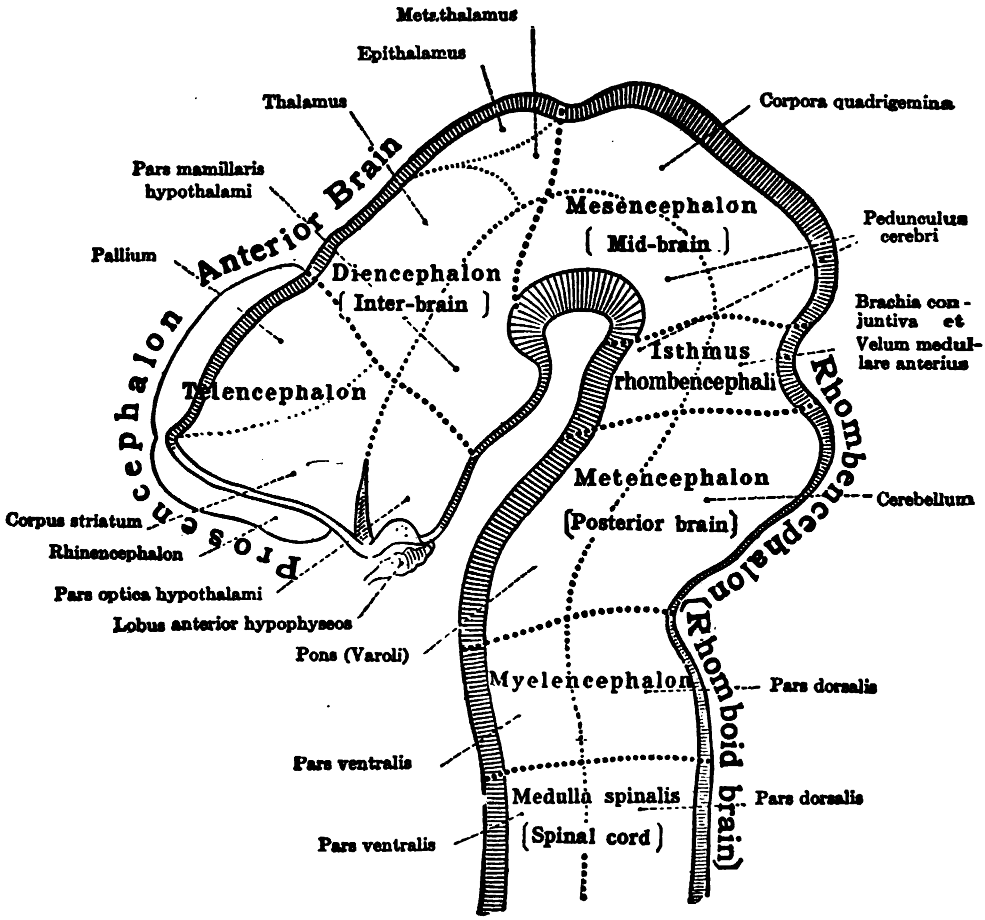

FIG. 1.—MEDIAN SAGITTAL SECTION THROUGH THE BRAIN OF A HUMAN EMBRYO AT THE END OF THE FIRST MONTH. (After W. His.)

or *inter-brain*; the posterior cerebral vesicle by curving forward, the varolian bend, is divided into the *posterior brain* and the *after-brain* (metencephalon), so that five cerebral vesicles are now present and communicate with one another, being separated from the spinal cord posteriorly by the cervical flexure.

We, therefore, differentiate the *telencephalon* or anterior brain with the olfactory lobes at the base, the corpus striatum and the *pallium* which later

extends to the hemispheres of the cerebrum, the *diencephalon* or inter-brain with the thalamus opticus, the *mesencephalon* or mid-brain with the corpora quadrigemina, the *metencephalon* or after-brain with the cerebellum and pons, and the *myelencephalon* or cerebrospinal axis with the medulla oblongata. The telencephalon—above all, the *pallium*—from which the cerebrum of the vertebrate animals is formed, is the most highly developed of these divisions, and attains its mightiest conformation in man. While in vertebrate fish the simple epithelial covering is permanent, in man this finally covers to a surprising extent the entire brain. In the telencephalon the process is the same as in all other parts of the brain, the basal section becomes very thick while the dorsal portions are thin but become very broad. Hence, in the telencephalon, the closure of the medial wall of the hemispheres does not develop nervous elements but fills out the lateral ventricles as the *choroid plexus.* The communications of the cerebral cavities with the surface of the brain, which are at first free, are arrested toward the end of the fourth month by the development of the *corpus callosum.* Through this, as well as the *fornix* which lies below it, the two hemispheres of the cerebrum expand, while the *anterior cerebral ganglia* develop from the basal portion the so-called ground plate of the *telencephalon,* from the *corpus striatum* which is divided by the projection fibers which pass from the cortex through the internal capsule into the *nucleus caudatus*, and from the *nucleus lentiformis* to which the *nucleus amygdalæ* and the *claustrum* are added. Owing to the adhesion of the corpus striatum to the thalamus opticus in the perfectly developed brain the space between the telencephalon and diencephalon is hardly noticeable.

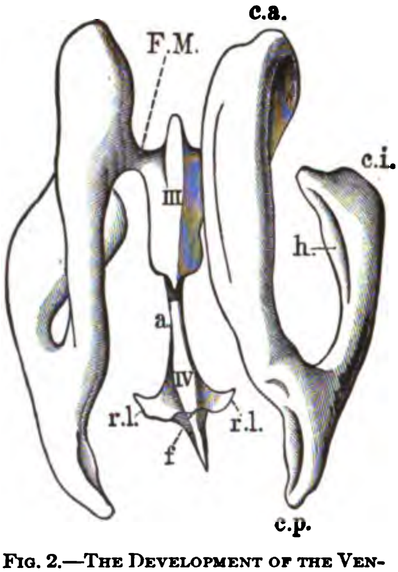

FIG. 2.—THE DEVELOPMENT OF THE VENTRICULAR SYSTEM. (After Welcker.)
IV, Fourth ventricle; *r.l.*, Recessus lateralis; *a.*, Aquæductus Sylvii; *III*, Third ventricle; *F.M.*, Foramen Monroi; *c.a.*, Cornu anterius; *c.p.*, Cornu posterius; *c.i.*, Cornu inferius of the lateral ventricles; *h.*, Pes hippocampi major.

Simultaneously with the formation of the different portions of the brain and the more or less greatly increased thickness of their walls, the original spaces in the cerebral vesicles develop into a *ventricular system* varying in extent and form. In accordance with the development of the hemispheres two ventricles form in the cerebrum; the *lateral ventricle*, which at first communicate with the surface, are subsequently shut off from it but communicate with each other by means of the *foramen Monroi,* through which also we pass to the *third ventricle* in the cavity of the diencephalon. The latter is limited laterally by the optic thalamus, and above by epithelium from the choroid plexus, while it passes downward in funnel shape into the infundibulum. Through the comparatively wide third ventricle we pass dorsally to the narrow *aqueduct of Sylvius,* the canal of the mesencephalon, the upper bound-

ary of which is formed by the corpora quadrigemina, the lateral limits by the structures beneath, the red nucleus, the lemniscus tracts, etc., while the cerebral peduncles are found upon the floor. In the region of the metencephalon these canals dilate and form the *fourth ventricle,* the so-called rhomboid fossa, the roof of which is formed by a thin membrane, the tela chorioidea posterior, above which we find the layers of the cerebrum developed from the dorsal wall of the occipital vesicles; this structure with its arms which extend to the other parts of the brain forms the lateral boundary of the fourth ventricle, while the mass of the pons and the medulla oblongata spreads out caudally toward the base. In the medulla the fourth ventricle gradually narrows and becomes the central canal of the spinal cord, and in various ways, in adult man being partly obliterated, extends through the entire length of the cord, and terminates in the conus terminalis.

After the development of the central nervous system of man in its external forms, in the adult human being the greatest interest centers in the formation of the *fossæ and convolutions* from the pallium. A few so-called "transitory fossæ" develop from the second to the third month but subsequently disappear, after which, in the fifth month, the first permanent fossa, the *Sylvian fossa,* is formed by that portion of the pallium which communicates with the corpus striatum, the island of Reil, which is slower than the remaining parts of the pallium in growth. At first there is a flat depression which, by the rapid extension of the remaining pallium, constantly becomes deeper so that at about the middle of the fifth month the anterior and posterior portion of the fossa Sylvii is well developed while the rest of the brain is still

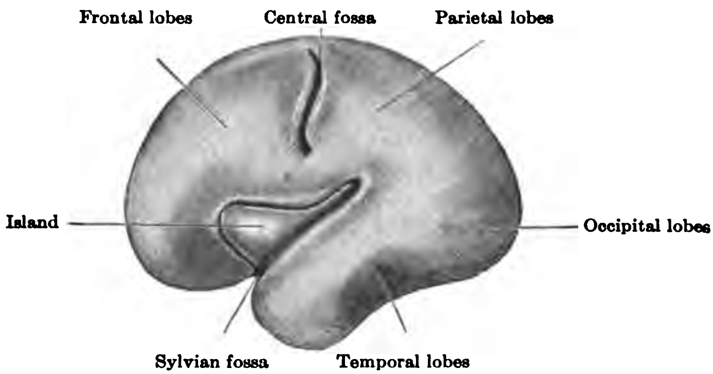

FIG. 3.—BRAIN OF A HUMAN FETUS AT THE END OF THE FIFTH MONTH.

quite smooth. At the end of the fifth month the *central fossa* is formed, and in the sixth and seventh months the remaining fossæ, which, however, are still primitive but permit the principal convolutions of the perfect brain to be distinctly recognized.

Even to-day the importance of the formation of convolutions and fossæ has not been positively determined. Although it is generally maintained that the formation of fossæ in the cerebrum occurs only in the higher animals and in these constantly undergoes greater development, and that, as we have just seen, this cerebral furrowing in the embryo of man takes place compara-

tively late, there is no absolute parallel between the mental development of the animal and the formation of fossæ in the cerebrum. We know that some animals have perfectly flat brains and that in others the brain is markedly convoluted, yet we are unable to determine a preponderance of cerebral activity in the latter. Of course, in the highest group of mammals, monkeys, from the lowest forms up to the anthropomorphic apes, there is an increasingly rich formation of fossæ and convolutions which finally reaches its acme in the human brain.

The importance of the fossæ unquestionably depends upon the greatest possible surface distribution of the cortex of the cerebrum within the limited space of the cavity of the skull. Although the ganglion cells of the cortex constantly increase in numbers by the development of the brain, and space is formed by their overlapping each other in layers, the thickness of the cortex thus being permanently increased, a juxtaposition of the nervous elements is of great importance, above all for the development of the projection fiber systems which unite the cortex of the brain with the deeper portions of this structure. Since, as Baillarger pointed out, when the brain begins to grow its volume forms the cube of its diameter while the surface equals only the square of its diameter, many convolutions must develop in the surface in order to preserve this ratio between volume and diameter.

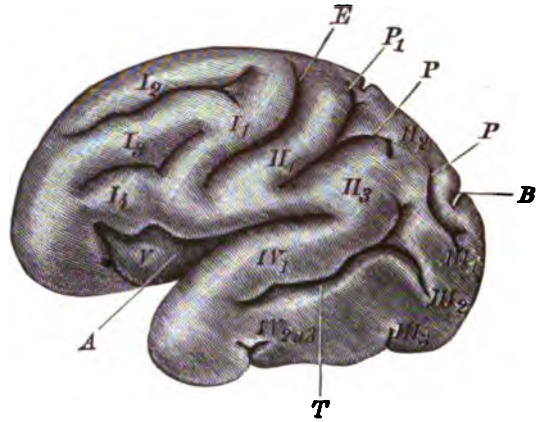

FIG. 4.—BRAIN OF A HUMAN FETUS AT SEVEN MONTHS.

A, Fiss. Sylvii; I_1, gyr. central. ant.; I_2, gyr. front. sup.; I_3, gyr. front. med.; I_4, gyr. front. inf.; II_1, gyr. central. post.; II_2, lob. parietal sup.; II_3, lob. parietal. inf.; III_1, gyr. occ. sup.; III_2, gyr. occ. med.; III_3, gyr. occ. inf.; IV_1, gyr. temp. sup.; IV_2, *u.*3, gyr. temp. med. u. inf.; *V*, insula; *B*, fiss. parieto-occip., lateral portion; *E*, sulc. centralis; *P* and *P*, anterior and posterior portions of the sulc. interparietalis; P_1, sulc. postcentralis; *T*, sulc. temporalis sup.

At this point it may be interesting to discuss the question in how far in man the *weight of the brain* and its *richness in convolutions* may presage his mental development. In beginning our investigations into the brain we incline to the general opinion that great weight of the brain is combined with a great intellect. Numerous measurements made for this purpose among various races and also measurements in the case of noted men have proven that there is for this no absolute rule, but that while the brains of some prominent men were very large, others did not exceed the average or were even below it. The fact that the brain in men is, upon the average, heavier than in women, and that in various races the weight of the brain does not markedly differ, may be attributed almost wholly to the difference in the general size and weight of the body, with which the weight of the brain appears to correspond. A little reflection will also show that the nervous elements in the cortex of the brain which mold our psychical life determine also the weight of the brain rather than the basic substance of the brain,

the amount of blood it contains, or the size of its lower parts. As the weight of the brain cannot be a gauge for the intelligence of a person, neither is the large number of fossæ and convolutions a general sign of high mental development. Whether in persons who have achieved distinction in special directions—mathematicians, musicians, orators, poets, etc.—definite areas of the brain show a marked development and an unusual richness in convolutions cannot be certainly determined because of the comparatively small number of distinguished men whose brains have been examined for this purpose. Careful researches in this direction will, no doubt, give us valuable information.

The study of the **fossæ and convolutions** of the adult brain of man was for a long time very superficial, and no great practical interest was attached to it, while physiology, according to Flourens' teachings, demonstrated that the various sections of the cortex and cerebrum were of equal importance. But, after the important discovery of cerebral localization by Fritsch and Hitzig, a triumphal progress was begun which enabled us to localize correctly pathologic foci upon the surface of the cerebrum, and even to operate in suitable cases. Then scientific interest was concentrated more and more upon the exact determination of the fossæ and convolutions. It was soon observed that these are positively and inseparably related to the localization of individual functions, and that their position in regard to each other and to the bony skull was of the greatest scientific and practical significance. Hence it appears expedient to mention here the most important points. I should like to state at once that, in the living brain, after opening the dura mater, the membrane which closely surrounds the arachnoid and pia mater presses the fossæ so closely together that no clear view into these fossæ, some of which enter deeply into the convolutions, can be obtained until after the removal of the membranes. This, however, is possible in a fresh normal brain, and still more easy after hardening it for a few days in a 10 per cent. formol solution. Then the deep furrows of the cerebrum may be seen very distinctly. While we were formally inclined to regard the convolutions as primary, and the fossæ as secondary, we have lately recognized that the fossæ undoubtedly develop first, and form convolutions only after entering deeply into the surface which was previously smooth.

The fissures may be classified as **main fissures** which first develop and are characterized by great depth, as *typical secondary fissures* which are much more superficial although they may be found in every normal brain, and as *atypical secondary fissures* which show great variations.

Among the main fissures of the brain the *Sylvian fissure* must first be mentioned, on account of its early formation and size, as well as because of the importance of the adjacent portions of the brain in which prominent centers and association tracts for the function of speech are located. These consist of the ascending *truncus* fissuræ Sylvii, the ramus posterior horizontalis which ascends from the base of the brain to the lateral surface of the hemispheres, the posterior portion passing upward as the ramus posterior ascendens, while at the anterior end two small, deep, lateral fissures appear, one, the ramus anterior horizontalis passing forward, and the other, the ramus anterior verticalis running upward. Next in importance to this is the *sulcus centralis*, the *fissure of Rolando*, around which the centers for the head and extremities are situated. This fissure runs upon the lateral sur-

face obliquely forward and downward from the middle of the median border of the hemispheres, usually making two bends (the superior and inferior genu of the sulcus centralis) and then runs downward terminating a little above the horizontal limb of the Sylvian fissure. The two principal fissures are chiefly located upon the median surface of the occipital lobe, and are the *fissura parieto-occipitalis* which usually extends somewhat upon the lateral surface, and the *fissura calcarina,* these uniting at an acute angle and terminating immediately below the splenium of the corpus callosum, in the surroundings of which in the cortex, the sense of sight in man appears to be situated. The *sulcus corporis callosi,* sometimes also known as the fissura hippocampi, and the *fissura chorioidea,* which is scarcely perceptible in the developed brain, are principal fissures in the embryo but do not proceed to complete development.

To facilitate its description the division of the hemispheres of the cerebrum into separate lobes formed as far as possible by the main fissures is

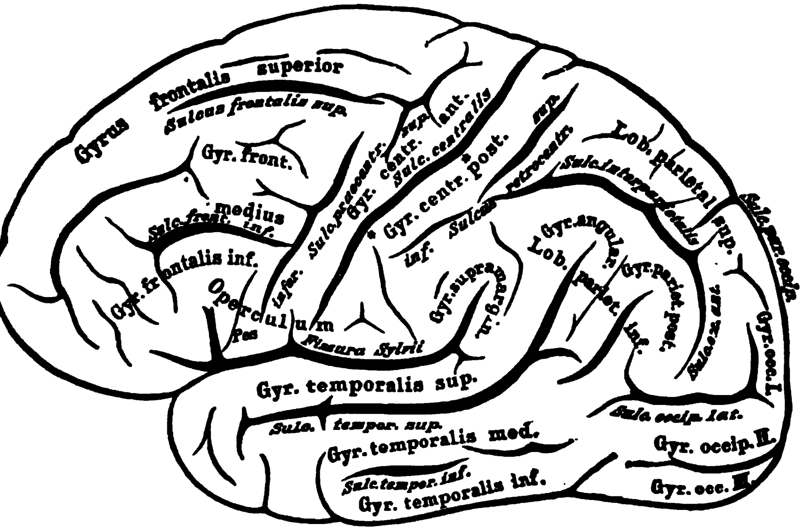

FIG. 5.—LATERAL VIEW OF THE BRAIN. (After Edinger.)

important. Hence, we differentiate a *frontal lobe* situated in front of the central fissure and extending downward to the Sylvian fissure, also a *parietal lobe* which begins behind the central fissure and is also limited below by the Sylvian fissure. The boundaries of the *temporal* and *occipital lobes* are not so distinct, but by the aid of the fissura parieto-occipitalis and the Sylvian fissure, which must be extended, these may be determined. The island situated at the base of the Sylvian fossa is to be regarded as a special portion of the brain.

The *frontal lobe,* which in no animal known to us, not even the anthropoid apes, attains the development that it does in man, has three surfaces, lateral, median, and basal. The last is adjacent to the osseous roof of the orbit, and is therefore called the orbital surface. On the lateral surface we invariably differentiate three fissures, the *sulcus præcentralis* which lies parallel with the sulcus centralis and runs in front of it, often bifurcating into two parts,

the *sulcus frontalis superior and sulcus frontalis inferior*, both of which run anteriorly from the sulcus præcentralis toward the median border of the hemisphere. The many smaller fissures, some of which are not invariably found, will not be enumerated. These fissures form four convolutions: (1) The *gyrus centralis anterior*, lying between the central fossæ, the sulcus præcentralis, and the Sylvian fissure; (2) The *gyrus frontalis superior* (first frontal convolution) between the sulcus præcentralis, sulcus frontalis superior, and median border of the hemisphere, and extending above the latter to the median surface of the hemisphere; (3) The *gyrus frontalis medius* between the upper and lower frontal fissures; (4) The *gyrus frontalis inferior*, known as the third or Broca's frontal convolution, and so designated by Broca, but previously described by Gall; Broca, however, first definitely recognized it as the seat of the motor speech function. This is divided by the ramus anterior verticalis and the ramus anterior horizontalis of the Sylvian fossa into the pars opercularis, the pars triangularis, and the pars orbitalis; the latter extends to the orbital surface of the frontal lobe, as also the two other frontal convolutions. Here the inconstant fissures, such as the *sulcus cruciatus orbitalis* and, extending medially from it, the *sulcus olfactorius*, are included; the latter receives the tractus olfactorius.

The *parietal lobe* with its lateral and medial surfaces has but one typical fissure, the *sulcus interparietalis*, which rises immediately above and parallel with the Sylvian fossa, behind the sulcus centralis, and forms an arch behind the occipital lobes. A small branch of this sulcus, which extends upward to a greater extent than and parallel with the central fissure, is called the *sulcus retrocentralis*. Here are formed the following convolutions: (1) The *gyrus centralis posterior* behind the central fissure; above this and upon the median surface of the hemisphere it enters the *paracentral lobe* and unites with the gyrus centralis anterior; below it enters the central fossa and joins the *operculum* above the Sylvian fossa. This convolution is much narrower than the anterior central, from which, according to the investigations of Ramón y Cajal, it differs decidedly in its finer structure; (2) the *gyrus parietalis superior* (the upper parietal convolution) behind the gyrus centralis posterior and above the sulcus interparietalis, with the *precuneus* at the medial surface of the hemisphere; (3) the *gyrus parietalis inferior* (lower parietal lobe) is divided below the sulcus interparietalis into gyrii, the *gyrus supramarginalis* and the *gyrus angularis*, of which the former surrounds the posterior end of the Sylvian fossa, while the *pli courbe*, much discussed in scientific combats as the gyrus angularis and the cortical center of sight, surrounds the posterior portion of the sulcus temporalis superior, and is therefore not sharply separated from the occipital lobes.

The *occipital lobe* has the form of a triangle with its base upon the parietal and temporal lobes while its apex points backward; it is called the *occipital pole*. Corresponding to this is a *frontal pole* upon the most anterior portion of the frontal lobe. Fossæ are not always found on the lateral surface of the occipital lobe. As a rule, the main furrows are the *sulcus occipitalis transversus* which is situated transversely behind the interparietal fissure and runs through that area in which we find in monkeys a deep space separating the posterior and parietal lobes, the *sulcus occipitalis lateralis*, situated in the lower part of the occipital lobe and running close to the posterior pole, and a

sulcus occipitalis anterior in the anterior portion of the occipital lobe near the extension of the posterior ascending part of the sulcus temporalis medius. The difference in these furrows accounts for the varied formation of three convolutions upon the lateral surface, which like the three frontal convolutions extend from before backward as an *upper, middle* and *lower gyrus occipitalis.* Each of these convolutions passes forward uninterruptedly into another convolution, the upper into the gyrus parietalis superior, the middle into the gyrus angularis, and the lower into the middle temporal convolution.

Before describing the median division of the occipital lobe, we must first consider the *temporal lobe* which occupies the lowest part of the cerebral hemisphere, and is limited anteriorly and above by the Sylvian fissure, while posteriorly it passes into the parietal and occipital lobes without a sharply defined limitation. Here, following each other from above downward and running parallel with the Sylvian fossa, are the *sulcus temporalis superior,* a part of which ends with the gyrus angularis, and which has also an anterior horizontal and a posterior ascending portion, the *sulcus temporalis medius,* a less developed one, the *sulcus temporalis inferior* which extends along the lower surface of the hemisphere, and the *sulcus occipito-temporalis inferior.* Here are also three convolutions upon the lateral surface, the *gyrus temporalis superior* which forms the lower boundary of the Sylvian fossa and upon whose surface externally a few invisible gyrii temporalis transversi extend to the island, a *gyrus temporalis medius and inferior,* the latter partly upon the lower surface of the hemisphere, and wholly upon the latter the *gyrus occipito-temporalis lateralis* or *gyrus fusiformis,* lying between the sulcus temporalis inferior and the occipito-temporalis inferior which to a great extent forms posteriorly the basal surface of the occipital lobe; finally, the *gyrus occipito-temporalis medialis* or *gyrus lingualis* which is situated between the sulcus occipito-temporalis inferior and the fissura calcarina previously described as one of the main fissures on the median and lower surface of the hemisphere, and which lies almost entirely within the occipital lobe. Anteriorly it passes into the gyrus hippocampi which will shortly be described.

In viewing that portion of the *medial surface of the hemisphere* which has not yet been considered we see the *gyrus fornicatus* passing from before backward around the arc-like corpus callosum. The upper portion, situated upon the corpus callosum, is called the *gyrus cinguli,* the lower, bordering upon the temporal lobe, the *gyrus hippocampi*; these are united upon the posterior portion of the corpus callosum by the narrow *isthmus*—gyri fornicati. The anterior portion of the gyrus hippocampi shows a marked thickening and curvature, the *uncus,* which must be considered as being within the anterior portion of the temporal lobe, and which is analogous to a process in the interior of the ventricle, the *cornu ammonis.* Corresponding to these convolutions, and above the corpus callosum, we note the *sulcus corporis callosi* as the upper limit of the gyrus hippocampi, the *fissura hippocampi,* which joins the *fascia dentata* and the *nervi Lancisii* in man, apparently being embryonic convolutions which pass into the lower horn of the lateral ventricles. An important fossa is the *sulcus calloso-marginalis* or sulcus cinguli, which extends as an arch above the cingulum posteriorly from the genu of the corpus callosum, and passes upward over the splenium of the corpus callosum and immediately behind the central fossa into the lateral surface of the hemi-

sphere. Anterior to this is a secondary fossa of this sulcus extending vertically; above it is the *sulcus paracentralis.*

Among the anterior convolutions on the median surface of the hemisphere we find the *gyrus frontalis superior* and a few of its subdivisions, then the one mentioned above, the lobulus paracentralis, which corresponds to the central convolutions between the sulcus paracentralis and the ascending portion of the sulcus calloso-marginalis behind the *precuneus,* it is of a square form, and reaches posteriorly to the parieto-occipital fissure. This is joined dorsally by the triangular *cuneus* between the last mentioned furrow and the fissura calcarina, the anterior point of which runs as the pedicle of the cuneus to the isthmus gyri fornicati; finally we observe the *gyrus descendens* which passes downward behind the fissura calcarina to the *gyrus lingualis,* which was described when outlining the temporal lobe.

A brief description of the *island* must be given. The *island of Reil* is covered by the surrounding portions of the brain, and externally immediately

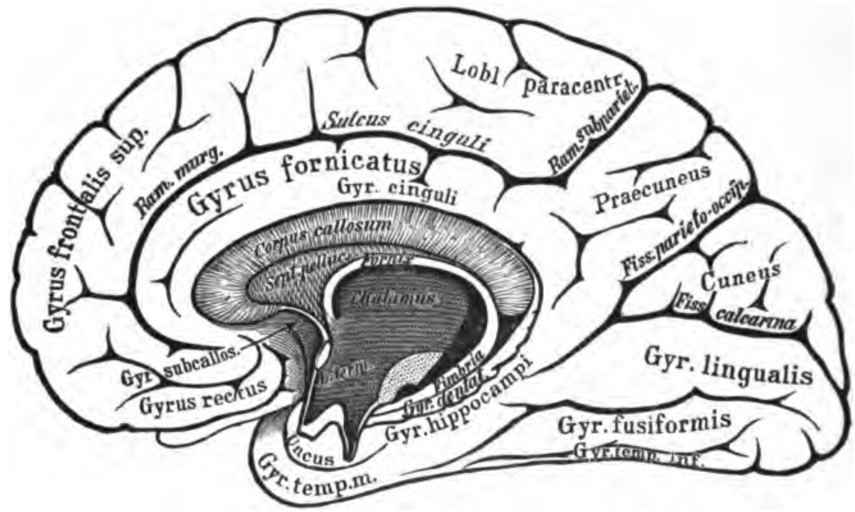

Fig. 6.—Longitudinal Section through the Middle of the Human Brain. (After Edinger.)

adjoins the ganglia of the cerebrum. Around this structure runs the *sulcus circularis Reilii*; anteriorly and ventrally the island passes into the free structure at the base of the brain, and at the limen insulæ directly into the *lamina perforata anterior.* A permanent sulcus, the *sulcus centralis insulæ,* divides the island into two lobes; the larger anterior (pars frontalis) possesses three to four gyri breves, the smaller posterior (pars parieto-occipitalis) has one to two gyri longi which pass to the temporal pole. The region of the island, which is especially developed in man as the association region of human speech, shows in animals a gradually ascending development. Waldeyer found in his investigations of the brain of anthropomorphic apes that the construction of the island in the monkey is fundamentally the same as in man; there is first an arching convolution with strongly linked frontal and weaker temporal arches; an increased development is apparent as we pass

from the gibbon to the orang-utan, the chimpanzee and the gorilla, the structure in the last named most closely resembling that of man.

The only rational method for fixing in the mind the fossæ and convolutions of the brain is to study the hardened brain, preferably that freed from its membranes. The great variety of fossæ and convolutions will then be apparent; it is frequently difficult to recognize an individual fossa unless typical fossæ, such as the Sylvian or the sulcus centralis, are chosen as the starting point for the investigation. The whole cortex of the brain with its fossæ and convolutions becomes interesting to the practitioner with the development of **cerebral localization**, which shows that definite areas of the cortex of the brain are the origin of definite functions, and that after the destruction of these parts of the brain the functions cease or show deterioration. This truth, which in part was taught by Gall at the beginning of the nineteenth century, so that, notwithstanding many errors, this writer deserves an honorable position among the investigators of the brain, was subsequently promulgated by Broca who developed the localization of the function of speech, but it was generally accepted only when Fritsch and Hitzig in 1870 for the first time demonstrated the faradic irritability of definite areas of the cortex, after which these authors began by experiments and extirpation to determine the symptoms of disease in these areas. It would lead us too far afield to discuss this subject fully, and I will merely state that through the untiring labors of men like Hermann Munk, Goltz, Ferrier, Horsley, Beevor, Schäfer, Sherrington, and others, a structure has been erected which reveals to us many of the functions of the cortex of the brain, and this has been a prerequisite for surgical successes in this difficult realm. To mention briefly the most important, the *region of the central convolutions* and their immediate surroundings is to be regarded as the seat of *sensation of the body,* and is the region in which sensations coming from the *periphery of the body* are expressed in movement, the uppermost portion of both central convolutions and the paracentral lobe controlling the lower extremity, the middle portions of the central convolutions the upper extremity, the lower portion of the central convolution the facial nerve, and; still lower, the operculum, controlling the mouth, the tongue and the larynx. Contractions of definite muscle groups upon the opposite side of the body originate from definite areas of these so-called "centers"; these, however, can be produced only by the surface portions of the convolutions, and extirpation of entire centers may cause paresis in the affected extremities. It must be borne in mind that not isolated muscles but muscle synergists are located in the cortex of the cerebrum, and these are the better developed the more they are consciously called into action. In man, the surgery of the brain has exposed but little of the cortex of the brain to observation, and we must therefore depend chiefly upon investigations of the brain of anthropomorphic apes. Beevor and Horsley have made careful experiments in orang-utans, and these have been almost absolutely confirmed by the results in man; the recent experiments of Grünbaum and Sherrington in eleven (!) anthropomorphic apes (gorilla, chimpanzee, orang-utan) have in the main confirmed these, but have shown that only the anterior central convolution is stimulated by weak faradic currents.

But it must be emphasized that the results of irritation only confirm the position of individual centers which, as is especially clear from the classical

investigations of H. Munk, are sharply separated from one another in their entire circumference and occupy a much larger area. In front of the centers for the extremities is the center for the muscles of the back of the neck, and the center for the muscles of the trunk is in the frontal brain which has not as yet been accurately studied. The sense of sight is in

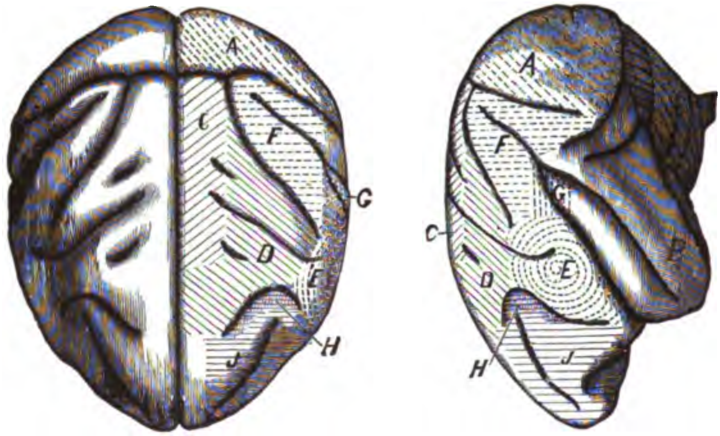

FIG. 7.—CORTEX OF THE CEREBRUM OF APES. (After H. Munk.)

A, Center for sight; *B*, center for hearing; *C–I*, sensory sphere; *C*, hind-leg region, sensory sphere; *D*, fore-leg region; *E*, head region; *F*, eye region; *G*, ear region; *H*, neck region; *I*, trunk region.

the occipital lobe, that of hearing in the temporal lobe. Besides experimental investigations among which those of H. Munk are here also to be regarded as pioneer—we owe to him the recognition of *mind blindness* and *mind deafness* which have since become of the greatest importance in human pathology—valuable studies have been made of the pathological affections of these areas in man. The center for most acute vision does not appear to be in the lateral surface of the occipital lobe but in the region of the fissura calcarina; it is impossible, however, at this point to discuss minutely the complicated conditions which produce hemianopsia. The relations of the temporal lobe to the sense of hearing are similar to those of the occipital lobe to sight, but they have not yet been accurately studied. It is usually the upper temporal convolution which is considered; but the relations in man are much more complicated than in the most highly organized animals because of the power of speech which, when these areas are diseased, shows impairment known as *sensory aphasia*; this corresponds with a much rarer pathologic picture, *optic aphasia*, in destruction of the communications of the occipital lobe with the other "speech centers." The centers for movements of the eye and ear are not the same as the centers for sight and hearing; on the contrary, the centers for optical movements appear to be localized in two areas, in the gyrus angularis of the parietal lobes immediately in front of the center of sight and in the region of the second frontal convolution, while aural movements originate in the posterior division of the first temporal convolution. In man the third, or Broca's, frontal convolution must be borne in mind; here closely joined are localized the centers for the muscle groups

brought into play in *motor speech function,* the destruction of which, therefore, produces the well known picture of *motor aphasia* with its various modifications. We cannot here discuss the enormous extension of the localization of speech disturbances in the teachings of the last few years. In the physiologic localization of the centers for *smell* and *taste,* nothing decisive has been revealed. Comparative anatomical researches, it is true, point to the region of the gyrus hippocampi, the gyrus fornicatus, the fascia dentata, and, above all, to the cornu ammonis as the center for the sense of smell, since these portions of the brain are highly developed if the bulbus olfactorius is prominent, and in the dolphin in which the lobe of smell is absent they are greatly decreased; therefore these areas of the brain together with the lobus olfactorius are known as the *rhinencephalon.*

Nevertheless, the study of cerebral localization is far from being exhausted; on the contrary, it still promises rich results; definite lobes of the brain and

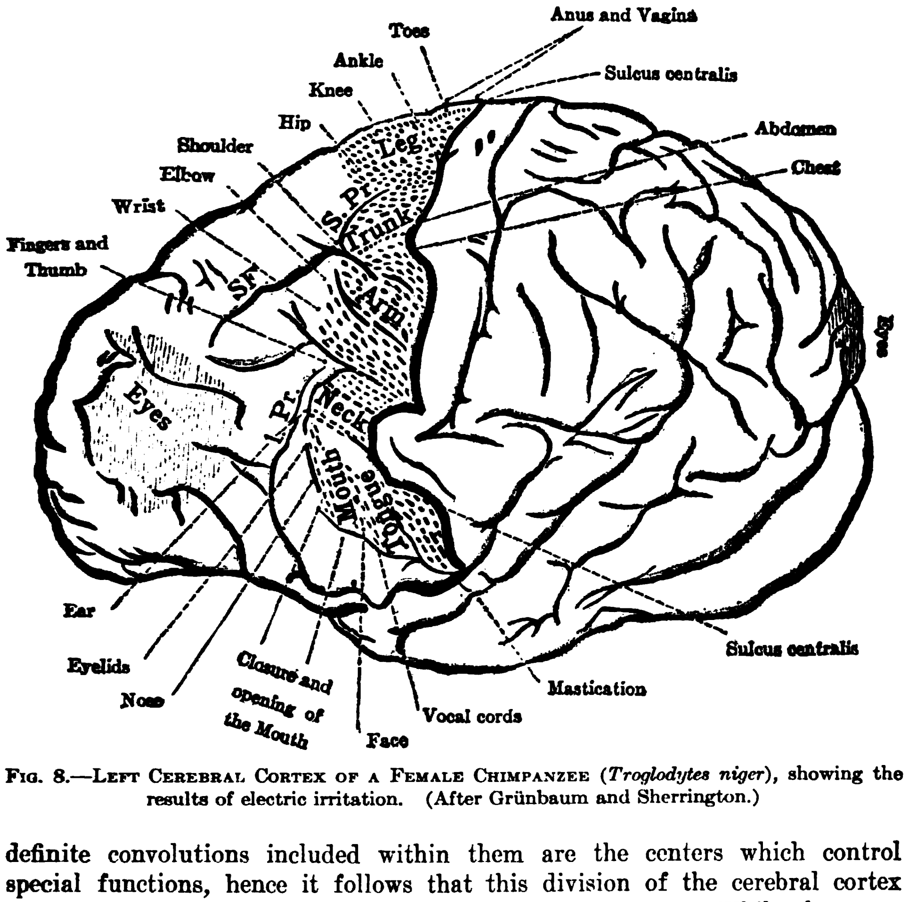

FIG. 8.—LEFT CEREBRAL CORTEX OF A FEMALE CHIMPANZEE (*Troglodytes niger*), showing the results of electric irritation. (After Grünbaum and Sherrington.)

definite convolutions included within them are the centers which control special functions, hence it follows that this division of the cerebral cortex into fossæ and convolutions is physiologically important. While, however, in the human brain at least, some centers have been recognized, there remain large areas of the cortex of the brain which at present permit no recognition of the localization of definite cerebral functions, and Flechsig has attempted,

by the same method which he instituted so successfully in the investigation of the spinal cord and the development of the medullary sheath, to divide

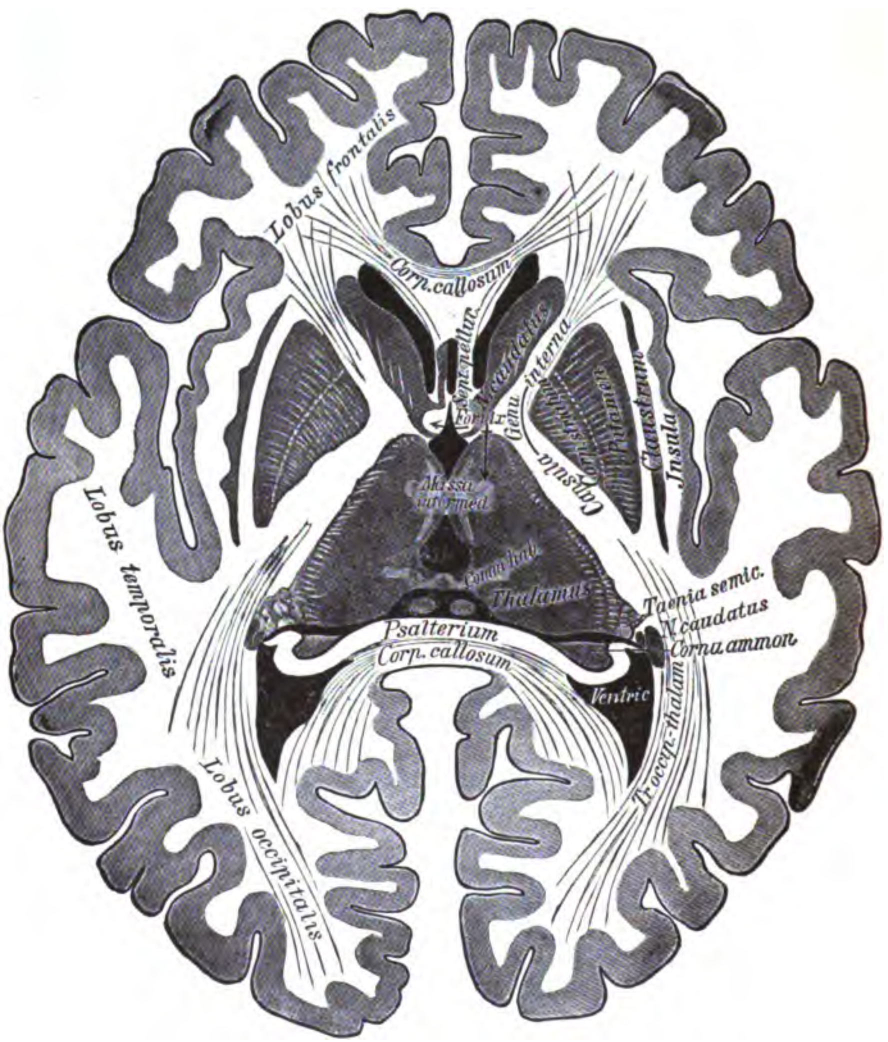

Fig. 9.—Horizontal Section through the Brain with the Nucleus Caudatus, Thalamus Opticus, and Nucleus Lentiformis. (After Edinger.)

the cerebral cortex into separate areas in which, besides the projection centers just described which are connected by the corona radiata fibers with deeper sections of the central nervous system, he differentiated as higher psychical centers association centers said to be almost entirely free from fibers of the corona radiata and therefore parallel with Munk's centers of special sense from the fact that they unite the activity of the latter to higher units. After finding these three main *association centers*, a larger posterior area in the parietal lobe and the lateral portion of the occipital brain, a middle area

in the island, and an anterior one in the anterior region of the frontal brain, and to facilitate the more comprehensive embryologic investigation of the brain, Flechsig differentiated many similar but smaller centers. Whether the teaching of Flechsig, whose anatomical and physiological theory has been often attacked, is correct is by no means yet certain, although its fructifying effect upon the study of the brain is undoubted.

So far we have exclusively considered the cerebral cortex, which in man represents the dominating portion of the brain, and we now turn to the *ganglia of the cerebrum,* situated in the interior of the brain, and separated from the cortex by white medullary masses. To these primarily belongs the trunk ganglion, the *corpus striatum,* which in the early embryonic stages and in the lower animals is a uniform body, but in the fourth month and subsequently is divided into two parts by fiber masses which pass down from the pallium; also an external one, the *nucleus lentiformis,* and an internal one, the *nucleus caudatus,* while the fiber mass which separates them is called the *internal capsule.* The *nucleus caudatus* is a large gray mass which is directed forward into the lateral ventricle; its broadest part is the anterior extremity or "head" in front of the thalamus opticus, constantly becoming narrower posteriorly, and terminating laterally as a narrow, medullary fold, the *stria terminalis,* which runs posteriorly downward, at last anteriorly, and at the lower horn in the neighborhood of the apex of the temporal lobe it terminates as the

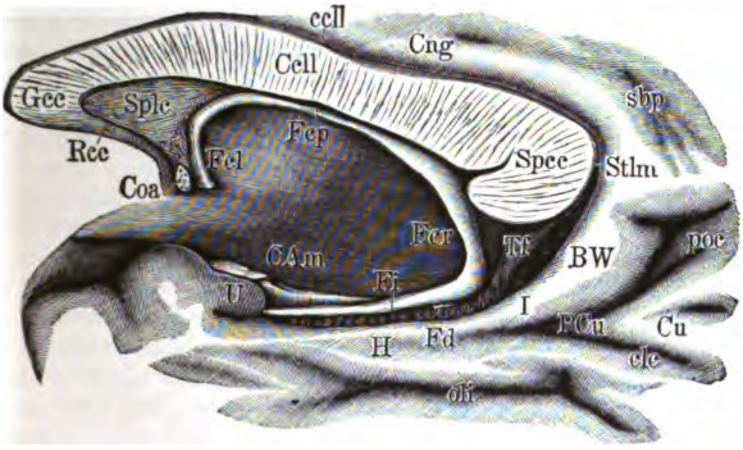

Fig. 10.—Portion of a Median Section through the Cerebrum. (After Obersteiner.)

CAm, Cornu ammonis; *Cell,* corpus callosum; *cell,* sulcus corporis callosi; *clc,* fissura calcarina; *Cng,* gyrus cinguli; *Coa,* commissura anterior; *Fcl,* columna fornicis; *Fcp,* corpus fornicis; *Fcr,* crus fornicis; *Fd,* fascia dentata; *Fi,* fimbria; *Gcc,* genu corporis callosi; *H,* gyrus hippocampi; *Rcc,* rostrum corporis callosi; *Spcc,* splenium corporis callosi; *Splc,* septum pellucidum; *Stlm,* stria longitudinalis med.

"tail." Outward from the nucleus caudatus is the *nucleus lentiformis,* a wedge-shaped mass of gray matter almost wholly separated from the former, and entirely from the thalamus opticus by the fibers of the internal capsule; neither anteriorly nor posteriorly does it reach as far as the nucleus caudatus; it is separated into three divisions by thin medullary sheaths of which the medial portion is of lighter color and is known as the *globus pallidus,* while

the outer, darker, and largest portion is called the putamen. The lateral surface of the putamen is parallel with the island of Reil from which it is separated by a narrow gray streak, the *claustrum*; and between the latter and the putamen runs the narrow medullary lamina, the *external capsule.* Posteriorly to the nucleus lentiformis and close to the tail of the nucleus caudatus, communicating with both, is the sharply defined gray mass, the *amygdala,* which is also united with the hippocampus major. The relation of these gray ganglia to each other and to the adjacent portions of the brain, and their physiologic importance in man and in the higher mammals, can be determined only by microscopic and comparative anatomical examination, and in spite of many experiments and pathologic observations we are still completely in the dark concerning these.

The cerebral hemispheres are united with one another by several **commissures**: (1) The most important of these is the corpus callosum which is seen upon the floor of the great medial, longitudinal fissure. Besides this portion of the corpus callosum, called the truncus, we find also the posterior *splenium corporis callosi* which is thickened and lies above the corpora quadrigemina, anteriorly bending somewhat downward the *genu corporis callosi,* which may be followed backward to the fornix as a narrow line, the *rostrum.* Between the corpus callosum and the rostrum the *septum pellucidum* spreads out in the form of two thin plates and contains the ventriculus septi pellucidi. (2) Below the corpus callosum the *fornix* unites both hemispheres in the form of a narrow band (the *fimbria*), passing out of the lower horn of the lateral ventricles at each side in the region of the cornu ammonis, uniting upon the two *crura fornicis,* passing along the lower surface of the corpus callosum, to divide in front at the lower end of the septum pellucidum into two columns, the *columnæ fornicis*; the latter extend to the base of the brain, to the *corpora mamillaria.* (3) The *anterior commissure* as a white band joins the hemispheres in front of the columnæ fornicis, which unite the basal portions of the temporal and frontal lobes.

In their entire extent the cerebral hemispheres are permeated by cavities, the **lateral ventricles** mentioned above. These cavities pass arch-like from above anteriorly downward posteriorly and permit the differentiation of a middle portion, the *cella media,* and three horns, the *anterior horn* which passes into the frontal lobes, the *posterior horn* entering the occipital lobe, and the *lower horn* extending to the temporal lobes. The cella media shows upon its floor, besides other structures, the tail of the nucleus caudatus, while above is the middle portion of the corpus callosum. The *anterior horn* passes around the head of the nucleus caudatus into the posterior portion of the temporal lobe, and is bounded above by the corpus callosum, while medially it reaches the septum pellucidum. The *posterior horn* runs dorsally close to the occipital pole. The most important structure upon its base is the *calcar avis* or *pes hippocampi minor,* which is produced by the sinking in of the fissura calcarina on the medial surface of the occipital lobe. Here are also a number of fiber strands which play a rôle in the law of cerebral localization; the optic radiation of Gratiolet which passes from the thalamus opticus to the occipital lobe, the unilateral interruption of which produces hemianopsia, and the *fasciculus longitudinalis inferior,* which connects the temporal and occipital lobes. The posterior horn is covered from above by the fiber masses

of the *tapetum* coming from the posterior portion of the corpus callosum. The lower horn passes downward and forward close to the tip of the temporal lobe and is covered on the upper side by the tapetum, also by the finer processes of the tail of the nucleus caudatus which here passes into the nucleus amygdalæ. The cerebral structures arranged upon the floor of the lower horn are very complicated. Here we find all of the structures with which we have become familiar in the description of the medial surface of the hemispheres, the *gyrus hippocampi*, the *fascia dentata*, the *fimbria*, the marked swelling of the *cornu ammonis* (pes hippocampi major), and the prominence of the *eminentia collateralis Meckelii* due to the deep entrance from without of the sulcus occipito-temporalis inferior which permits a passage to the posterior horn.

The lateral ventricles do not directly communicate with each other but are indirectly united by the *foramen Monroi*, by the open space on each side between the thalamus opticus and fornix, and also between the lateral ventricles and the third ventricle. All of these ventricles are now filled by the plexus chorioidei, profuse vascular twigs covered by an epithelial layer; the latter corresponds to the inserted remains of the medial wall of the hemisphere and the roof of the inter-brain. Here we differentiate first a triangular middle leaf, the *tela chorioidea ventriculi tertii*, which is spread out under the *fissura transversa cerebri* (Bichat's furrow) situated under the splenium corporis callosi and the columnæ fornicis, thence the vascular villi reach the lateral ventricles by way of the foramen Monroi where they chiefly occupy the cella media and the lower horn as the *plexus chorioidei lateralis*, and then pass to the most anterior point of the latter.

The white medullary masses situated below the cortex of the cerebrum of both hemispheres are called the **centrum ovale**. Among the various projection fiber tracts here included, such as the corona radiata and the association tracts which have been thus far investigated, we shall minutely concern ourselves only with the *internal capsule*, i. e., that portion of the medulla of the hemispheres formed in thick bundles in which projection fibers are inserted by the lenticular nucleus from without, the caudate nucleus and the optic thalamus from within, a region especially important because here originate the hemorrhages which produce hemiplegia and its sequels. As the tracts are embraced within a narrow space formed by various parts of the cerebral cortex, and their fibers do not interlace, and as the tracts which pass to the cerebral cortex from the large ganglia, especially from the thalamus opticus, begin to separate and later pass to the corona radiata and the various cortical centers, a comparatively small focus may cause extensive symptoms as to motility, sensation, and the higher spheres of special sense, while these may be definitely localized according to whether such a focus is situated anteriorly or posteriorly. The internal capsule forms an obtuse angle in the genu of which the two crura meet; the anterior, smaller, and physiologically, so far as our knowledge extends, less important crus, the *pars frontalis*, is situated between the nucleus caudatus and the lenticular nucleus. The posterior, longer crus, which is most frequently affected and which is the usual seat of cerebral hemiplegia, the *pars occipitalis*, runs between the thalamus opticus and the lenticular nucleus and extends beyond the latter more occipitally. Hence the entire internal capsule may be divided into four portions, the ventral,

the lenticulo-striate, the knee portion, the lenticulo-optic, and the retrolenticular part. Not to enter minutely into the exact division of the fibers, it need only be stated that the pyramidal tract, which is the most important and the only direct means of communication between the cerebral cortex and the spinal cord, runs in the anterior half of the lenticulo-optical portion, therefore in the posterior peduncle of the internal capsule. Starting at the knee we pass to the beginning of the posterior third of the lenticulo-optic division, therefore the irritative points in the various groups of muscles of the body (which we have chiefly learned to recognize by the studies of Beevor and Horsley in anthropomorphic apes) are situated in this area, from before

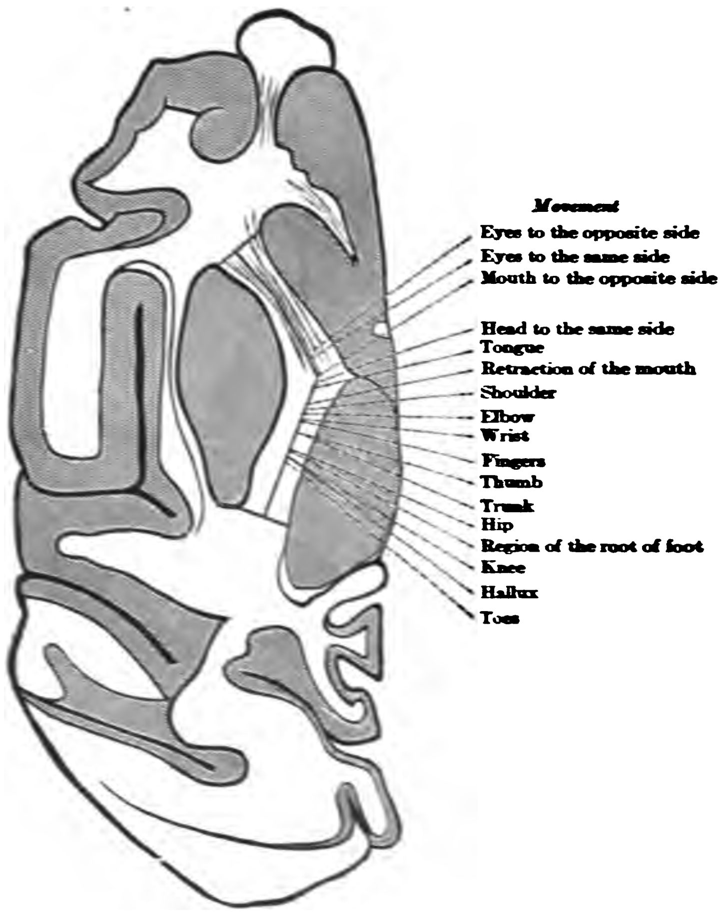

FIG. 11.—ARRANGEMENT OF THE MOTOR FIBERS IN THE ANTERIOR CAPSULE. (After Beevor and Horsley.)

backward, for the head, arm and leg. Uniting with the latter in the posterior third of the lenticulo-optic division is the sensory or lemniscus tract, and dorsally from this, in the retrolenticular portion of the internal capsule, the optic radiations of Gratiolet which pass to the occipital lobes. For this rea-

son foci in the anterior portion of the posterior peduncles of the internal capsule produce uncomplicated hemiplegia, while the further the focus extends posteriorly the earlier hemianesthesia and even hemianopsia appear.

Within the region of the internal capsule of the cerebrum we find the *inter-brain* whose most important structure is the *optic thalamus*, but which also includes the *pineal gland* or *epiphysis*, the *ganglion habenulæ* and, at the base of the brain, the *corpora mamillaria*, the *chiasma nervorum opticorum*, and other structures. The two optic thalami form the borders of the third ventricle, being directly connected with the gray substance of its cavities. The mighty gray masses of the optic thalami are divided into several parts which may readily be recognized macroscopically after transverse section. These *nuclei of the optic thalami* are the following: The tuberculum anterius, the median nucleus, the ventral nuclei groups differentiated by v. Monakow in his experiments as vent. a, vent. b, vent. c, and vent ant. Dorsally from these are the lateral nucleus and the pulvinar; finally, at the point where the optic tract enters the brain on the lateral side of the cerebral peduncles, the corpus geniculatum externum and the corpus geniculatum internus adjoining medially the pedunculus cerebri from the external geniculate body. The corpus mamillare situated upon the base of the brain may be included with the nuclei belonging to the thalamus opticus; it is united with the hippocampus major. Nearly the entire structure of the thalamus opticus has been investigated; its connection with the cortex of the cerebrum was discovered by v. Monakow during his pioneer investigations in new-born animals, by which he was able to prove that certain regions of the optic thalamus are intimately related to definite divisions of the cortex of the brain, and that when the latter, the so-called *portions of the cerebrum*, are removed they become atrophic. Certainly it might be proven that all of the nerves of special sense are connected with the cerebrum by means of the optic thalamus; for instance, the cortical lemniscus, the tract of sensation for the body through the ventral nucleus of the thalamus, the acoustic tract through the corpus geniculatum internus, the optic tract through the corpus geniculatum

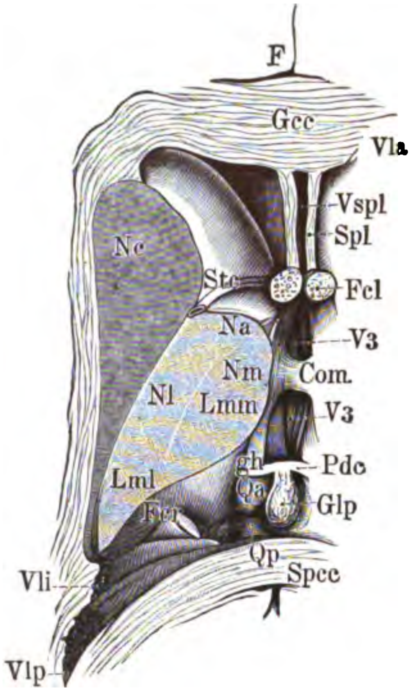

FIG. 12.—HORIZONTAL SECTION THROUGH THE INTER-BRAIN, ONE-HALF CENTIMETER BELOW THE SURFACE OF THE THALAMUS AND THE NUCLEUS CAUDATUS. (After Obersteiner.)

Com, Commissura mollis; *Fcl*, columna fornicis; *Fcr*, crus fornicis; *Gcc*, genu corporis callosi; *gh*, ganglion habenulæ; *Glp*, glandula pinealis; *Na*, nucleus anterior thalami; *Nc*, nucleus caudatus; *Nl*, nucleus lateralis thalami; *Nm*, nucleus medialis thalami; *Pdc*, pedunculus gland, pinealis; *Qa*, anterior; *Qp*, posterior, corpora quadrigemina; *Spcc*, splenium corporus callosi; *Spl*, septum pellucidum; *Stc*, stria cornea; *Vli*, lower horn of the lateral ventricle; *Vlp*, posterior horn; *V3*, third ventricle.

externus and the pulvinar. v. Monakow's investigations, which have recently been absolutely confirmed by Probst in another way, therefore show that the thalamus opticus is a receptacle for the fibers coming from deeper portions of the brain and from other trunk ganglia, also for the corticifugal fibers, while the structure sends comparatively few fibers downward. It is chiefly an organ of transmission between the periphery and the cerebrum, but our knowledge of its minute physiologic and pathologic function is extremely limited.

The space between the two optic thalami, the *third ventricle*, is bordered above by the plexus chorioideus, at its posterior end by the *pineal gland* or the *epiphysis*, close to the anterior division of the corpora quadrigemina. Immediately in front of it and equidistant at each side from the posterior region of the thalamus is the small ganglion *habenulæ*. The epiphysis consists of solid, very vascular, epithelial tubules which contain peculiarly layered calcium concretions, the *brain sand*. We know nothing of the importance of the epiphysis in man; comparative anatomy, however, teaches us that this is a rudimentary organ which, in the lower vertebrate animals, extends forward as a long tube, its frontal end forming externally the frontal spot which is perceptible on the skull between the orbital spaces; a few saurians also contain a second frontal "parietal organ," the terminal vesicles situated within one of the spaces of the parietal bone, its structure closely resembling an eye with cornea, lens and retina. In its lower portion the third ventricle shows the *tuber cinereum* which extends to the base of the skull, the cavity of which is called the *infundibulum*. The structures below the thalamus opticus compose the *subthalamic region*; but this area has been but little investigated microscopically and seldom gives opportunity for macroscopic examination. Immediately below the optic thalamus is the *nucleus ruber tegmenti* which a little lower passes into the corpora quadrigemina, and outside of this is the corpus subthalamicum, Luys's body. At the base of the brain the cerebral peduncles run to the pons; here is situated the pes pedunculi which receives the fibers from the internal capsule in the form of an accumulation of markedly pigmented ganglion cells, the *substantia nigra Soemmeringi*, which are distinct from the fibers of the lemniscus situated above.

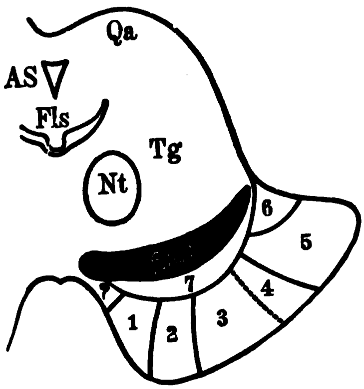

FIG. 13.—DIAGRAM OF THE CEREBRAL PEDUNCLES. (After Obersteiner.)

Qa, Anterior corpora quadrigemina; *AS*, aqueduct of Sylvius; *Fls*, posterior longitudinal bundle; *Tg*, tegmentum; *Nt*, red nucleus; *SnS*, substantia nigra Soemmeringi; *1*, frontal pontal tract; *2*, tract of the motor cranial nerves; *3*, pyramidal tract; *4*, tract of muscle sense; *5*, sensory portion of the fibers of the cerebral peduncle; *6*, bundle from the lemniscus to the foot; *7*, stratum intermedium.

We now reach the region of the **midbrain**, the principal structure of which is the *corpora quadrigemina*, so called because here, immediately behind the third ventricle and the epiphysis, four small eminences are recognizable, two in front and two behind; each of these structures possesses an arm, the *anterior* and *posterior superior peduncle* which passes to the inter-brain. The *ante-*

rior eminence in man is exceedingly small compared with that of the lower animals, for its function as the center of the optic nerve is almost entirely merged in that of the occipital lobe and the thalamus opticus. Immediately below its center is the narrow posterior continuation of the third ventricle, the *aqueduct of Sylvius*; at either side extending downward is the *region* of the

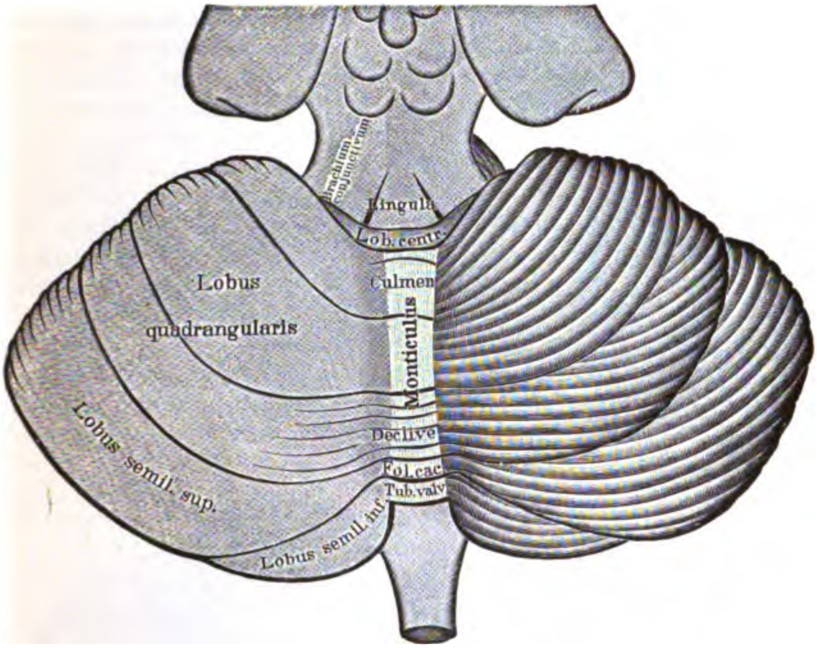

FIG. 14.—THE CEREBELLUM FROM THE DORSAL SIDE. (After Edinger.)

tegmentum which, as we have just seen, is separated from the foot of the cerebral peduncles by the substantia nigra. The *posterior eminences* appear macroscopically to be more sharply defined than the anterior. In all transverse sections of the corpora quadrigemina the *red nucleus* may be recognized as a round red structure, lying dorsally from the aqueduct of Sylvius and near the median line; in this structure generally terminates the *brachium conjunctivum* which comes from the cerebellum and has just crossed it. Between the two red nuclei are the nuclei and fiber bundles of the third cranial nerve, the oculomotor, the exit of which at the base of the brain will be later described, and behind it are the nuclei of the trochlear nerve.

The aqueduct of Sylvius after dilating becomes posteriorly the *fourth ventricle*, the rhomboid fossa, the roof of which is formed by the *cerebellum*, the floor and sides by the continuation of the foot and tegmentum of the midbrain which form the pons. The foot of the cerebral peduncle is here covered by large, transverse, running masses of fibers and is permeated by them, while the tegmental fibers receive their peculiar configuration from the disappearance of the red nucleus and by the passing to and fro of the fibers from the cerebellum.

The **cerebellum** itself bears undoubtedly an alternate relation to the cere-

brum, for in animals in which the latter is markedly developed the former is likewise, and when there is atrophy of one hemisphere of the brain, atrophy of the crossed half of the cerebellum is also observed. The cerebellum is composed of the vermis situated in the middle and of two *hemispheres*. It is united with the red nucleus and the thalamus opticus by the *anterior brachium conjunctivum*, sends the *middle brachium conjunctivum* or pontal arms to the pons, and by means of the *posterior brachium conjunctivum*, the *corpora restiformia*, is connected with the medulla and the spinal cord. The vermis of the cerebellum is divided into lobes by furrows; the most important which lie dorsally upon the upper vermis, and extend from before backward, are the lingula, the central lobe, and the monticulus, and upon the lower vermis the nodulus, uvula, pyramis and the tuber vermis. The hemispheres of the cerebellum show above, dorsally, and anteriorly a *quadrangular lobe* at the side of the monticulus, and posteriorly the *lobulus semilunaris superior*; below, besides the nodulus, is the *flocculus*, upon the uvula the *tonsilla*, laterally from this the *cuneiform lobe*, to which the *inferior posterior* lobe is posteriorly joined. The vermis and hemispheres possess a medullary nucleus; in its interior, particularly in the vermis, gray nuclei are found, also the *corpus dendatum* at the boundary of the vermis and hemispheres, and medially from this the embolus, the *nucleus globosus*, and the *tegmental nucleus*. From the cerebellum over the fourth ventricle to the corpora quadrigemina passes ante-

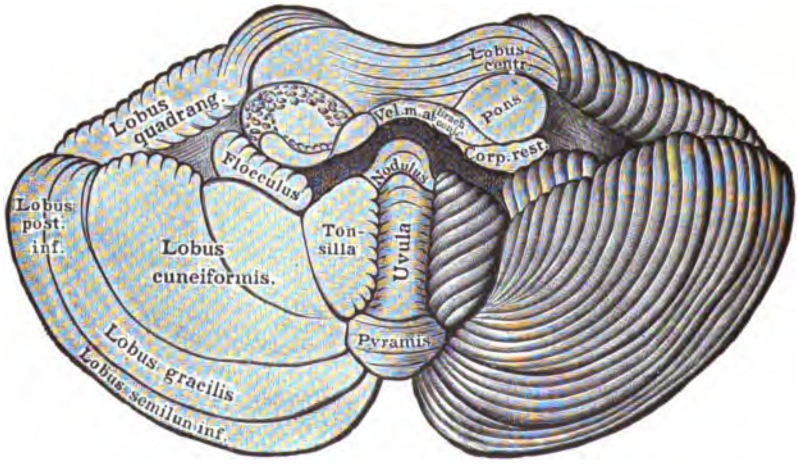

FIG. 15.—THE CEREBELLUM FROM THE VENTRAL SIDE. (After Edinger.)

riorly the velum medullare anticum, posteriorly the velum medullare posticum, and these extend to the posterior columns of the spinal cord. The *plexus chorioidei*, situated in the region of the ventricles, and which also passes into the sinuses of the fourth ventricle and into open spaces the largest of which is the *foramen of Magendie*, permits communication with the arachnoid space; this is of importance in equalizing variations of pressure. The manifold communications of the cerebellum with most of the other structures of the central nervous system by means of the brachium conjunctivum cannot here be mentioned; they have become known only by minute comparative anatom-

ical investigations in degeneration. It is certain that the cerebellum is indirectly connected with the cortex of the cerebrum by the pontal arms, to the brachium conjunctivum by the red nucleus and thalamus opticus, and thus indirectly with the cerebrum, while the corpus restiformia communicates with the cerebrum from the spinal cord and medulla.

The *cerebellum* has recently acquired a special importance because, owing to tumors and suppurations in this region or in its immediate vicinity, it has

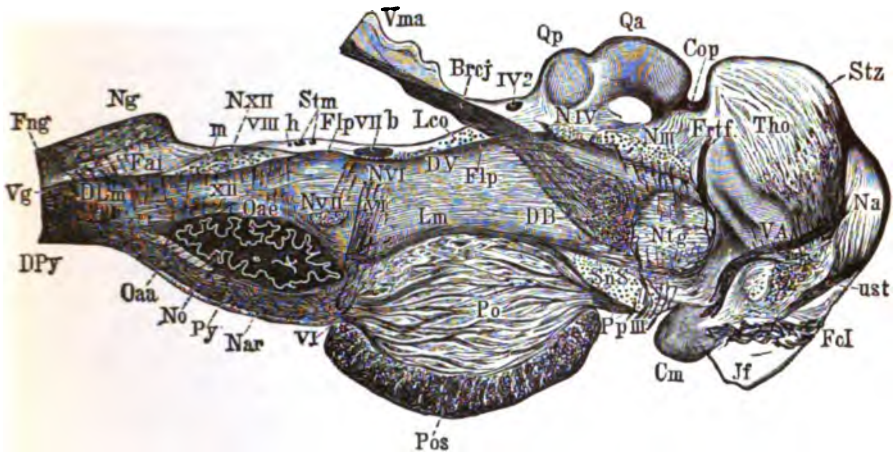

FIG. 16.—COMBINED SAGITTAL SECTION THROUGH THE STEM OF THE BRAIN, TWICE ENLARGED. (After Obersteiner.)

Brcj, Brachium conjunctivum; *Cm*, corpus mammillare; *Cop*, posterior commissure; *DLm*, Lemniscus decussation; *DPy*, pyramidal decussation; *Fng*, funiculus gracilis; *Jf*, infundibulum; *Lm*, lemniscus; *Na*, anterior nucleus of the thalamus; *Ng*, nucleus gracilis; *No*, olivary nucleus; *Ntg*, red nucleus; *Oaa* and *Oae*, ventral and dorsal secondary olive; *Po*, pons; *Pp*, pes pedunculi; *Py*, pyramid; *Qa* and *Qp*, anterior and posterior corpora quadrigemina; *SnS*, substantia nigra; *Tho*, thalamus opticus; *Vg*, anterior column ground bundle; *Vma*, velum medullare anterius.

been laid bare, and has been operated upon. Experimental and pathologic observations have conclusively shown that the cerebellum serves to maintain equilibrium, for which it is especially designed by its connection with the varied sensory and motor apparatus of the central nervous system. Disturbances arising after affections of the cerebellum are usually referred to the vermis, while but little is known of the hemispheres. At all events the fact is interesting that by electric irritation of the cortex of the cerebellum muscular twitchings similar to those originating in the cortex of the cerebrum may be produced, only they do not occur in the extremities of the same, but in the opposite, side.

As the fourth ventricle in its path toward the cauda gradually becomes smaller, and the pyramids pass ventrally to the surface, the comparatively large pons enters the **medulla oblongata**, which also gradually becomes narrower. At the point where the pons passes into the medulla, the abducens, facial and auditory nerves originate, while the trigeminal nerve arises in the lateral portions.

The *lower olive* (nucleus olivaris inferior) lies alongside the very prominent pyramids, which are characteristic of the medulla upon all transverse

sections up to the lemniscus decussation, which then becomes visible medially as an open, peculiarly serrated, arc-like body behind the pyramids. In the upper portions of the medulla, dorsally and laterally, we note the large corpus restiforme which in its downward path rapidly decreases in size and finally disappears. On the other hand, the lower half of the medulla oblongata after the fourth ventricle has closed to the narrow central canal, dorsally of the latter, the nuclei of the spinal posterior columns, the *nucleus gracilis* and the *nucleus cuneatus* appear, while from the pons up to the upper spinal cord the so-called *ascending root* of the *trigeminal nerve* (which in reality degenerates downward in combination with the *substantia gelatinosa* which passes into the posterior horn of the spinal cord) becomes prominent. The nuclei of the cranial nerves from the ninth to the twelfth are situated in the dorsal portion of the medulla, some immediately upon the floor of the fourth ventricle; they are insusceptible to macroscopic investigation. Medially behind the pyramids is the *lemniscus layer* which, immediately above the well known pyramidal decussation, forms the lemniscus decussation.

Before passing to the study of the spinal cord which begins at the height of the pyramidal decussation, we will briefly review the **structures at the base of the brain**, arranged in an antero-posterior direction. Their relations are of practical importance inasmuch as we know there are a number of basal processes, tumors, syphilitic exudates, etc., in which an exact, local diagnosis necessitates a knowledge of the sequence of these individual parts, particularly the order in which the cranial nerves are affected. Most anteriorly at the base of the brain and below the frontal lobes are the olfactory bulbs, which narrow posteriorly toward the olfactory tract. Behind them is the *substantia perforata anterior*, above which thin white strands, the roots of the *olfactory nerve,* pass into the medulla of the hemispheres.

Posteriorly to these is the *optic chiasm* from which the optic tracts run posteriorly and outwardly, surrounding the cerebral peduncles, and extending to the thalamus opticus and the corpora quadrigemina. Behind the optic chiasm we find the floor of the third ventricle, the tuber cinereum with the infundibulum, at the extremity of which is the *hypophysis*. This latter structure has in the last few years attracted great attention because it has frequently been found to be enlarged in acromegalia, and the pressure upon the chiasm has produced bitemporal hemianopsia. In how far these enlargements of the hypophysis are the cause of acromegalia has not yet been determined. Behind the hypophysis are the two *corpora mammillaria,* also a gray substance, the *substantia perforata posterior,* lying upon the floor of the subthalamic region between the two cerebral peduncles. At either side of this structure the cerebral peduncles pass posteriorly and medially to the pons. The oculomotor nerve appears in front close to the origin of the great transverse fibers of the pons and on each side of the internal border of the peduncles, while somewhat further on, and posteriorly between the lateral posterior border of the peduncles and the pons, the trochlear nerve arises. The fifth nerve with its narrow motor and broad sensory divisions emerges from the lateral posterior portion of the pons. While, up to this point, the cranial nerves are isolated at the base of the brain, when the pons enters the medulla and the cerebellum begins, the origins of the cerebral nerves are close together, so that, for instance, tumors which here implicate the bones of the posterior

groove of the skull or the cerebellum, even without attaining great development, may cause multiple cranial nerve paralyses. The abducens emerges medially from the transverse furrow between the pons and the medulla, while the facial and acoustic (VII and VIII) nerves appear at the side of the medulla oblongata. The glosso-pharyngeal and vagus follow caudad,

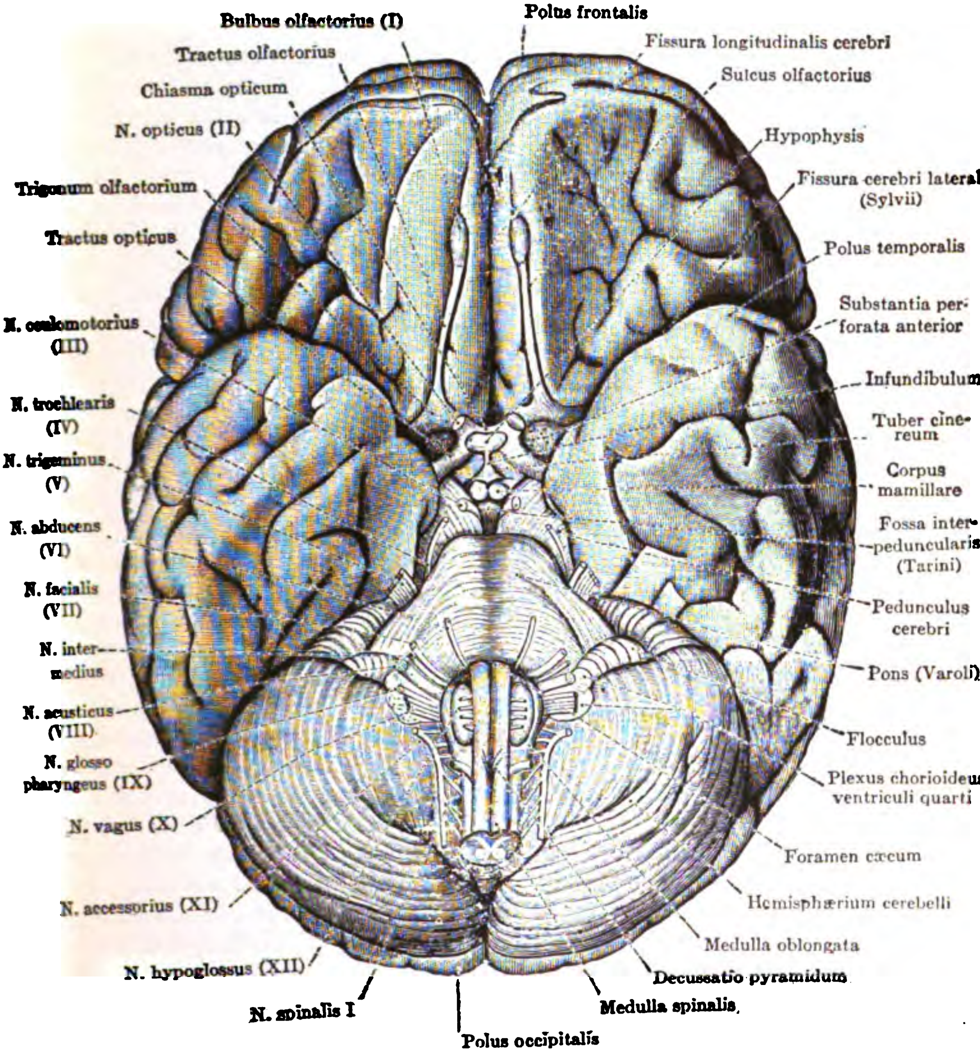

FIG. 17.—THE BASE OF THE BRAIN SHOWING THE ORIGIN OF THE ROOTS OF THE CRANIAL NERVES. The basal surface of the cerebrum in its posterior division is covered by the cerebellum.

while the spinal accessory and hypoglossal nerves arise from the lateral parts of the lowest portion of the medulla oblongata, the former, indeed, from the uppermost part of the cervical cord. The individual parts of the cerebrum and the cerebellum at the base of the brain have been discussed in preceding sections.

It is characteristic of the **arterial blood of the brain** that it is supplied by two channels; the anterior portion of the brain by the internal carotid artery, the posterior part by the vertebral artery which is a branch of the subclavian. The internal carotid divides at the external border of the optic chiasm into the anterior cerebral artery, which runs forward and medially and into the Sylvian or large middle cerebral artery, which passes into the Sylvian fossa.

At the same point the posterior communicating and the choroid arteries are given off posteriorly from the internal carotid. The vertebral arteries on both sides unite at the point where the pons passes into the medulla and form the basilar artery, which, after giving off a number of branches for the pons and the cerebellum, divides near the anterior end of the pons into the two posterior cerebral arteries which pass on laterally. The anterior cerebral arteries are united in front by the anterior communicating artery. The posterior communicating artery on each side passes into the posterior cerebral artery, and produces the well known *circle of Willis* which unites the arterial regions of the carotid and vertebral arteries, being composed of ten arteries, as follows: 1, Anterior communicans; 2 and 3, the anterior cerebral arteries; 4 and 5, the arteries of the Sylvian fossa (the middle cerebrals); 6 and 7, the posterior communicating arteries; 8 and 9, the posterior cerebral arteries; 10, the basilar artery. It is true many variations occur here. Of the most important arteries of the brain, the anterior cerebral supplies a large portion of the frontal brain, the gyrus fornicatus, the paracentral lobe, and the precuneus as well as the corpus callosum. The large, middle, cerebral artery (artery of the Sylvian fossa) supplies to a wide extent the area of the large ganglia and the internal capsule, and is of the greatest clinical importance as the usual seat of cerebral hemorrhage. It then gives off numerous collateral branches of which the first passes to the third frontal convolution, the second supplies the central convolution and the operculum, the third the gyrus supramarginalis and angularis, and the fourth and fifth extend to the temporal convolutions, while special branches supply the island and, at the same time, the claustrum and the external capsule. The *posterior cerebral artery* encircles the pedunculus, and then passes posteriorly across the medial wall of the occipital lobe. It supplies almost all of the walls of the ventricle, and also the posterior divisions of the optic thalamus, the corpora quadrigemina, and the hippocampus major. The *calcarine artery* is the most important of those arteries which pass to the cortex and supply the occipital lobes, the gyrus hippocampi and the third temporal convolution; this supplies the area of the fissura calcarina and the posterior division of the optic radiation of Gratiolet, an occlusion of which seriously impairs the sense of sight.

Among the arteries visible upon the base of the brain we see the vertebral and basilar arteries and their branches, also the circle of Willis which is the most important; the middle cerebral artery just after its origin passes under the lowest part of the temporal lobe and disappears in the depth of the brain.

Diseases of the cerebral arteries and hemorrhages, thromboses and emboli with resultant secondary softening of extensive areas of the brain form the largest proportion of all diseases of the brain. Therefore it is important for us to recognize that the distribution of the individual arteries throughout

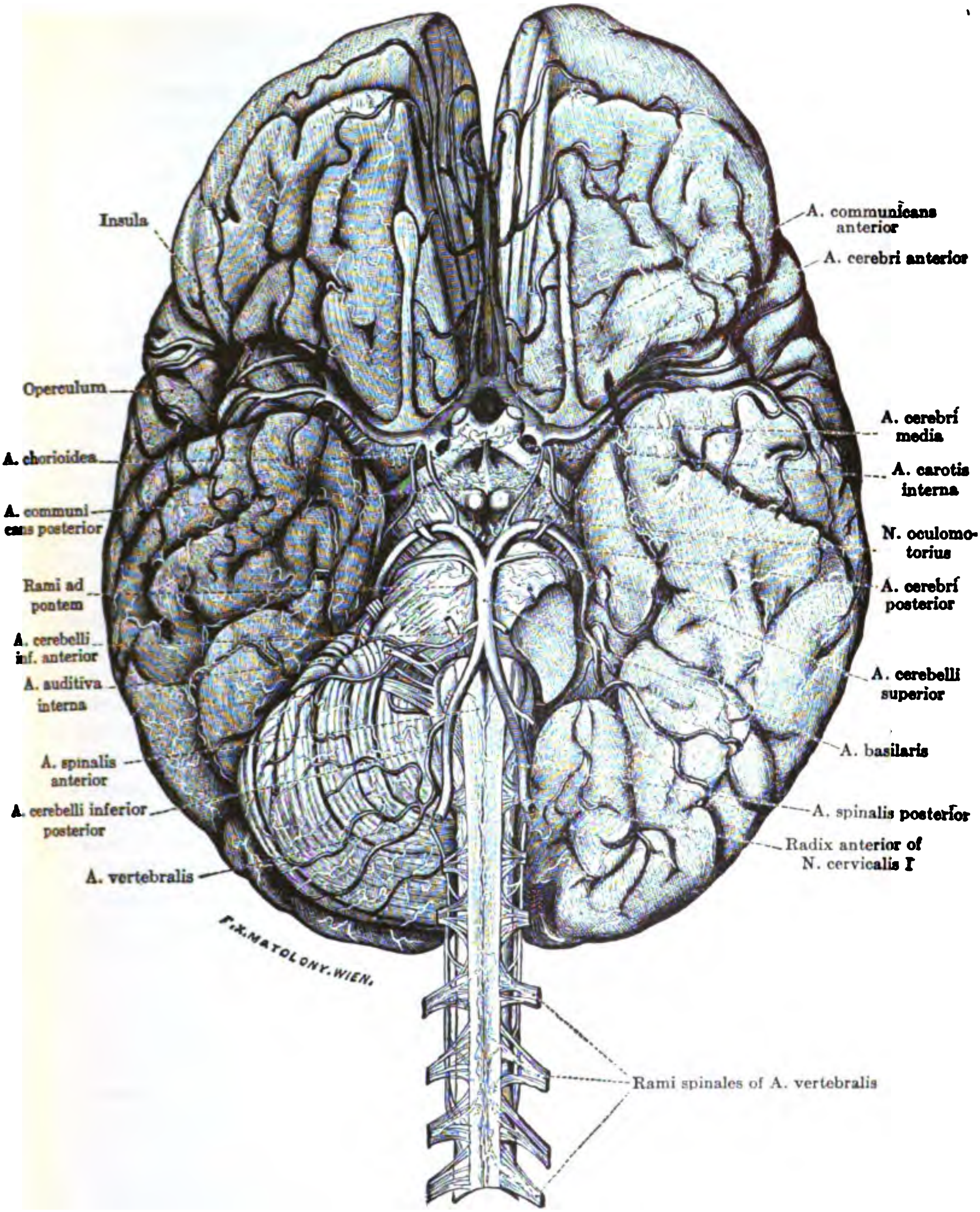

FIG. 18.—THE DISTRIBUTION OF THE ARTERIES AT THE BASE OF THE BRAIN AND THE CIRCLE OF WILLIS.

The frontal lobes have been slightly separated to show the course of the two anterior cerebral arteries as far as the genu of the corpus callosum; the right lateral cerebral fissure has been somewhat widened to show the branching of the middle cerebral artery. On removal of the left cerebellar hemisphere, the distribution of the posterior cerebral artery upon the basal surface of the occipital and temporal lobes becomes visible.

the cortex of the brain coincides in the main with the areas of the cerebral centers, so that, following hemorrhages or embolisms which are not too extensive, isolated symptoms of the absence of definite functions may appear. The most frequent are hemorrhages from branches of the middle cerebral artery into the internal capsule and the basal structure surrounding it, and this produces paralyses, motor irritative symptoms, anesthesia and hemianopsia, also hemorrhages which by rupture into the ventricles are often the cause of death.

We must now devote some attention to the **spinal cord** which develops gradually without a line of demarcation from the medulla, this structure tapering in its downward course, the pyramidal decussation being regarded as the beginning of the cord, therefore that area where the fibers of the pyramids running dorsally cross each other, and, with the exception of the small pyramidal anterior column tracts, extend to the lateral columns. As the nuclei of the posterior columns disappear, the characteristic formation of the spinal cord is seen, a white medullary mantle surrounding the substance which in shape resembles a butterfly and only at the entrance of the posterior roots reaches the periphery. The human spinal cord from the first cervical vertebra to the first lumbar vertebra or, at most, to the upper border of the second, attains a length of from 40 to 50 cm.; the lowest portion of the spinal cord in man, as well as in anthropomorphic apes, is quite undeveloped but is analogous to the tail in animals. The spinal cord in two areas is markedly enlarged showing a cervical enlargement at the fifth or sixth cervical vertebra and a lumbar enlargement in the region of the lowest thoracic vertebræ, both corresponding to the spinal centers for the muscles of the extremities. We differentiate the *cervical cord* with eight cervical roots, the *thoracic cord* with twelve dorsal roots, the *lumbar cord* with five lumbar roots, the *sacral cord* with five sacral roots and, finally, a pair of coccygeal nerves. The sacral cord terminates in the lowest portion of the spinal cord, the *conus medullaris,* which then separates into thin, terminal threads about 25 cm. long, the *filum terminale.* The sulcus longitudinalis ventralis medianus is situated ventrally in the middle, and as the anterior sulcus penetrates into the medullary mass. Corresponding to it is the sulcus longitudinalis medianis dorsalis which is merely a narrow fissure. Alongside of this, near the point where the posterior roots enter, we note the sulcus lateralis dorsalis and a very indistinct sulcus lateralis ventralis at the point of exit of the anterior roots. In consequence of the shortening of the spinal cord which takes place in man the spinal roots must run toward the cauda in order to reach the corresponding intervertebral openings, and the deeper their origin the longer their course. Hence, in the region of the conus medullaris and below it there is a rich sheaf-like collection of nerve roots running downward, the so-called *cauda equina.* The furrows of the spinal cord permit us to differentiate an *anterior* and a *lateral* column, which, however, are only very indistinctly separated from one another, also a *posterior* column. The latter is divided in the cervical cord by a furrow, the sulcus paramedianus dorsalis, into Goll's or the medial column, and Burdach's or the lateral column. The *gray substance* is divided into the anterior horn which contains the larger portion of the motor ganglion cells, and the much smaller posterior horn the apex of which passes close to the periphery, and into the lateral horn which is not everywhere equally developed.

A narrow, *central canal* extends down the middle of the spinal cord, but in adult man this is often obliterated; the anterior and posterior halves of the spinal cord are united by commissures, the anterior white commissure and the

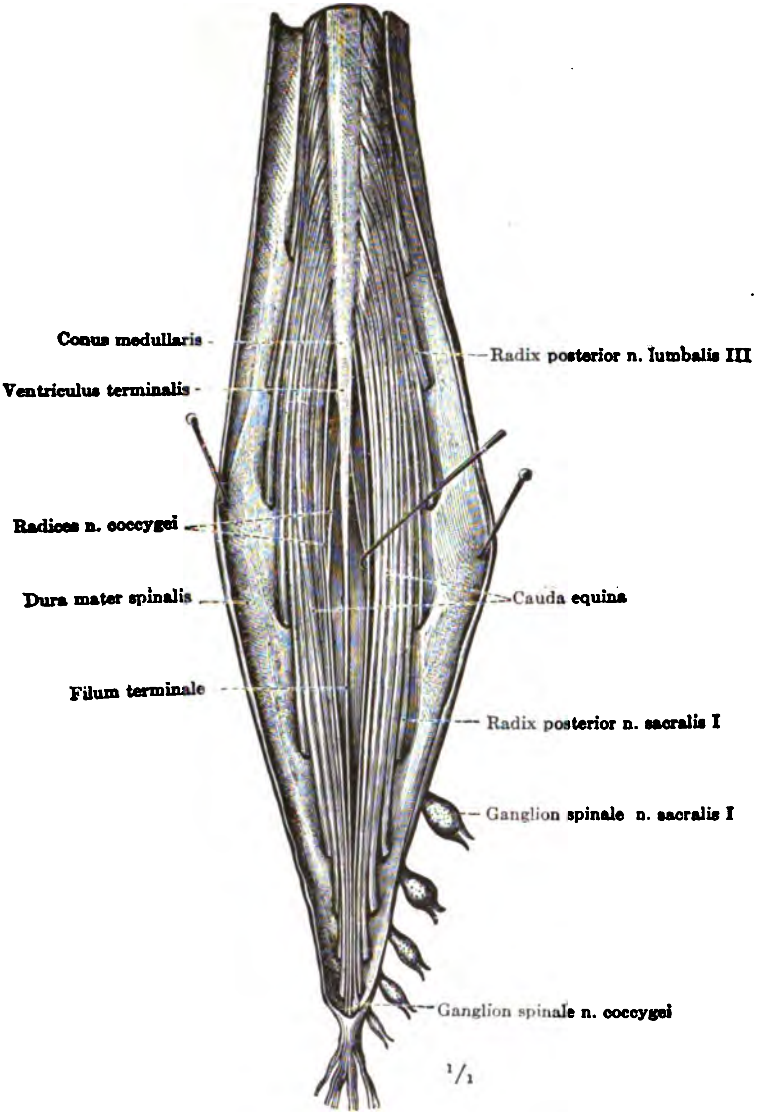

FIG. 19.—THE LUMBAR CORD, PARS LUMBALIS MEDULLÆ SPINALIS, WITH THE CONUS MEDULLARIS, THE FILUM TERMINALE, AND THE CAUDA EQUINA, SEEN FROM BEHIND.

posterior gray commissure, which encircle the central canal. The proportion of white to gray substance differs in almost every transverse section; but the latter appears in greatest bulk in the lowest portions of the spinal cord.

For the finer structure of the spinal cord and its microscopic relations, the reader is referred to the article upon "Histology of the Nervous System" in

this volume, and here we will consider only the **vascular supply of the spinal cord.** This is formed partly by branches of the vertebral, partly by branches of the intercostal, the lumbar, and sacral arteries. At each side a vertebrospinal branch passes down from the ventral artery; these unite in the upper cervical cord and form the anterior spinal artery, which sometimes alone, sometimes as a tract composed of anastomosing branches of the intercostal, lumbar and sacral arteries, extends down to the conus. Thence the arteriæ sulci pass

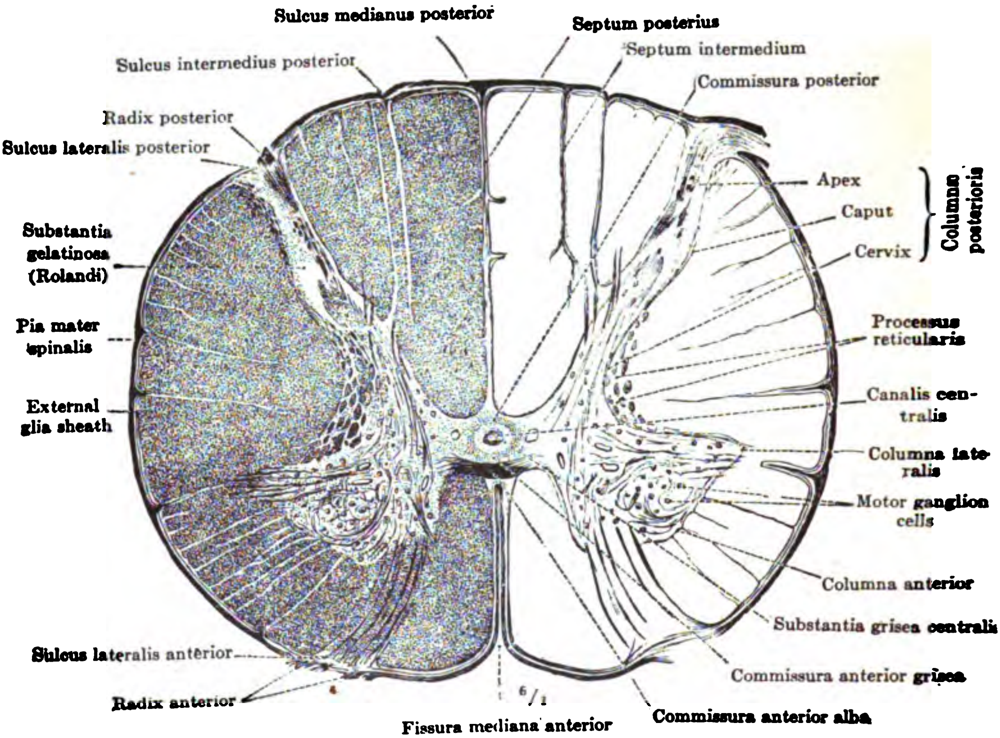

FIG. 20.—TRANSVERSE SECTION THROUGH THE CERVICAL ENLARGEMENT, INTUMESCENTIA CERVICALIS, OF THE SPINAL CORD AT THE POINT OF EXIT OF THE ROOTS OF THE SIXTH CERVICAL NERVE.

through the anterior sulcus into the spinal cord, especially into the gray substance, where they divide as terminal arteries. The dorsal vertebrospinal artery (the posterior spinal artery), which originates from the vertebral and anastomoses with branches of the intercostals, etc., passes along the dorsal side of the spinal cord at each side of the posterior roots. A number of smaller arteries which radiate into the white substance of the spinal cord, and are also terminal arteries, are designated *vasocorona*. Their occlusion produces wedge-shaped infarcts in the white substance, while occlusion of the arteriæ sulci leads to almost complete exclusion of the gray substance.

The shortening of the human spinal cord, and the pushing of its individual segments upward to correspond with the intervertebral openings, is practically important for two reasons. In the first place, the entire canal below the second dorsal vertebra contains no spinal cord, hence without fear of injury

to the latter we can introduce a needle into the vertebral canal through the wide intervertebral openings of the lower lumbar vertebræ and withdraw cerebrospinal fluid for diagnostic and therapeutic purposes. *Spinal puncture,* introduced by Quincke, has already been much practised, and it has lately become possible by this means to inject drugs into the arachnoid sac. Here analgesia produced by injections of cocain soon conquered an extensive field. But in other ways also important advances have been made; above all, by the successful injection of tetanus antitoxin as advised by v. Leyden.[1] Furthermore, exact knowledge of the relations of the individual segments of the spinal cord to the vertebræ has become of vital importance in the surgery of the spinal cord, which has lately developed to an extraordinary extent with an increasing number of successes. Diagrams have been made, especially by Reid, which minutely portray these relations and their variations.

Here we reach a point in cerebral localization, namely, **the relation of the individual portions of the brain, especially of the cortex, to the bony skull,**

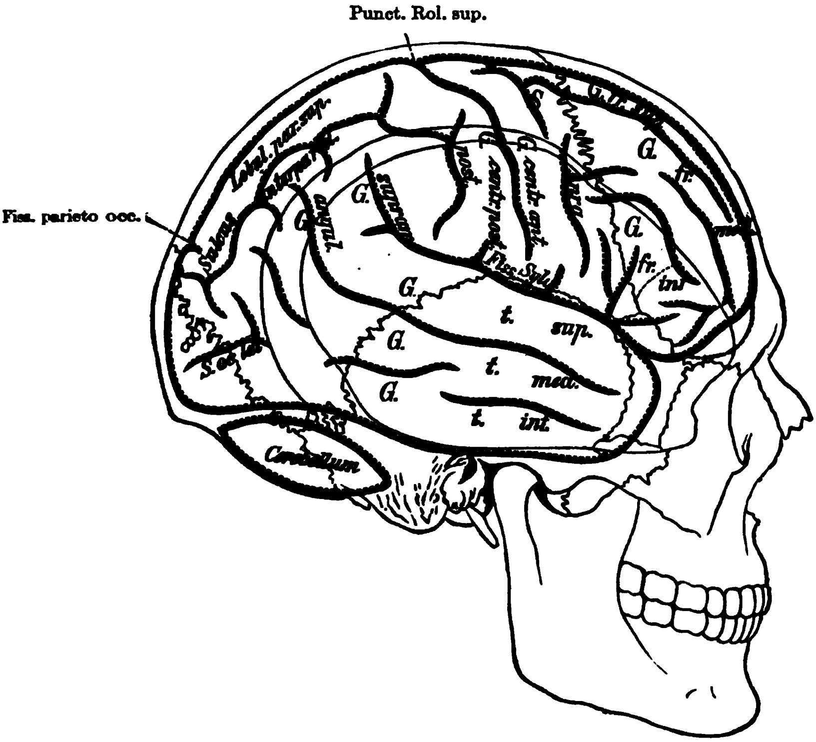

Fig. 21.—Topographic Relations Between the Surface of the Brain and the Skull. (After Thane.)

which, with the increasing importance of cerebral surgery, is rapidly becoming of greater practical significance. The frontal lobe is situated under the frontal bone and extends posteriorly somewhat below the parietal bone; under the latter are the central convolutions, the parietal lobes, and a portion of the

[1] See article "Tetanus" in volume on "Infectious Diseases."

occipital lobes, while the temporal bone covers the temporal lobe, and the highest point of the squamous suture reaches the Sylvian fissure. Under the plate of the occipital bone lie the occipital lobes and the cerebellum. A number of methods have been proposed by which to determine the fissures which should be traced in operations, especially the Sylvian fissure, the central furrow, etc. Waldeyer, in a very instructive article, "The Topography of the Brain," [1] recently described the most important relations of these, combining the chief features in the systems of Thane, Poirier and others. Here I shall cite only the main points enumerated by Waldeyer.

(*a*) During life, the *lower end of the central furrow* may be traced externally on the skull by drawing a German horizontal (a line from the deepest point of the infraorbital border through the highest point of the upper border of the porus acusticus externus) at a right angle from the tragus close behind the jaw and upon this a line extending 5 to 6 cm. upward;

(*b*) The *upper end of the central sulcus* is found by tracing a perpendicular line upon the posterior circumference of the base at the point where the mastoid process intersects the edge of the hemisphere;

(*c*) The *Sylvian point,* the point of division of the Sylvian fossa, is situated at a point 4 to 4.5 cm. perpendicularly above the middle of the zygomatic arch.

(*d*) The *upper end of the Sylvian fissure* corresponds to the middle of the lower border of the parietal tuberosity;

(*e*) The *parieto-occipital fissure* is found on the lambda where the lambdoid and sagittal sutures unite.

Besides the direct topographic relations of the brain and skull an interesting question is, *in how far the development of definite portions of the brain is manifested by the shape of the bony skull,* and in how far an examination of the latter permits us to draw conclusions as to the development of the brain of any particular person.

It is known that, at the beginning of the nineteenth century, the discussion of this point led the great investigator, Gall, to entirely erroneous conclusions; he divided the skull into various regions, and the greater or less development of these areas was supposed to indicate greater or less mental development along certain lines. Even during the lifetime of Gall there was great opposition to this teaching, which actually impaired the value of Gall's otherwise great services, for subsequent exact investigation of the brain and its faculties proved these theories to be absolutely incorrect. Lately Möbius has reverted to Gall's teaching inasmuch as he regards an unusually marked development of the upper external angle of the orbit (which, according to Gall, is the seat for the sense of figures) as a sign of especial mathematical power, as a "mathematic organ," due to an abnormal and increased development of the anterior end of the third frontal convolution. This view which Möbius proposed has not as yet been generally accepted nor has it been confirmed by others. On the other hand Schwalbe, in the Festschrift, issued upon the occasion of Kussmaul's 80th birthday, described the "*relations of the internal and external conformation of the skull*" and has shown that a part of the rilievo of the brain is recognizable upon the external surface of the skull, best in those

[1] *Deutsche med. Wochenschr.*, 1901, Nr. 26 et seq.

parts covered by muscles, but most clearly in the temporal region where, occasionally, four convolutions on the surface of the brain, the lower or third frontal, and the three lateral temporal convolutions may be detected as prominences. But Schwalbe himself expressly warns us not to take this as a basis for new phrenologic calculations, especially as the development of the prominence of the third frontal convolution is by no means an indication of mental power; on the contrary, particularly in the skulls of the lower races, this is usually well developed. Here a lesson for the future appears—we must seek to acquire more accurate knowledge of the relations of the brain to the skull.

We approach the end of our discussion. It is most unsatisfactory to attempt to describe the macroscopic anatomy of the central nervous system within the compass of a short article. If I have succeeded in giving a general picture of the relations here existing, and have interested the practitioner by considering the physiologic importance of this somewhat dry subject, I have fulfilled my object. The structure of the brain can, of course, only become more interesting as our knowledge of these individual tracts and their functional activity increases. All the relations which have been disclosed by experiment and microscopic study have been thoroughly discussed in this article

NORMAL AND PATHOLOGICAL HISTOLOGY OF THE CENTRAL NERVOUS SYSTEM

WITH SPECIAL REFERENCE TO THE NEURON THEORY

By H. ROSIN, Berlin

It is well known that the conspicuously rapid advance of the last few decades in the science of neurology is attributed to a sharper limitation and to an increase of clinical pictures, to a more comprehensive study of their symptom-complex, and, above all, to the important findings which *histology* has added, particularly in the study of the structure of the nervous apparatus. Although our knowledge is to-day still incomplete, nevertheless we have advanced so far beyond the mere composition of the central nervous system that we are enabled to study a number of systems of nerve tracts coursing from the center to the periphery, and to determine the origin and source of stimulation of certain sensory and motor impulses. In pathology, histology has also led us to conclusions regarding anatomical changes which disclose to us the seat of many diseases of the nerves.

By the *comparison* of the histologic finding with the clinical symptom-complex neurology has given us important data; and it may, therefore, be interesting to *review* the normal and pathological histology of the nervous system from the *present standpoint* of scientific investigation, after which many symptoms, many a pathologic picture, will become intelligible.

In the following I shall attempt such a portrayal without, on account of the limited scope of this article, entering further into details; nevertheless, a prerequisite is a knowledge of macroscopic relations, those relations in the structure of the spinal cord and, above all, in the brain, which should be familiar to all physicians from their studies of anatomy and embryology. In regard to these structures views have not materially changed in recent years. The old nomenclature is employed for the cerebral convolutions, for the central ganglia in the interior of the brain, for the cavities and fossæ, for the varying arrangement of the gray and white substance, and these are sufficiently explained in the various text-books upon anatomy and embryology. To any one especially interested in these subjects, I would commend the text-books of Edinger and Obersteiner, which are accurate and excellent treatises. Those who desire to investigate closely the theme under discussion may well study v. Bechterew's "Conduction Tracts in the Brain and Spinal Cord."

I. PRELIMINARY REMARKS: THE HISTOLOGIC COMPONENTS OF THE NERVOUS SYSTEM

The nervous organs are composed of the true *nervous* and of the *gray or vesicular substance* (including the vessels).

The nervous tissue consists of *nerve-cells* and *cell processes.*

The vesicular or cineritious substance—apart from the true connective tissue in the gross coverings, the adventitia, and the actual walls of the vessels—is composed of a peculiar connective substance which, since the time of Virchow, has been designated by the term *neuroglia.*

A. THE NERVE TISSUE

(1) THE NERVE-CELLS

I shall first state what we at present know of the structure of the nervous substance, and I shall begin with the *nerve-cells.* Recently the finer structures of these tissues have been revealed to us as of such exceeding richness and peculiarity that, so far as we know, they may justly be included among the *most complicated* cells of the organism.

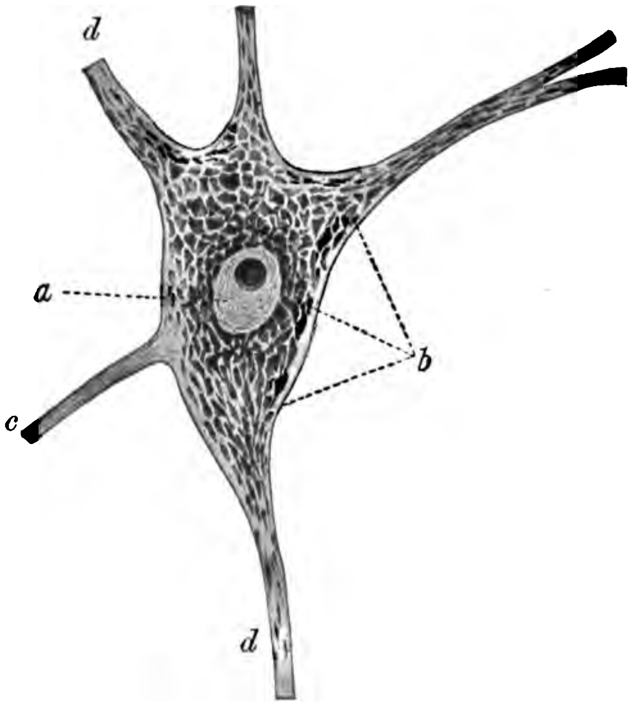

FIG. 22.—NERVE-CELL FROM THE ANTERIOR HORN OF THE SPINAL CORD OF A RABBIT, HARDENED IN FORMALIN-ALCOHOL. Neutral red staining.

Even the *nucleus* of the nerve-cell (Fig. 22, *a*) is characteristic on comparison with the nuclei of other cells; first, by its *size,* particularly in those large cells which are found in the motor cortex of the brain, in the nuclei of the medulla oblongata, and in the anterior horns of the spinal cord. Here the germinal vesicle has the appearance of an ovum. But the nucleus of even the smallest nerve-cells is always relatively large.

The *structure* and the tinctorial property of the nucleus is also typical. In the large cells it is separated from the body of the cell by a sharp contour. On a light background, comparatively

few but dark, more markedly refractive staining bodies are visible, among which one, the *nucleus* (Fig. 22), is conspicuous among the large nerve-cells and resembles the germinal spot of the germinal vesicles. The reaction of the nucleus to certain stains is noteworthy. While cell nuclei have in common the property of taking up basic stains with their chromatin substance (and, therefore, are called nuclear stains), and while the nuclear corpuscle (if present) frequently selects a stain which has an acid property, the reverse of this takes place in the nucleus of the nerve-cell: Its chromatin, scant in amount, shows an affinity for acid stains even when used in neutral staining mixtures; for instance, in staining with triacid, with methylene-blue, eosin, etc.; on the other hand, the nuclear corpuscle (at least in man) stains uniformly and readily with both colors, and reveals to neutral color mixtures a neutrophilic property; in some animals this is even basophilic.

The body of the nerve-cell is, however, much more interesting than the nucleus. Recently attention has been specially called to the so-called Nissl's *bodies* or tigroid substance (Fig. 22, *b*), the importance of which must be pointed out.

Formerly, when the only agent for preserving and hardening the nervous tissue was Müller's fluid, i. e., a mixture of potassium chromate, chromic acid and sulphuric acid, it was impossible to get a clear idea of the structure of the body of the cell even by employing several stains; Müller's fluid makes only the nucleus structurally distinct, rendering the substance of the body of the cell homogeneous. By using *alcohol* for hardening (Nissl), and recently *formalin* which was introduced into histology by Blum, the individual parts of the body of the nerve cell have been revealed. In addition to the old methods of hardening, other *stains* are necessary for the detection of Nissl's bodies. In place of *carmin*, which Gerlach considered most important for staining in general, especially for nerve staining, Nissl used the more powerful *basic anilin colors*. If material that has been hardened, embedded, and cut in alcohol is stained with *methylene-blue soap solution* (or neutral red solution) coarse granules appear in the body of the cell and cover a considerable portion of it, extending almost to the periphery; they cluster around the nucleus in groups which penetrate to some extent into the processes of the cell (Fig. 22, *d*), and, under powerful magnification, are seen to be composed of individual threads and granules which have absorbed the basic stain. Nissl's method, therefore, embraces two different processes, the hardening in alcohol and the utilization of the *basophilia* (Rosin) of the granules in the choice of suitable staining substances.

This appearance of basophilic substances in the body of the cell is quite peculiar, and is not found in other cells of the human body (except in mast-cells and, to a certain extent, in the plasma cells and lymphocytes); it is evident, therefore, that the histologic characteristics of the nerve-cells just described are of *peculiar nature*, particularly shown in their standing and analysis.

Between Nissl's bodies in the substance of the nerve-cell a *basic substance* (Fig. 22) remains which, on staining according to Nissl's method, does not show the color. On staining with different mixtures (the triacid, according to Rosin, the methylene-blue erythrosin, according to Held), in contrast to the granules, it shows a preference for acid colors, upon employing the red in acids and the yellow neutral red in alkalies it becomes yellow, but it shows

no affinity for basic stains (unprepared). In this "oxyphilic" basic substance yellow granules are deposited which stain intensely with fatty staining substances, such as osmium, sudan and scarlet, and which are to be included among the *lipochromes*, i. e., stained fatty substances (Rosin), and in adults these deposited in dense clumps often form a large portion of the basic substance (Fig. 23, *A* and *B*).

The remaining portion of the basic substance was formerly supposed to be structureless, but Apathy and Bethe, by a peculiar process of maceration and subsequent staining, have enabled us to make a differentiation which may possibly be of great importance in the study of the structure of the nervous system and the course of nervous stimulation; this will be considered later. These authors noted that the ground substance is composed of a fine *fibrillary net* which passes through the body of the cell between Nissl's granules. The fibrillæ pass from one process to another through the protoplasm, thence into the fibrillæ (see below) which compose the axis cylinders as well as the dendrites.

The nerve-cell is composed of a small, non-granular peripheral zone (Fig. 22) and a delicate structureless covering; the point where the nerve process originates, as well at its beginning, appears to be free from granules (Fig. 22, *c*) which is in contrast with the dendrites (Fig. 22, *d*); in these statements I have mentioned the most important and the most complicated and peculiar features of the nerve-cell.

(2) THE CELL PROCESSES

Since the advent of microscopy of the nervous system, the *processes* have quite properly been regarded as the important and characteristic constituents of the nerve-cell. The familiar designations, unipolar, bipolar and multipolar nerve-cells, are now almost discarded; formerly every point of exit of a cell

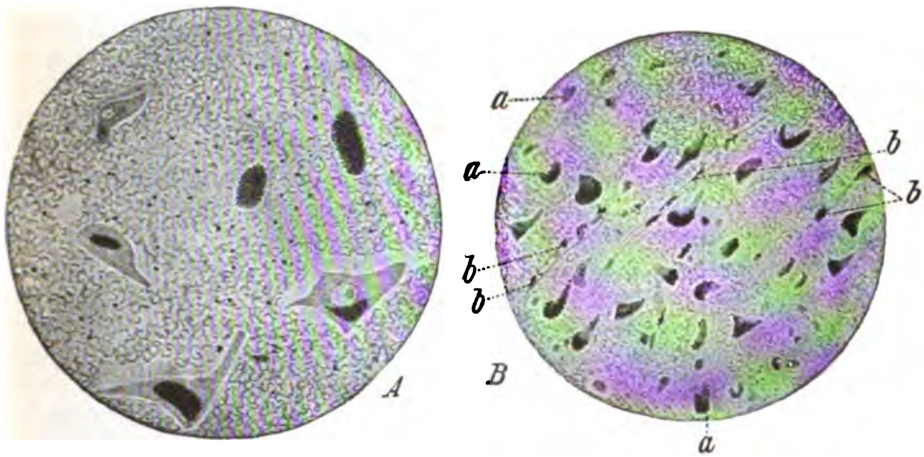

FIG. 23.—*A*, Sudan staining of the anterior horn of the spinal cord (man); *B*, Sudan staining of the cerebral cortex (man).

process was designated as a pole of the nerve-cell. But the teaching of cell processes has recently undergone a change, and is now based upon a more comprehensive foundation, which, in its completeness, at present dominates

the theory of the histologic structure of the central nervous organs, namely, Waldeyer's neuron theory, which will subsequently be explained. I shall now attempt to present briefly the most important of these views regarding the *behavior of the cell processes.*

The importance of suitable methods of staining, which has always been appreciated, is particularly evident in the histology of the nervous system.

It was a matter of paramount importance to find *stains* which would clearly reveal the distribution of the cell processes.

One of these stains is used in the so-called *Golgi method,* with which and with its modifications this author and other pioneers have made brilliant discoveries. Among them we must mention Ramón y Cajal, further van Gehuchten, Kölliker, v. Lenhóssek, and Bechterew. The Golgi process is based upon a sometimes rapid, at other times slow, *impregnation* of the nervous organs (hardened in chromium) with a *silver, gold* or *mercury* salt, and the subsequent reduction of the salt to the metal that was contained in the salt. This metal is deposited externally upon the cells and their processes, where, as a black substance, it gives the structure prominence. A fault in the method, namely, that only certain isolated cells are stained, others remaining uncolored, proves an advantage in the investigation, since, in suitable preparations, instead of a net that cannot be disentangled, a few cells only with their entire processes become distinctly visible.

A second method enables us to stain cells and their processes throughout their entire extent. This is designated as *vital methylene-blue staining.* It was introduced by Ehrlich. The methylene-blue solution is either injected into the living animal until the tissue of the nervous system is saturated with the color (usually resulting in a white reduction product which again turns blue on exposure to the air), or into "tissue kept alive outside of the body," i. e., the tissue of freshly killed animals, until a definite intensity of staining is attained. As the color is prone to disappear rapidly, it is retained by fixation; for example, with ammonium picrate or ammonium molybdate. Retzius, Dogiel and S. Meyer have successfully used this Ehrlich stain which requires considerable practice.

By these methods the relation of the nerve-cell to its processes has been revealed as follows: Every nerve-cell of the central nervous system has a process which, as a rule, is subsequently medullated (always when it leaves the gray substance, but in the sympathetic ganglia the majority of the nerve processes are non-medullated), the so-called *nerve process* (formerly axis-cylinder process) (Fig. 22, *c*; Fig. 24, *b*; Fig. 25, *). The other processes, the number of which varies greatly in the different cells, are called dendrites (formerly protoplasm processes). They are usually thicker than the nerve processes, are never medullated, and are never as long as the nerve processes (Fig. 22, *d*; Fig. 23, *a*).

While the nerve process usually extends forward horizontally from the cell (Fig. 25, *, 26, *a*, and 27, *a*) and gives off many lateral branches (*collaterals*) which pursue a varying convoluted course (Fig. 26, *b*), the dendrites usually soon bifurcate, occasionally into extraordinarily dense *tree-like branches* (Fig. 24, *a*, 25, 26, *c*, and 27, *c*) (for example, the pyramidal cells of the cerebral cortex, Purkinje's cells of the cerebellar cortex). This branching of the dendrites *finally ceases, and they either have free terminations,*

being contiguous to similar terminal branches of other dendrites, or they are *wrapped basket-like* around other nerve-cells and their processes. The nerve process itself has a varying length; sometimes it is quite short, and after giving off many or few collaterals it divides near its origin into *terminal twigs similar* to the dendrites. Thus it is brought into contact with other cells and terminal twigs, and the same occurs with its collaterals. Often, however, the nerve process is of great *length*, it traverses wide areas of the brain or spinal cord, constantly giving off collaterals, often extends from the brain into the spinal cord, or leaves the central nervous system and passes by peripheral nerves to those organs which it supplies. Such nerve processes, for example, the fibers of the cerebral

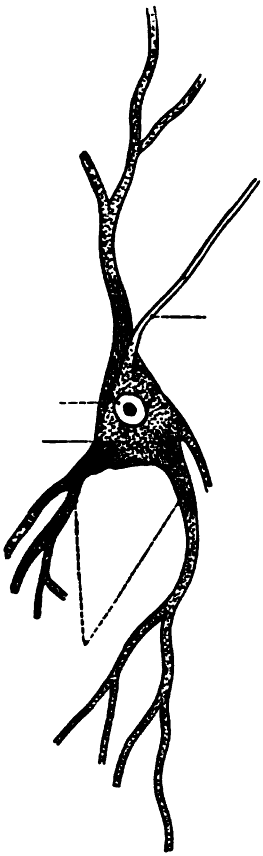

Fig. 24.—Motor Anterior Horn Cell with its Processes. (After Toldt.)

Fig. 25.—Ganglion Cell of the Anterior Horn, Golgi Staining. (After Lenhóssek.)

nerves or those of the anterior and posterior roots, often have a length of many centimeters. Those nerve processes which extend to the periphery, for example, the motor nerves, terminate in the muscle (Fig. 19) by splitting into minute primitive fibrillæ. The sensory fibers have a similar termination. The dichotomic division appears to be characteristic for the termination of all nerve processes.

The course of the nerve process, therefore, may be almost microscopically short, or of extraordinary length considering its minute size at the point of origin.

Those who remember former views regarding the branching of processes of the nerve-cells must recognize the significance of the decided change in view. The nerve process histologically differs from the dendrite. Often it alone is destined for a long course, it alone is medullated, it never unites with another cell, as was once believed, but invariably terminates by splitting into fibrillæ. The old scientific principle of the *indivisibility* of nerve fibers has been forever abrogated. Even the so-called unipolar nerve-cells of the spinal ganglia (Fig. 20, *a*) show a division of the nerve process: Shortly after its exit from the cell it divides T-shaped into two long branches, one of which becomes the posterior root fiber (Fig. 20, *b*), the other the peripheral sensory nerve fiber (Fig. 20, *f*) (see below).

In the main there has been no addition to our knowledge of the structure

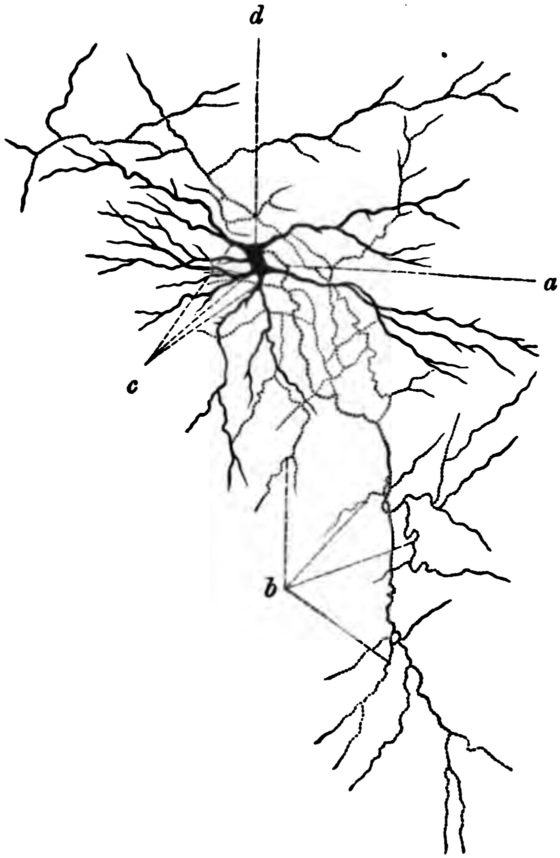

FIG. 26.—SENSORY NERVE-CELL. (After Golgi.)

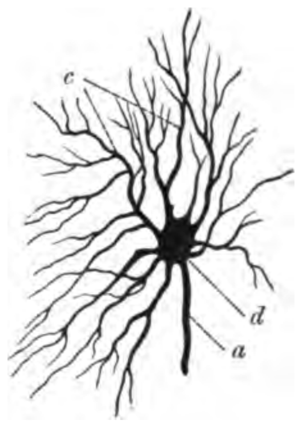

FIG. 27.—NERVE-CELL OF THE SYMPATHETIC.

of the *peripheral nerve fibers*, but the sympathetic system has not yet been thoroughly studied in man. Here comparatively little has been added to the knowledge acquired in the earlier microscopic period. What Ranvier, Remak and others believed is to-day still accepted.

We must now briefly discuss the histology of the peripheral nerve fibers. In the interior is the axis cylinder (Fig. 30, *c*), an *albumin substance*, the *fibrillary* structure of which was once doubted but is now acknowledged. The axis cylinder (axon), which is not visible in the fresh structure, has an especial affinity for acid stains (oxyphilia). It is surrounded by the *myelin sheath* (Fig. 30, *b*) which, traversed by the delicate *neurokeratin structure*,

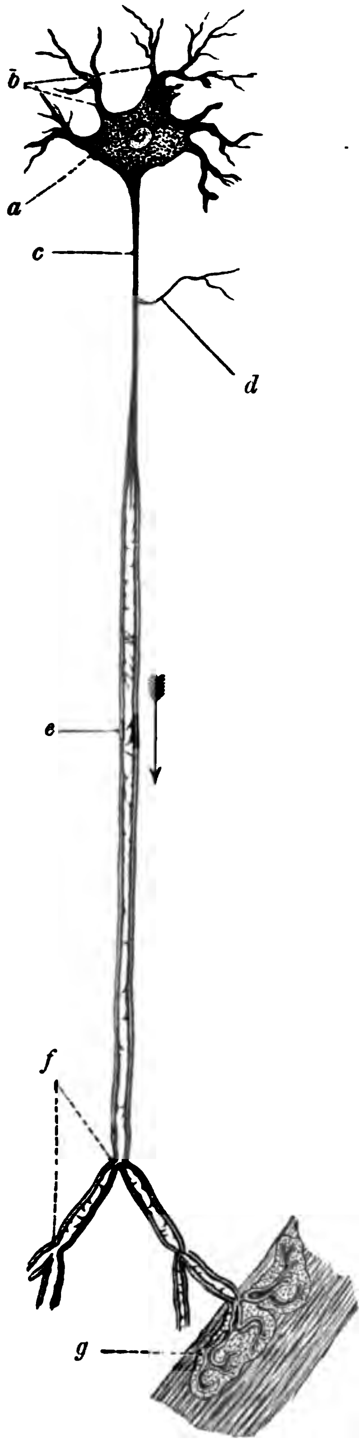

FIG. 28.—DIAGRAM OF A MOTOR NEURON; FIRST DIVISION. (After Toldt.)

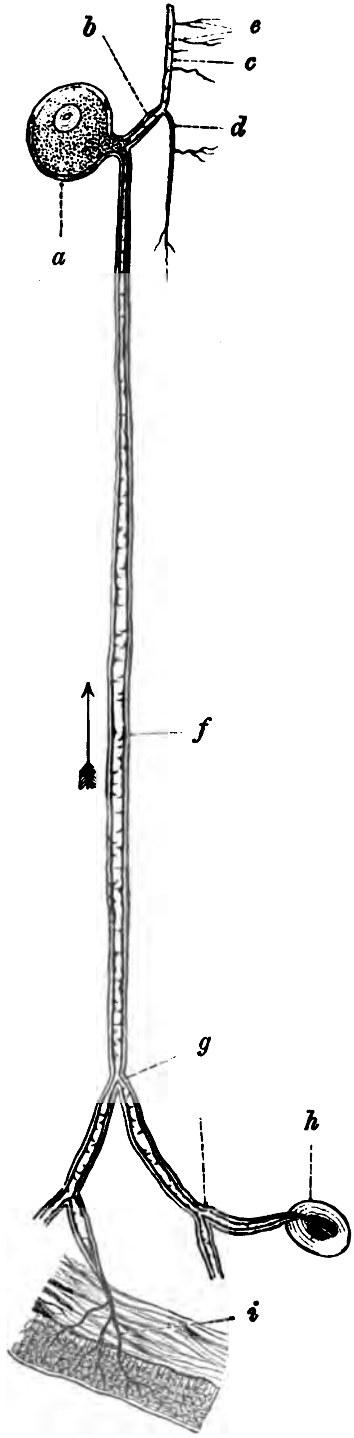

FIG. 29.—DIAGRAM OF A SENSORY NEURON; FIRST DIVISION. (After Toldt.)

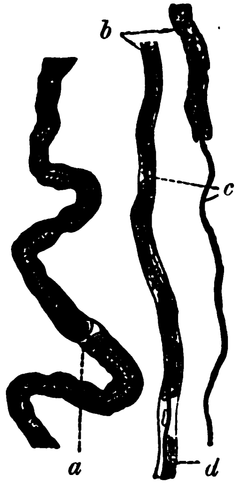

FIG. 30. — MEDULLATED NERVE FIBER, MACERATED IN MÜLLER'S FLUID.

consists of semi-fluid myelin, and is probably identical with or closely related (Wlassak) to Liebreich's *protagon.* The medullary sheath is surrounded by Schwann's *sheath* (Fig. 30, *d*), a structureless covering which contains oblong *nuclei.*

The medullary sheath shows here and there *deep annular constrictions,* Ranvier's nodes (Fig. 30, *a*), which almost completely divide it. Schwann's sheath covers the axis cylinder which is somewhat decreased in size. The nucleus is situated between these two constrictions in Schwann's sheath, and the medullary sheath at brief intervals shows clefts which are known as Lantermann's segments; these pass obliquely almost through the myelin, thus forming furrows one above another. The limits of this chapter upon the *central nervous system* do not allow us to discuss the peripheral termination of the nerves in the muscles, skin, internal organs, and the organs of special senses.

B. THE SUPPORTING SUBSTANCE. (THE GRAY OR VESICULAR SUBSTANCE.)

The soft, nervous pulp is held together by a peculiar, widely branching basic tissue. Only the coarsest fibers originating from the pia mater (Fig.

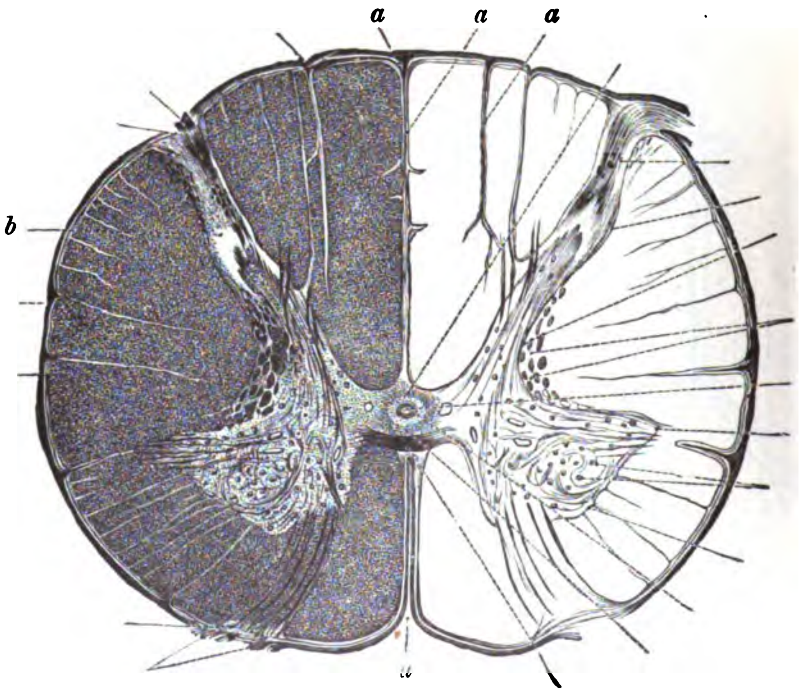

FIG. 31.—TRANSVERSE SECTION THROUGH THE CERVICAL ENLARGEMENT. (After Toldt.)

31, *b*, and 32, *b*) and from the vascular sheaths of the arachnoid (Fig. 32, c) which form septa between the convolutions and folds (Fig. 31, *a*, Fig. 32, *b*, and Fig. 32, *g*) are formed of *connective tissue*; these are carried from the larger vascular trunks a certain distance within the gray substance. The *vessels* themselves with their walls, and even the capillaries, form a trabecular structure which is not without importance.

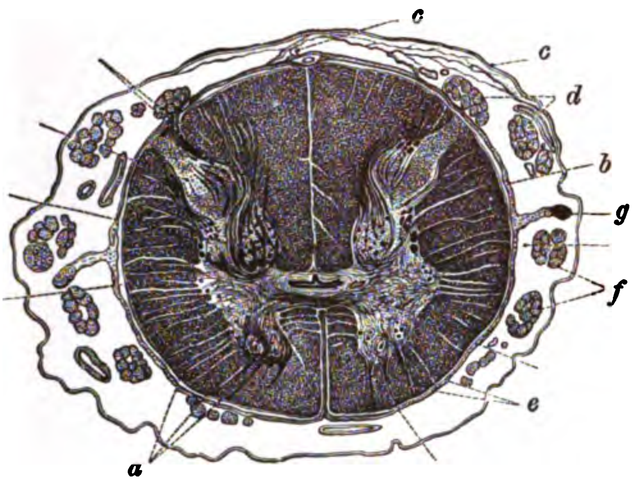

Fig. 32.—Transverse Section through the Upper Lumbar Cord. (After Toldt.)

The chief support is given by a specific substance, the glia tissue. This has a characteristic structure and a peculiar tinctorial property. Weigert, and subsequently other authors (especially Benda), succeeded in isolating the glia tissue from other tissues, especially the connective, and staining it. Like the *nerve substance originating from the ectoderm* (according to His, neuroblasts and spongioblasts), the neuroglia presents *embryologically* a compact net of trabeculæ which vary greatly in their caliber as well as in their density and arrangement. A thickly woven network of trabeculæ surrounds the brain and spinal cord, another covers the walls of the ventricles and the central canal of the spinal cord. It forms a network (Fig. 33) in the gray substance that surrounds the nerve-cells and their processes. The glia of the white substance is less dense; it passes between the nerve tubules in a loose fold, encloses isolated bundles (Fig. 34) of these, and to some extent penetrates into the anterior and posterior nerve roots.

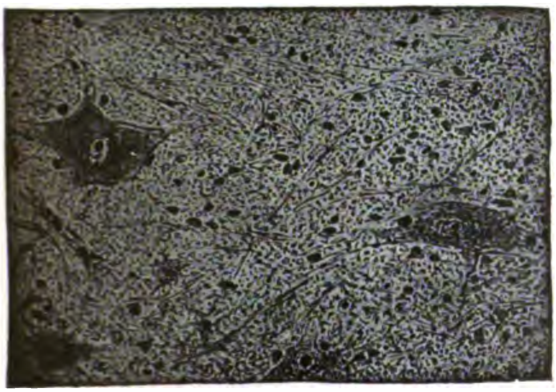

Fig. 33.—Transverse Section through the Normal Gray Substance of the Anterior Horn of the Spinal Cord.

Many of the nuclei which are sometimes surrounded by a layer of protoplasm are deposited in the midst of the glia net, preferably in the white substance, where several glia trabeculæ cross each other. It is still a mooted question whether these fibers unite with the glia cells (glia nuclei, Deiter's cells, spindle cells) (Fig. 36),

or only pass along side them. But the adhesion of glia fibers to connective tissue septa and vascular sheaths has been proven.

II. THE GROUPING OF THE NERVOUS SUBSTANCE AND THE NEURON THEORY

From the preceding description of its elements, it is apparent that the tissue of the nervous system is of very complicated composition, of which we have now obtained a somewhat clear idea.

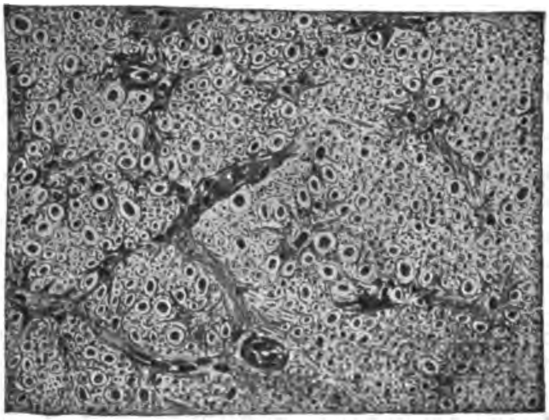

Fig. 34.—Transverse Section through the Normal White Substance of the Spinal Cord.

The majority of the nerve-cells are found only in the gray substance, rarely in those areas where the gray and white matter are intimately admixed (in the medulla oblongata, in the pons, in the mid-brain and inter-brain). The white substance itself contains none of them. In many areas of the gray substance the nerve-cells are arranged in *groups*.

In the spinal cord, especially in the dorsal portion, segmentation may be recognized from above downward, a metameric arrangement. In a horizontal section there is seen a threefold grouping of the anterior horn cells, especially in the cervical cord, to which is added the cell group of Clarke's column in the thoracic cord. There are also numerous nerve-cells which are apparently distributed irregularly throughout the gray substance of the spinal cord.

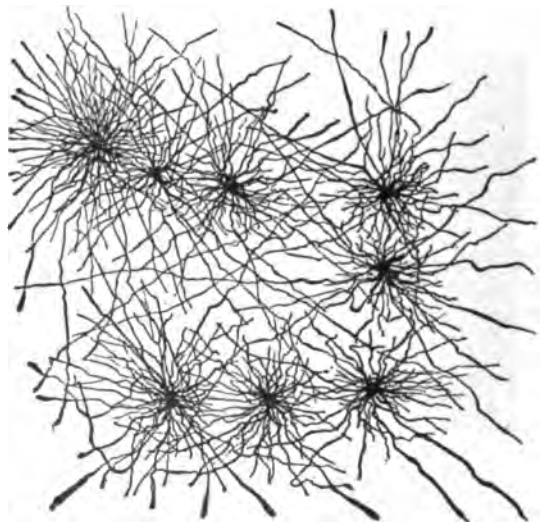

Fig. 35.—Glia Cells (Spindle Cells). (After Kölliker.)

The entire cortex of the brain shows the nerve-cells arranged in *layers*, which differ from each other in shape and size. The nerve-cells accumulate near the origin of the nerves of the brain, also in its central ganglia and in the gray nuclei of its trunk. The majority of these send nerve processes from the gray substance into the white; a few nerve-cells, usually the smaller

ones whose nerve processes are very limited in extent, remain wholly within the gray substance.

As the nerve-cells are partially grouped and partly isolated, so also the nerve fibers of the white substance either unite with well known nerve tracts and form columns, or pursue their own paths which are still unknown.

The *columnar nerve tracts* within the central nervous system pass from one structure to another by longer or shorter paths. At certain points upon the same side of the brain and spinal cord they unite with one another (*association system*), or at corresponding points on different sides (*coördination system*), or they pass caudalward from the cortex of the brain to the central ganglia, or even lower down into the spinal cord (*projection systems*). Some of the latter unite directly or indirectly with those coming from the brain and spinal cord and with the nerves extending into these structures. These are the *centrifugal* and *centripetal fibers,* among them the motor and sensory, the paths of which are best known, while we know little of the course of numerous other tracts, particularly of those which lead to the internal organs, nor of the vasomotors, many of which pass to the sympathetic, and reflex tracts.

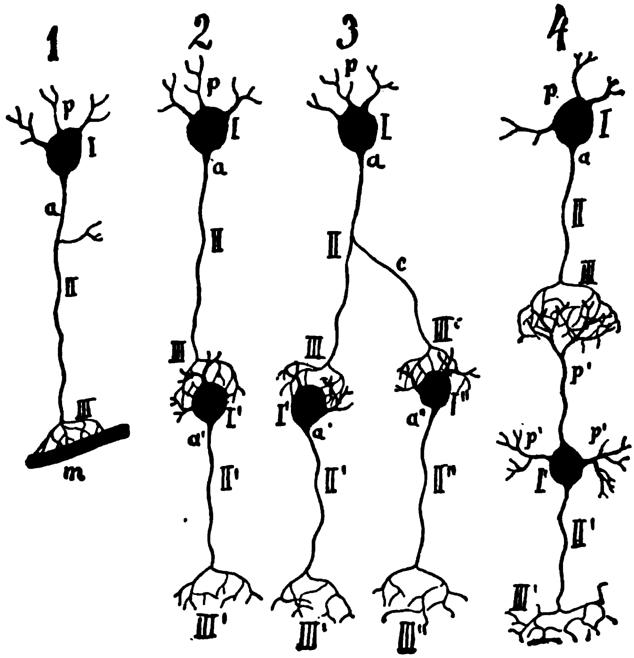

FIG. 36.—DIAGRAM OF THE STRUCTURE, THE COURSE, AND THE BRANCHING OF MOTOR NEURONS OF THE FIRST AND SECOND DIVISIONS. (After Obersteiner.)

If, in the description of the normal course as well as of the pathological changes of the nervous tracts, some areas are more or less obscure, I shall endeavor in the following to describe briefly all that has thus far been discovered in neurology, all that has become common knowledge and is of general interest. My description will necessarily be incomplete.

First, however, I must state the fundamental principle regarding the structure of the nervous system which is generally accepted to-day and is designated as the *neuron theory.* Previously, as a hypothesis rather than based upon histologic investigation, the conception of the relation of individual nerve tracts to each other differed little from our present view.

By the term *neuron we understand the totality of a nerve-cell and all of its processes* (Fig. 36, I). All nervous processes, all dendrites including their branches and collaterals, are merely parts of the body of the cell, the totality

(Fig. 36, I, II, III, Fig. 28, and Fig. 29) therefore forming a *histologic and functional unit.* The nerve is so intimately connected with the cell that it perishes if detached from it or if the cell dies. Inversely, the nerve-cell sustains injury if robbed of important parts of its processes, and signs of degeneration subsequently appear, with permanent atrophy unless there is regeneration of the fiber.

The *individual neurons* of which the entire nervous system is composed are in such close *contact* that, as a rule, the terminal twigs of one neuron closely approach the cell body of the other or surround it (Fig. 36, *2* and *3*). It is possible that the terminal branches of the two neurons approach so closely as to be contiguous (Fig. 36, *4*).

The long tracts of the nervous system are always composed of several neurons. Thus we differentiate between *neurons* of the *first, second,* and *third division, etc.,* and the *peripheral neuron* is invariably designated as the first.

In the motor tract we usually differentiate only two neurons (Fig. 36, *2* and *3*), in the sensory at least three (Fig. 36, *4*). Other associated and coördinated tracts within the central nervous system may be composed of still other groups of neurons, but of this we have no accurate knowledge.

III. THE FIBRILLÆ THEORY. (NEURO-FIBRILS.)

That the neuron theory is based upon exact data histologically obtained by the Golgi method has already been stated. That, so far as we are aware, it sufficiently explains the structure of the nervous system, its "fiber systems," the course and the communication of its tracts, and, above all, certain of its histologic changes in disease, will perhaps become evident in our later description. Notwithstanding all this, I must here at least mention the most recent theory of the histologic composition of the nervous substance, since it is based also upon microscopic findings which its founders (Apáthy and Bethe) repeatedly demonstrated. By a peculiar process of hardening, maceration, and staining, these authors succeeded in separating the *basic substance of the nerve-cells as well as their processes into fibrillæ.* They made preparations which showed that the fibrils pass uninterruptedly from the nerve fibers through the body of the cell into the dendrites, that they merely pass through the body of the cell, not stopping there, and that subsequently, by the communication of their processes, they run through other nerve-cells without terminating there. These histologic findings, if applied to the guiding nervous substance, must vitiate the law of the discontinuity of neurons and *nerve fibers, that is, the fibrillæ, and must be assigned a substantive position which, according to the neuron theory, is in sharp contrast with their nonuniformity.* In consequence, the terminal ramifications are said not to terminate free in front of the cell but, as was maintained some years ago by Held, to enter into the body of the cell or into the interior of the processes which it surrounds, there to unite with the fibrillæ. Hence, nerve stimulation must necessarily pass through an uninterrupted fibrillary substance, and the nerve-cell is relegated to a very subordinate position, much more so than that it held prior to the development of the neuron theory. I think it necessary to mention this fibrillæ theory, but we must await further investigations

before substituting it for the neuron theory, the conceptions of which, obtained by means of very subtle researches, are entitled to the closest consideration.

The majority of neurologists are at present decidedly inclined to adhere to the neuron theory, although in a somewhat modified form.

IV. NEURON SYSTEMS AND NEURON DISEASES

Our knowledge of the structure of the nervous system has not yet reached a point which enables us systematically to divide the countless, intertwining nerve fibers, to assign to each of these its definite path, and to every tract its cellular origin and its termination. This has been possible to only a com-

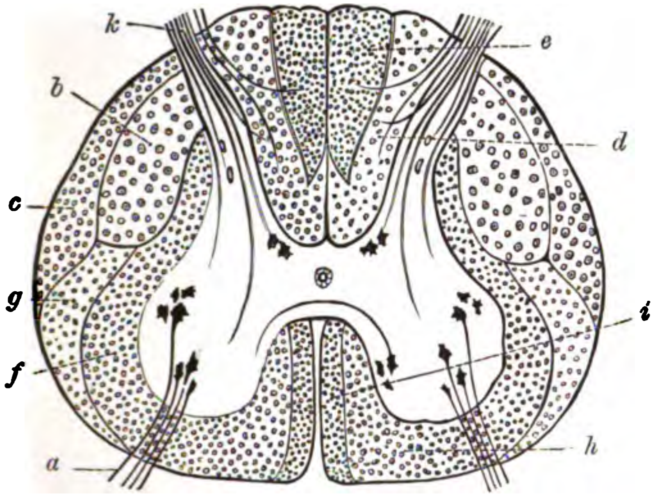

FIG. 37.—DIAGRAM OF THE CONDUCTION TRACTS OF THE WHITE SUBSTANCE OF THE SPINAL CORD.

paratively slight extent. Here we follow the neurons through their entire course, and here we are enabled to recognize certain pathologic changes principally confined to the individual neurons affected, and which are therefore appropriately called the *neuron diseases.*

We shall first consider the *normal relations* of the neurons.

At present but two of the large and important neuron systems have been comprehensively studied: The centrifugal motor and the centripetal sensory tracts including the paths of the organs of special sense. Embryologic investigation, the animal experiment (severing and degeneration), and, finally, microscopic investigation of the pathologic changes by approved methods among which those of Weigert and Marchi are in the front rank, have given us the results at present attained, and by these means we may hope to achieve additional successes.

A. THE MOTOR TRACT

(1) THE FIRST MOTOR NEURON

(*a*) *The First Motor Neuron in the Spinal Cord*

The *anterior roots* of the peripheral nerves spring from the spinal cord at either side of the anterior longitudinal space (Fig. 31, *c*, Fig. 32, *a*, Fig. 37, *a*, Fig. 38); they are accompanied for a short distance by a glia sheath which after passing through the soft membrane (Fig. 40, *a*) is separated from them by a connective tissue sheath. This extends along the anterior side of the spinal ganglion (Fig. 41, *b*) without entering it (Fig. 41, *a*) and, in common with the posterior sensory root, passes a sheath of the dura (Fig. 40, *b*) and, while giving off fibers to the sympathetic plexus, assumes the part of a mixed nerve (Fig. 41, *c*) and proceeds to the peripheral muscles which it supplies.

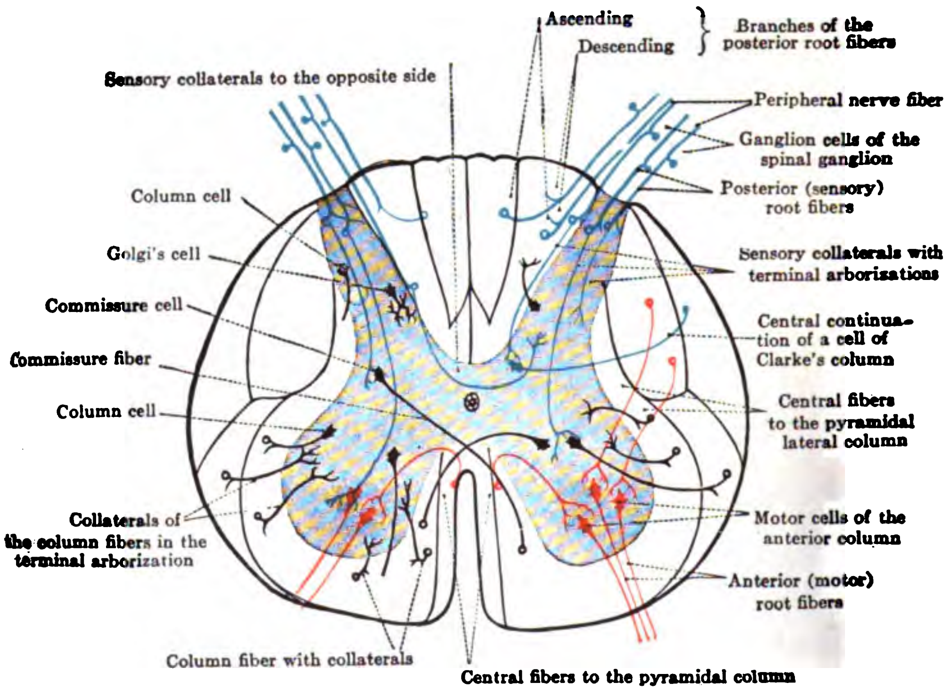

FIG. 38.—DIAGRAM OF VARIOUS TRACTS AND NEURON SYSTEMS IN THE SPINAL CORD.

It has been determined that the majority of fibers in the anterior roots, principally motor nerve fibers, run to the muscles (vasomotors?) but we are not certain that this is the case with all. We must first follow the central path of the motor anterior root fibers to the cells from which they originate, and of which, according to the neuron theory, they are constituents.

Tracing the anterior roots backward, they are seen to *pass through the white substance* with a slight ascent, isolated by their own medullary sheath they proceed between longitudinally rising nerve fibers which extend to the

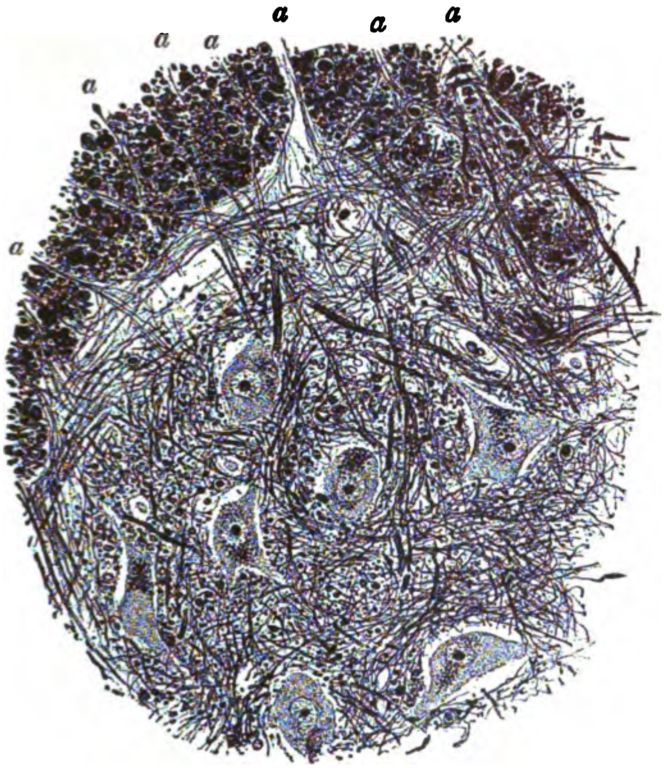

FIG. 39.—FROM THE ANTERIOR HORN OF THE GRAY SUBSTANCE OF THE SPINAL CORD. (After Kölliker.)

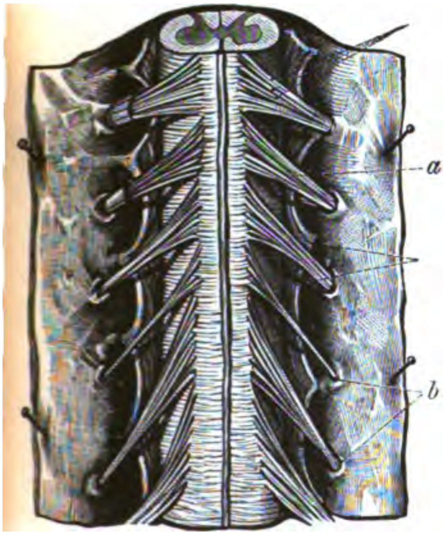

FIG. 40.—THE ANTERIOR ROOTS AND THEIR RELATIONS TO THE MEMBRANES. (After Toldt.)

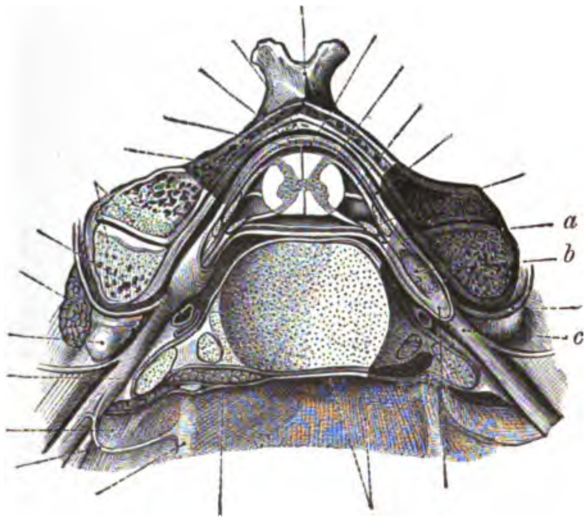

FIG. 41.—TRANSVERSE SECTION THROUGH THE INTERVERTEBRAL DISC, BETWEEN THE THIRD AND FOURTH CERVICAL VERTEBRÆ. (After Toldt.)

anterior columns, and pass to the lateral column at their boundary (Fig. 37, *a*, Fig. 38, Fig. 39, *a*). Thus they reach the *anterior horns of the spinal cord,* and prove to be nerve processes from some of the *large anterior horn cells* situated there (Fig. 39), particularly from the anterior median group (Waldeyer). It must be remembered that in the anterior horns there are many cells which have nothing in common with the anterior roots, and which, as Ramón maintains, belong to altogether different neurons.

The arrangement of the *first motor neuron* of the spinal cord is therefore simple and readily comprehended. It is composed of a number of large anterior horn cells with their dendrites, and of anterior roots which emerge from the lateral motor nerve. At the points of cervical and lumbar enlargement the number of neurons is much greater, because there is a larger number of muscles to be supplied than is the case in the thoracic cord.

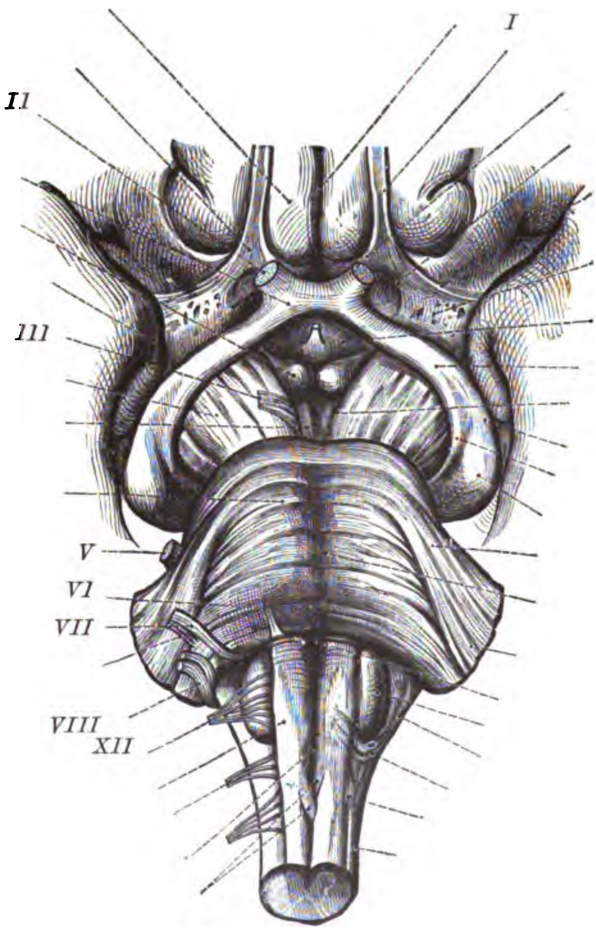

Fig. 42.—Medulla Oblongata, Pons, Peduncles, and Adjacent Parts Seen from the Base. (After Toldt.)

(*b*) *The First Motor Neuron in the Brain*

The origin of the first motor neuron of the brain is somewhat more complicated. Here we must first determine the *origin of the motor cerebral nerve*, the *nuclei* of which are identical with the neuron cells under consideration, and are similar to the anterior horn cells of the spinal cord. I take it for granted that the peripheral course of the nerve fibers is understood, and shall refer merely to the fact that the following nerves will be considered (Figs. 42 and 43): The oculomotor nerve (the third nerve), the trochlear (the pathetic or fourth nerve), the abducens (the sixth nerve), the facial (the seventh nerve), the spinal accessory (the eleventh nerve), and the hypoglossal (the twelfth nerve). These, as is well known, are purely motor nerves. In addition are the mixed nerves, the trigeminal (fifth nerve), the glosso-pharyngeal (the ninth nerve), and the pneumogastric (tenth

nerve). We know of the last three that they possess a motor root besides their main sensory root. We will now discuss the origin of this first motor tract of the cerebral nerves in so far as it has been revealed by recent investigations.

We seek the origin of the oculomotor nerve toward the top of the brain, but still in that portion which we designate the *middle brain*; here also we

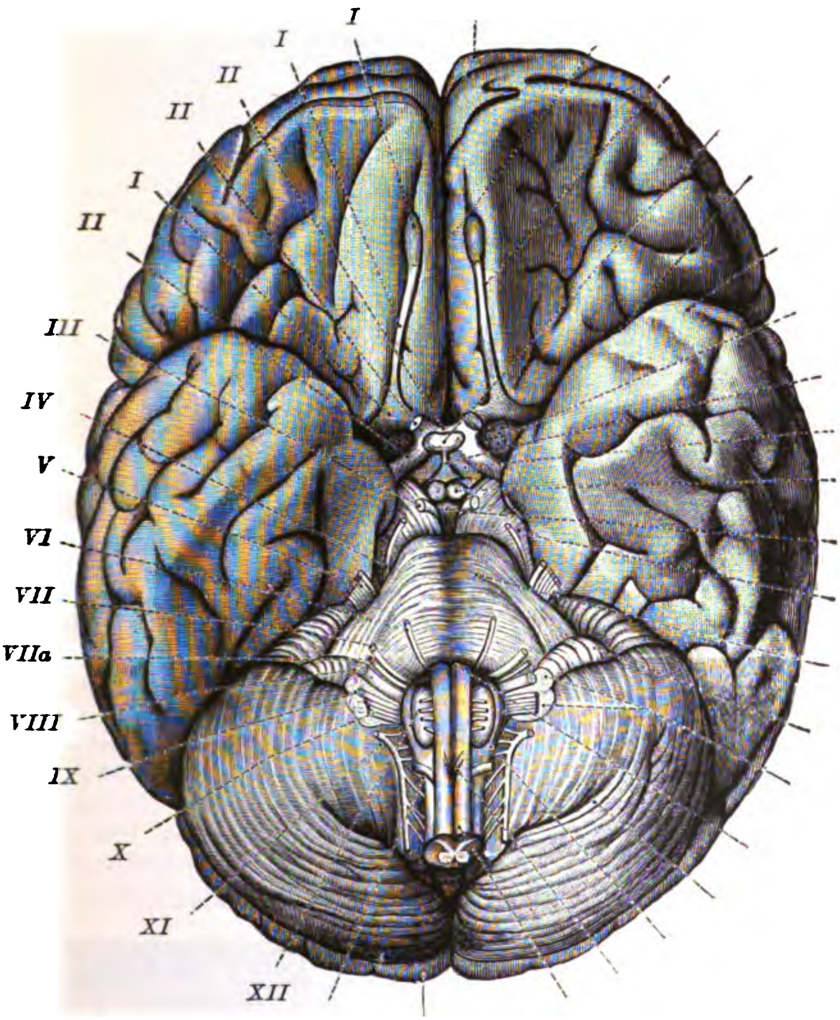

FIG. 43.—BASE OF THE BRAIN AND CEREBRAL NERVES.

look for its chief divisions in man, the corpora quadrigemina (see Figs. 44 and 45) above, the fissure of Sylvius below, and beneath these the fiber masses of the superior peduncles of the cerebellum and of the pedunculi cerebri (Figs. 46, 47, 48, 49). Its nuclei (neuron cells) lie along the entire length of the floor of the aqueduct of Sylvius, from its frontal origin almost to its caudal end;

the nuclei of the *trochlear* nerves are situated posteriorly where the aqueduct opens into the fourth ventricle. We differentiate several groups of

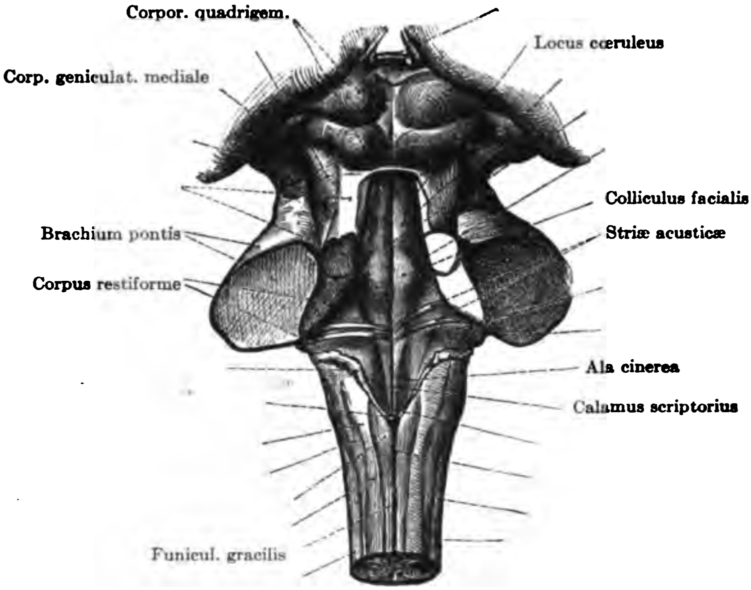

FIG. 44.—PROLONGED CORD WITH THE RHOMBOID FOSSA AND CORPORA QUADRIGEMINA FROM ABOVE. (After Toldt.)

oculomotor nuclei, the anterior small-celled nucleus, the large-celled, the long posterior nucleus, and, finally, that situated in the median line, the medial nucleus. The nerve processes of all these cells converge there, *without crossing*, and form the oculomotor nerve. The processes of a small proportion of the dorsal cells of the posterior nucleus cross to the other side. Clinical observations and autopsy findings show that the centers for individual ocular muscles have been approximately deter-

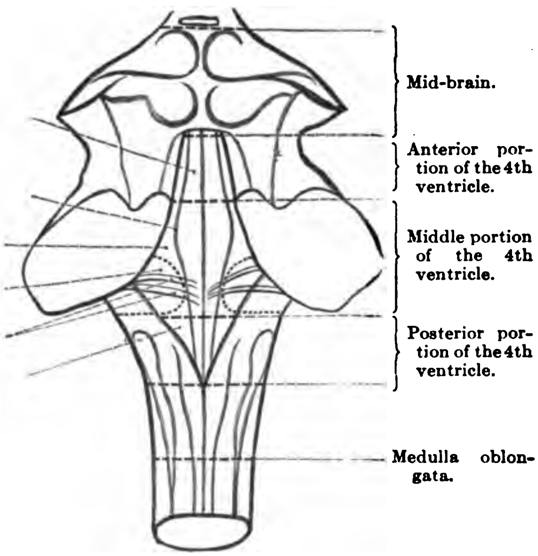

FIG. 45.—DIAGRAM OF FIG. 44. (After Toldt.)

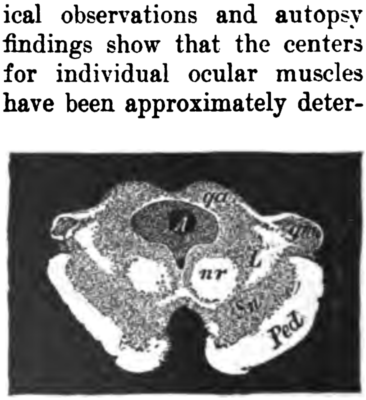

FIG. 46.—SECTION THROUGH THE ANTERIOR CORPORA QUADRIGEMINA.

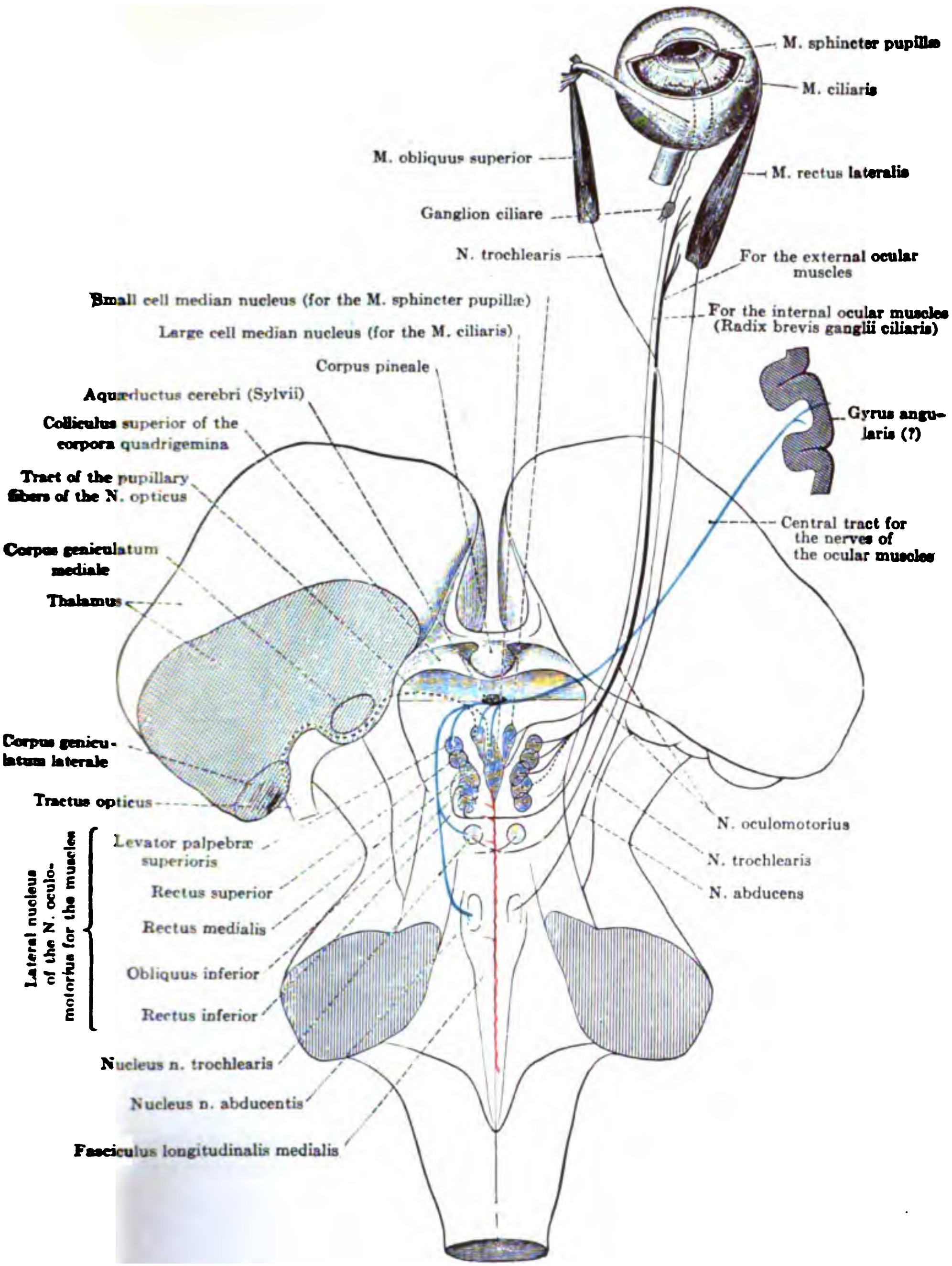

FIG. 47.—The nuclear origin of the oculomotor and trochlear in the middle brain; their central tract (blue) and their union with each other, as well as with the nucleus of the abducens through the median longitudinal bundle (red). The division of the nucleus of the oculomotor in the lateral principal nucleus, the small-celled median nucleus, and the large-celled median nucleus; the localization of the different fiber groups of the oculomotor in this nuclear area. The central course of the so-called pupillary fibers of the optic nerve (tract for the reflex contraction of the pupil). (Diagram based on Bernheimer's description.)

mined, the reader being referred to the works of Edinger, Westphal, Pick and Starr. The united fibers (among these a few crossed ones also) pass transversely through the tegmentum of the cerebral peduncles, ventrally downward, and subsequently appear as a nerve upon the inner side at the base of the brain

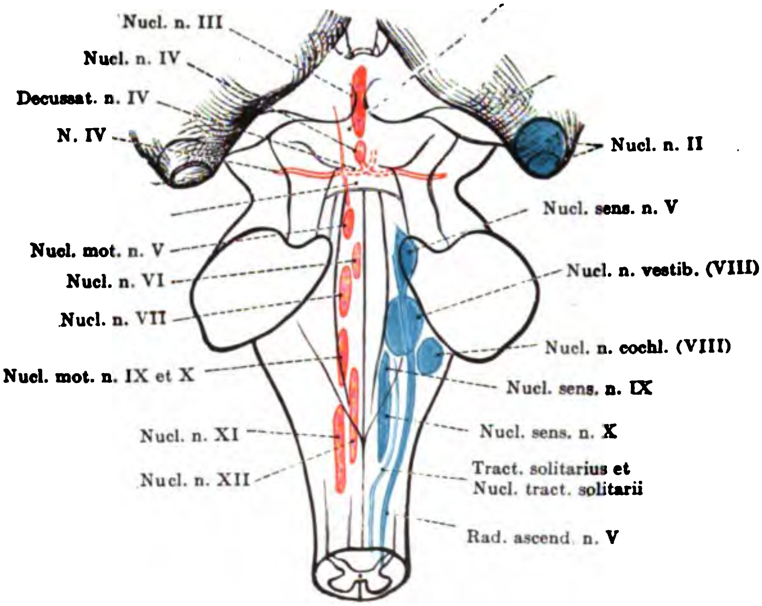

FIG. 48.—NUCLEAR ORIGIN OF THE CEREBRAL NERVES. (After Toldt.)

where the cerebral peduncles converge to an angle before disappearing below the pons.

The nuclei of the *trochlear nerve* unite (Figs. 47–49) behind the nuclei of the oculomotor nerve at the height of the posterior part of the posterior corpora quadrigemina. The nerve processes of its cells now follow a peculiar course. For a short distance they run horizontally and caudally, then vertically (dorsally), and internally, until they reach the velum medullare anticum which, as is well known, covers the fourth ventricle anteriorly, while the lingula of the cerebellum extends beyond it. Here in the velum (Fig. 48) the fibers cross those of the other side, run alongside the velum, and emerge as a nerve at the point where the fourth ventricle forms an angle anteriorly, because the superior peduncles of the cerebellum by their divergence from the corpora quadrigemina to the cerebellum mark the anterior structure of the rhomboid fossa (fourth ventricle).

The origin of the other motor cerebral nerves must be sought in the *medulla oblongata*, where they are distributed throughout its entire extent. Some are in the anterior part of the rhomboid fossa above the pons, others in the posterior part which is characterized by pyramids from the base, while a third area for their origin is in the medulla oblongata below the fourth ventricle and extending caudally far down into the cervical cord.

We would digress too far were we to trace accurately the topography of these various origins. I refer all those who are interested to the above men-

tioned text-books of Edinger, Obersteiner and v. Bechterew. I shall only attempt to review briefly their complicated relations.

The *motor root of the trigeminal or trifacial nerve* (the nerve for the muscles of mastication, Figs. 48 and 49) rises in the extreme anterior part of the medulla oblongata (therefore frontally). In contrast to the very prolonged sensory nucleus its origin is somewhat circumscribed, lying vertically above the place where the nerve emerges from the pontal fibers. We find its nucleus not far below the anterior third of the rhomboid fossa at its lateral border in the region of the locus cœruleus. Naturally the locus cœruleus must be divided, i. e., a part of the sensory trigeminal nucleus which has been stained by pigment, and which during its prolonged course lies laterally in the rhomboid fossa, is situated just below the surface. The fibers pass from the motor nucleus without crossing and almost horizontally (a

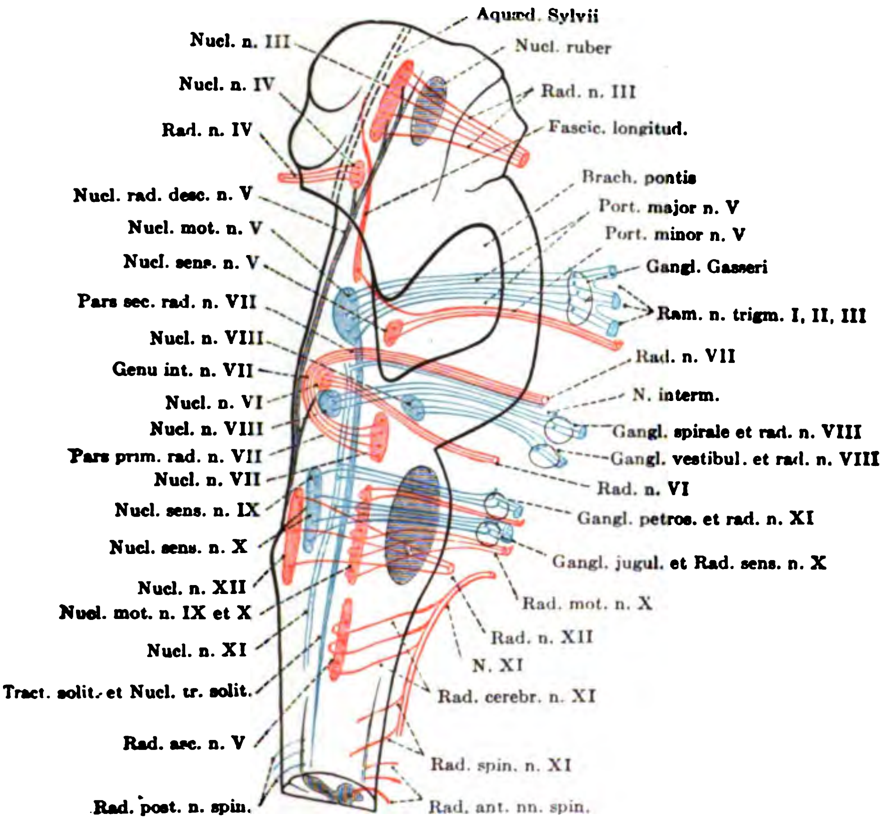

FIG. 49.—NUCLEAR ORIGIN OF THE CEREBRAL NERVES. (After Edinger.)

little outwardly) through the pontal fibers at the point where these run to the cerebellum as its superior peduncles. Its nerve fibers are situated within this longitudinal fiber layer through which they pass to those of the sensory trigeminal roots; thus we see at the base of the brain between the pons and superior peduncles a common root emerging from the cerebellum.

Close to the motor origin of the fifth nerve, and upon about the same plane, but extending rather caudally than ventrally, is the lengthy origin of the *facial nerve* (Figs. 48, 49, 50). Its nerve-cells extend caudally to that point upon the surface where the rhomboid fossa terminates. Here it is divided by the sensory root of the trigeminal nerve, and posteriorly by that of the pneumogastric nerve.[1] Its nerve fibers run a peculiar course. They

FIG. 50.—FACIAL ORIGIN AND ITS SURROUNDINGS; NAMELY, NUCLEUS AND FIBERS OF THE AUDITORY NERVE. (After Edinger.)

at first rise dorsally above the long series of nuclear origins until immediately below the floor of the fourth ventricle. Here nearly all make a right-angled curve to the front, and thus form a bundle which passes for a short distance anteriorly, then suddenly forms another angle, the crus of which again curves ventrally and somewhat outwardly, thence runs directly and without crossing to the base, where (after passing through the pyramidal tract) it appears behind the pons at that point where the posterior olive, the border of the pons, and the restiform body form a small triangular groove. Therefore, the facial nerve in its peculiar course forms a double bend.

In the convexity of this knee, immediately under the floor of the fourth

[1] I shall at present not discuss the other masses which force themselves between the origin of the facial and that of the superficial sensory nuclei; for example, the fibræ arcuatæ, and the reticular substance.

ventricle, about at the boundary of the anterior and posterior half of the same, and quite close to the median line we find in a narrowly limited area the nuclear origin of the *abducens* (Figs. 48, 49, 50). They, therefore, have a different plane from the nuclei of the motor trigeminal and facial nerves (Fig. 48). The nerve fibers of the abducens run horizontally, without crossing ventrally, somewhat outwardly but again inwardly after passing the pontal fibers, and thus at the height of the facial nerve, they appear at the base at just the point where the posterior olive and the pyramids unite with the posterior margin of the pons (Figs. 42 and 43) and form a groove.

The *motor nucleus* of the *pneumogastric*, also called the *nucleus ambiguus* (Figs. 48, 49, 52), lies caudally behind the facial, at the point where the rhomboid fossa reaches its posterior end. This nerve, like the facial, is overlapped by the sensory nuclei of the fifth nerve, above which (therefore dor-

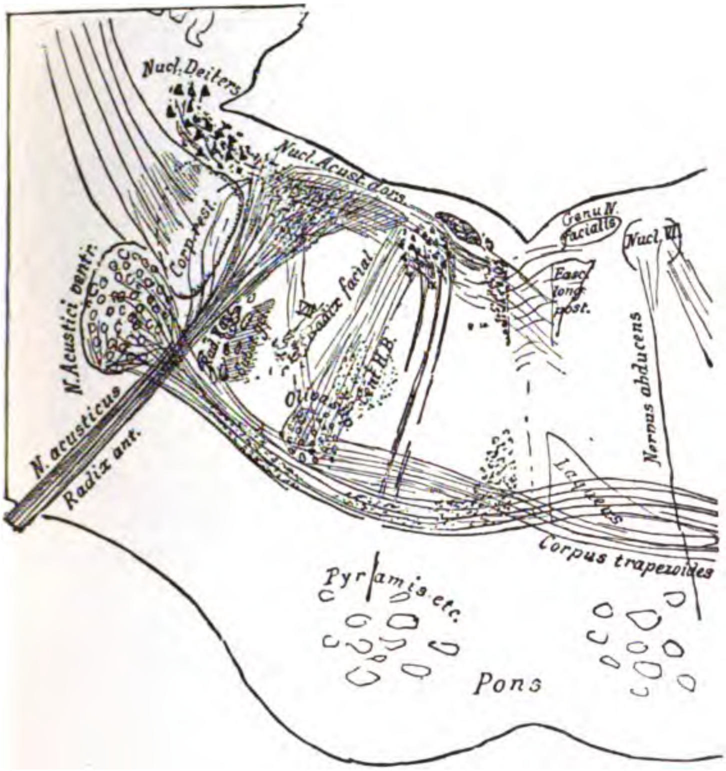

FIG. 51.—SECTION IN THE REGION OF THE ORIGIN OF THE ABDUCENS. THE VESTIBULAR NERVE. (After Edinger.)

sally) and immediately below the floor of the fourth ventricle, the nuclei of the sensory glosso-pharyngeal and vagus are arranged. Its nucleus extends caudally about as far as the posterior olive, but much further anteriorly, about half-way to the origin of the facial. Its nerve fibers follow a knee-shaped course similar to that of the facial, although they do not form a double knee, but merely pass dorsally and somewhat frontally to the region of the

sensory vagus nucleus, there bending and forming an angle to unite with the sensory main root which rises here. Together they pass outwardly and a little downward through the region traversed by the nuclei of the fifth nerve and their fibers (ascending trigeminal root), and emerge at the point where the main branch of the vagus also appears conjointly with the glosso-pharyngeal nerve; namely, in a space formed by the junction of the posterior olive with the remains of the lateral column and the restiform body (Figs. 42 and 43).

The *spinal accessory* (Figs. 48, 49 and 53) springs from behind the nucleus ambiguus in the same plane of the transverse section of the medulla oblongata.

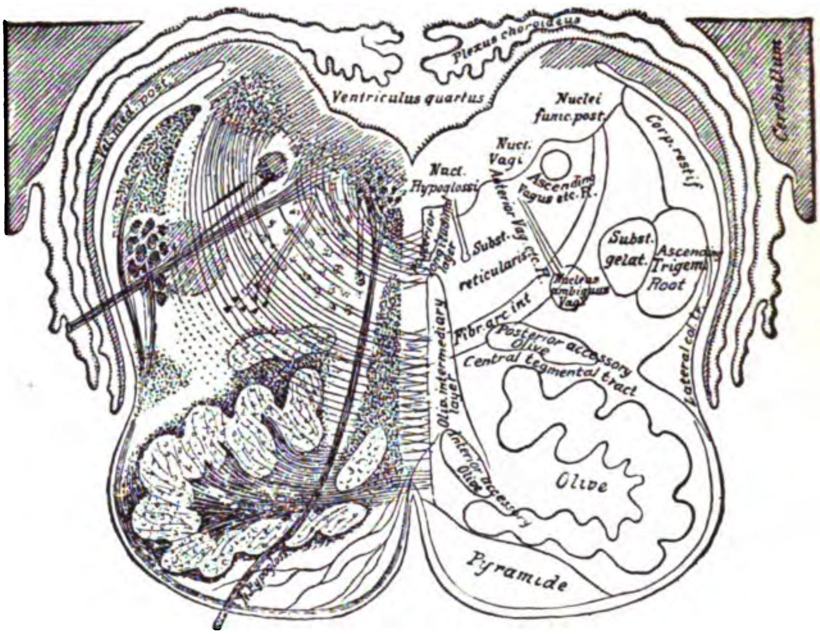

FIG. 52.—SECTION THROUGH THE MEDULLA OBLONGATA. (After Edinger.)

In its lengthy course its cells are found distributed as low as the sixth cervical vertebra. Its nucleus, therefore, has its frontal beginning at the pyramidal decussation just where the latter end of the posterior olive is visible upon transverse section, but it extends caudally far above the pyramidal decussation into the cervical cord (Fig. 49). The characteristic gray substance of the spinal cord is for some time distinctly visible, yet the original cells of the spinal accessory are still perceptible. We find them in the upper cervical cord, in those areas in which the lateral horns are more conspicuous than in the caudal cervical cord. Thence the fibers of the spinal accessory pass horizontally through the lateral column and outward. Still higher, at the height of the lateral horns, where, by manifold constrictions and changes of the gray substance, and especially by the pyramidal decussation, its characteristic butterfly appearance becomes obliterated, the nerve continues its course, and on the same frontal plane as the ambiguus vagi, the facial, and the motor trigeminal (Figs. 48, 49). In a simple bend the cells of the spinal accessory

send their fibers outwardly, first to the front, then horizontally and laterally. Thus these root fibers of the spinal accessory combine and form the nerve which passes out of the cervical cord, between its anterior and posterior roots, into the lower portion of the medulla oblongata at the point where the remains of the lateral columns extending caudally from the olive may still be recognized externally; from its lowest point of origin the canal of the spinal cord rises vertically, and its branches from the cervical cord and medulla passing to it, shoot-like, finally enter the base of the brain close to the trunks of the glosso-pharyngeal and pneumogastric nerves (Fig. 43).

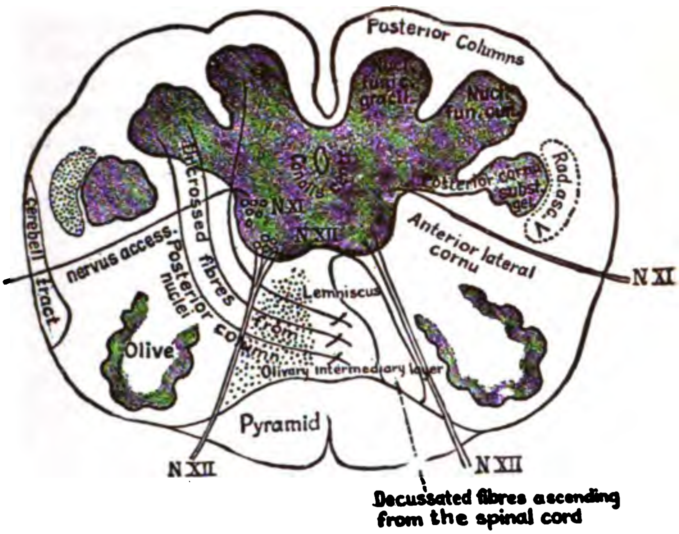

FIG. 53.—SECTION THROUGH THE MEDULLA OBLONGATA AT THE POINT OF EXIT OF THE PNEUMOGASTRIC. (Diagram after Edinger.)

The *hypoglossal nerve* remains as the last motor cerebral nerve (Figs. 48, 49, 53, and 54) to be discussed. Its nucleus, like that of the abducens (Fig.

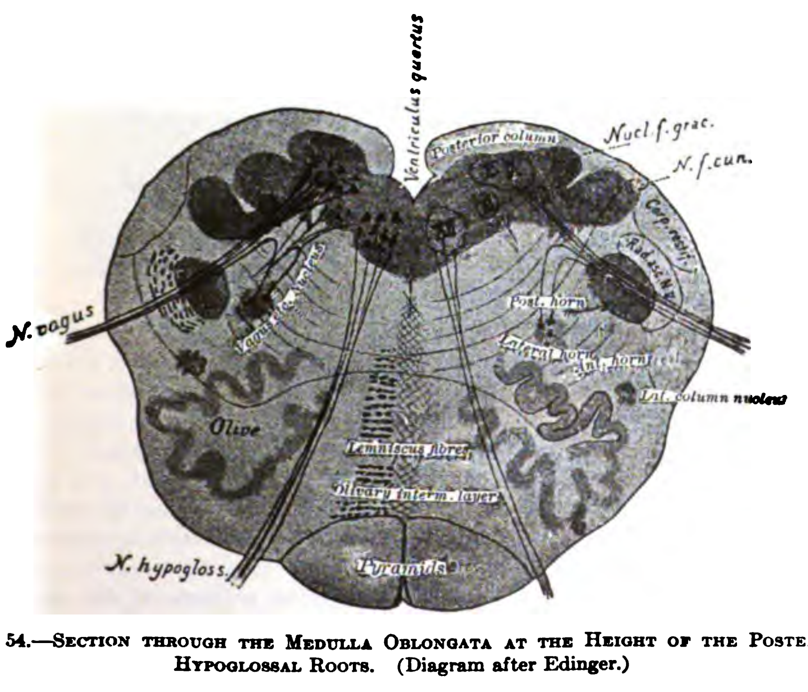

FIG. 54.—SECTION THROUGH THE MEDULLA OBLONGATA AT THE HEIGHT OF THE POSTERIOR HYPOGLOSSAL ROOTS. (Diagram after Edinger.)

48), is external to that chain of nuclei with which we have just become acquainted. Although a motor nerve, we find it in the region of the sensory nuclei of the medulla. Functionally it resembles the anterior horn cells of the spinal cord pressed into the median line and dorsally (by the pyramidal decussation through the reticular substance and the lemniscus tract), while the anterior motor cerebral nerve nuclei retain their position like the anterior horns of the spinal cord. Its caudal beginning, at about the height of the pyramidal decussation, occupies a corresponding position with that of the anterior horns of the spinal cord, ventrally close beside the nuclei of the spinal accessory. Frontally, however, the hypoglossal turns to the median line, and simultaneously into the dorsal area of the medulla, so that at the height of the fourth ventricle it is already upon its floor (Fig. 49). The central canal of the spinal cord, upon the anterior side of which the nucleus of the hypoglossal was originally situated, opens into the fourth ventricle. Thus we find the nucleus of the hypoglossal, when it has reached the region of the fourth ventricle, upon the floor of the same structure upon both sides of the median line, occupying the posterior quarter. The nerve fibers of the hypoglossal pass in a straight line without crossing, obliquely outward and downward, and form a number of bundles between the lower olive and the pyramid.

If, in connection with this description, we bear in mind the branches of the motor cerebral nerve passing to the periphery, we have a summary of the entire first motor neuron of the brain.

(2) THE SECOND MOTOR NEURONS

The cerebral nerve nuclei, as well as the motor cells in the anterior horns of the spinal cord, are united with the terminal twigs of other nerve fibers which, with the nerve-cells belonging thereto, form a *second neuron* which has also a motor function. This second centrifugal tract originates in the cortex of the brain, and passes in a course now to be described from a definite area of the same to the cells of the first neuron. In diseases of either of the two motor neuron tracts, the effect upon muscle movements invariably differs to a certain extent.

Degeneration of the first neuron leads to *flaccid paralysis* of the affected muscle which is *total* and irreparable, and atrophy rapidly appears in these muscles (see below).

It is different with the second motor tract, through which are conveyed the *conscious impulses of the will.* Without it, even with an intact first neuron, active contraction of the muscle is impossible, and only *unconscious and reflex movements* are produced. A permanent interruption of this second neuron does not produce flaccid paralysis, but spasms and contractures in the muscle areas affected by disease of the second neuron are induced, and these are attributed to the absence of control, therefore the inhibition, of impulses of the will, and to the preponderance of reflex irritation. Often, however, the paralysis is complete.

The second motor neuron originates in those portions of the *cortex of the brain* which we designate as the *motor.* From animal experiments (Ewald and Hitzig, H. Munk) as well as by embryologic investigation and clinical

research combined with the findings at autopsies, we know that the motor cerebral cortex is situated in the *posterior and lower portion of the frontal lobe, and in the anterior portion of the parietal lobe,* especially in the anterior and posterior (?) central convolutions. Here also there are *isolated fields* for certain motor regions.

Thus, the center for the lower extremities is situated in the central convolutions on both sides of the upper portion of the central furrow (Figs.

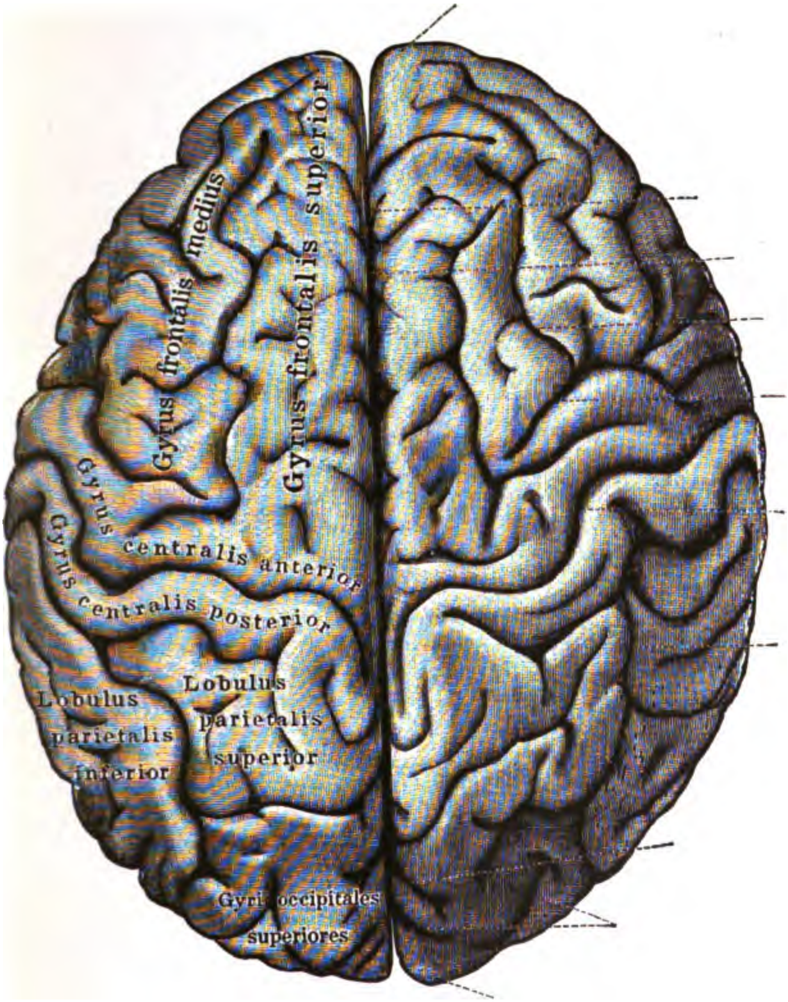

FIG. 55.—CONVEXITY OF THE BRAIN FROM ABOVE. (After Toldt.)

55–57); those for the upper extremity in the lower portion; for the facial and hypoglossal nerves the center is quite low down; that for the larynx is in the operculum; the center for movements of the trunk is in front of the centers for the extremities in the posterior part of the two upper frontal convolutions; that for speech beside the center for the larynx in the posterior lower frontal convolution close to the operculum. The center for the move-

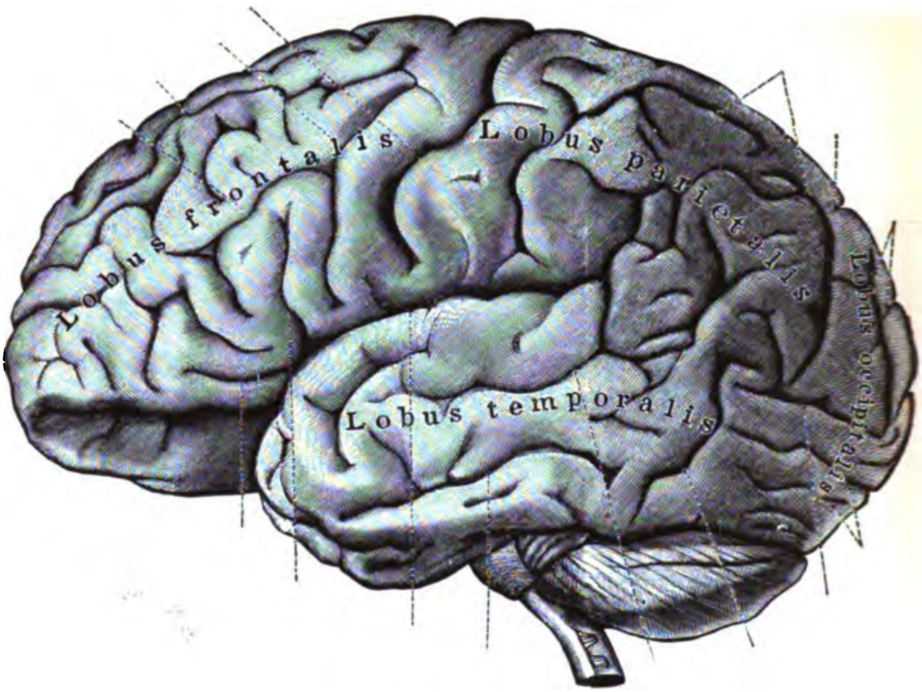

Fig. 56.—Convexity of the Brain Seen from the Side. (After Toldt.)

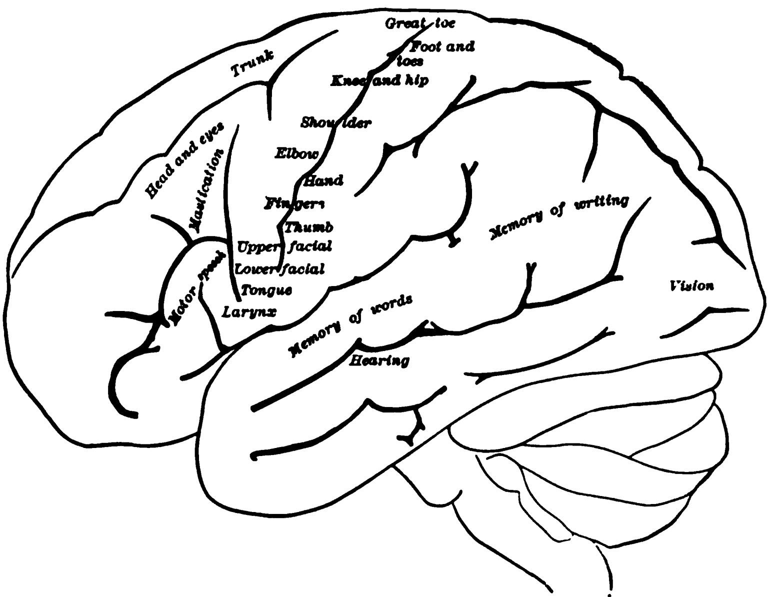

Fig. 57.—Motor Region of the Cerebral Cortex in Man. (After Oppenheim.)

ments of the eye is behind the center for the upper extremities in the anterior portion of the parietal lobe.

Here it is worth while to examine the *characteristic structure of the motor cerebral cortex* which is particularly rich in fibers and cells.

As is well known the entire cortex of the brain is composed of several layers. Layers of cells of various kinds alternate with rows of fine fibers which are arranged radially, horizontally or vertically. In different areas of the cortex of the brain this structure is sometimes simple, sometimes complicated, and perhaps is most complicated in the hippocampus major.

But the motor cortex of the brain also is characterized by manifold layers (Figs. 58, 59). First we have a layer of numerous small pyramidal cells below a tangential fiber mass composed of a few small cells. Then comes a broad, deep layer composed of less numerous but very large pyramidal cells; all have their apices pointing upward. Then follows a fourth layer of small, and not very numerous pyramidal cells, irregularly placed between dense vertical fibers. In all these layers there is a considerable number of disseminated polygonal cells and glia nuclei. The latter are particularly dense between the third and fourth layers. Any one interested in the structure of the cerebral cortex is referred to the excellent description of Ramón (Studien über die Hirnrinde des Menschen. Leipzig, 1900. Ambros. Barth).

Of all the cells of this part of the cortex (which with their dendrite nerve fibers and collaterals form a very dense network that has been explored by Golgi and Ramón), the *large pyramids* (Fig. 59, *III*, *i*) of the third layer are the only ones which appear to be regarded as cells of the second motor neuron. They are characterized by a many-branching dendrite net which extends to the surface as well as to the sides, and is intimately related to other dendrites. The nerve processes rise vertically or radiate from this while sending numerous collaterals downward into the white medullary mass of the centrum semiovale.

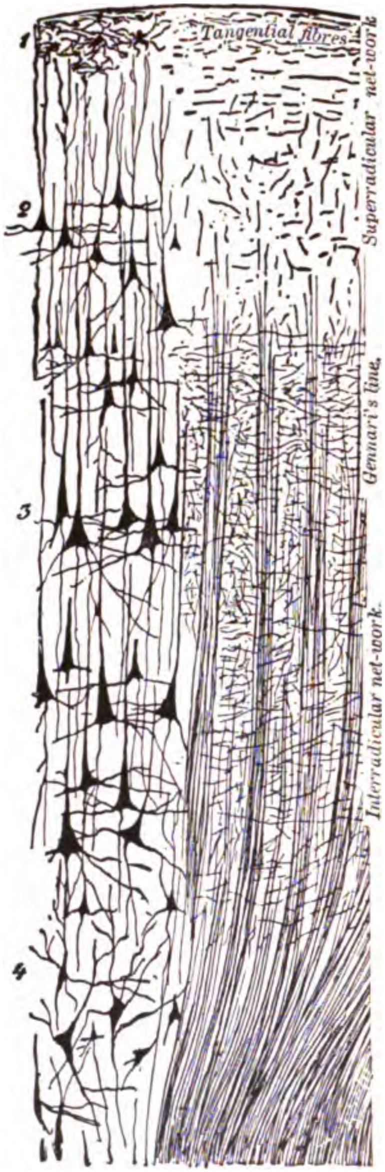

FIG. 58.—SECTION THROUGH THE CORTEX OF A FRONTAL CONVOLUTION; TO THE LEFT CELLS STAINED BY GOLGI'S METHOD, TO THE RIGHT FIBERS STAINED BY WEIGERT'S METHOD. (After Edinger.)

Here they take part in that *projection fiber system* which passes from all sides of the cerebral cortex to each hemisphere, just as the so-called fibrilla-

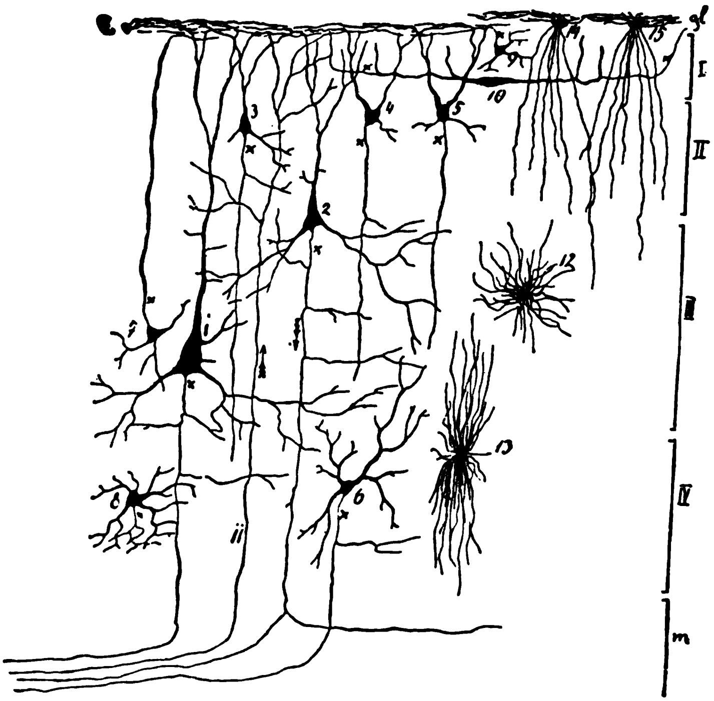

FIG. 59.—A PARTLY DIAGRAMMATIC PRESENTATION OF THE CORTEX OF THE CEREBRUM AFTER STAINING WITH SILVER.

I, Molecular layer; *II*, small pyramidal cells; *III*, large pyramidal cells; *IV*, polymorphic cells with medullary layer. (After Obersteiner.)

tion of the corona radiata passes to the caudally situated areas of the brain (Figs. 61 and 62). While a large portion of these fibers pass to the *root ganglion* (corpus striatum) or to the *inter-brain* (thalamus opticus), the nerve fibers of those pyramidal cells in the deeper parts of the medullary layer of the hemispheres form a distinct bundle, the *motor* or *pyramidal tract* (Fig. 61). At the level of the corpus striatum this enters into the medullary layer of the same called the *internal capsule*, which divides the corpus striatum into the caudate nucleus and the lenticular nucleus, and shows upon horizontal section an obtuse angular bend, which opens outwardly (Figs. 51, 55). Therefore, upon horizontal section the internal capsule reveals an *anterior and a posterior crus*. In the *anterior third of the posterior crus* is the motor bundle, which contin-

FIG. 60.—SECTION THROUGH THE INTERNAL CAPSULE.

ues ventrally and caudally and at the base of the brain enters the cerebral peduncle running under the thalamus and the corpora quadrigemina (Figs. 61, 62), the ventral portion of which it forms separated from the dorsal through the substantia nigra by the tegmentum. In the fibers of its foot are the projection fibers from the central convolutions, as well as branches from the frontal, parietal and temporal lobes.

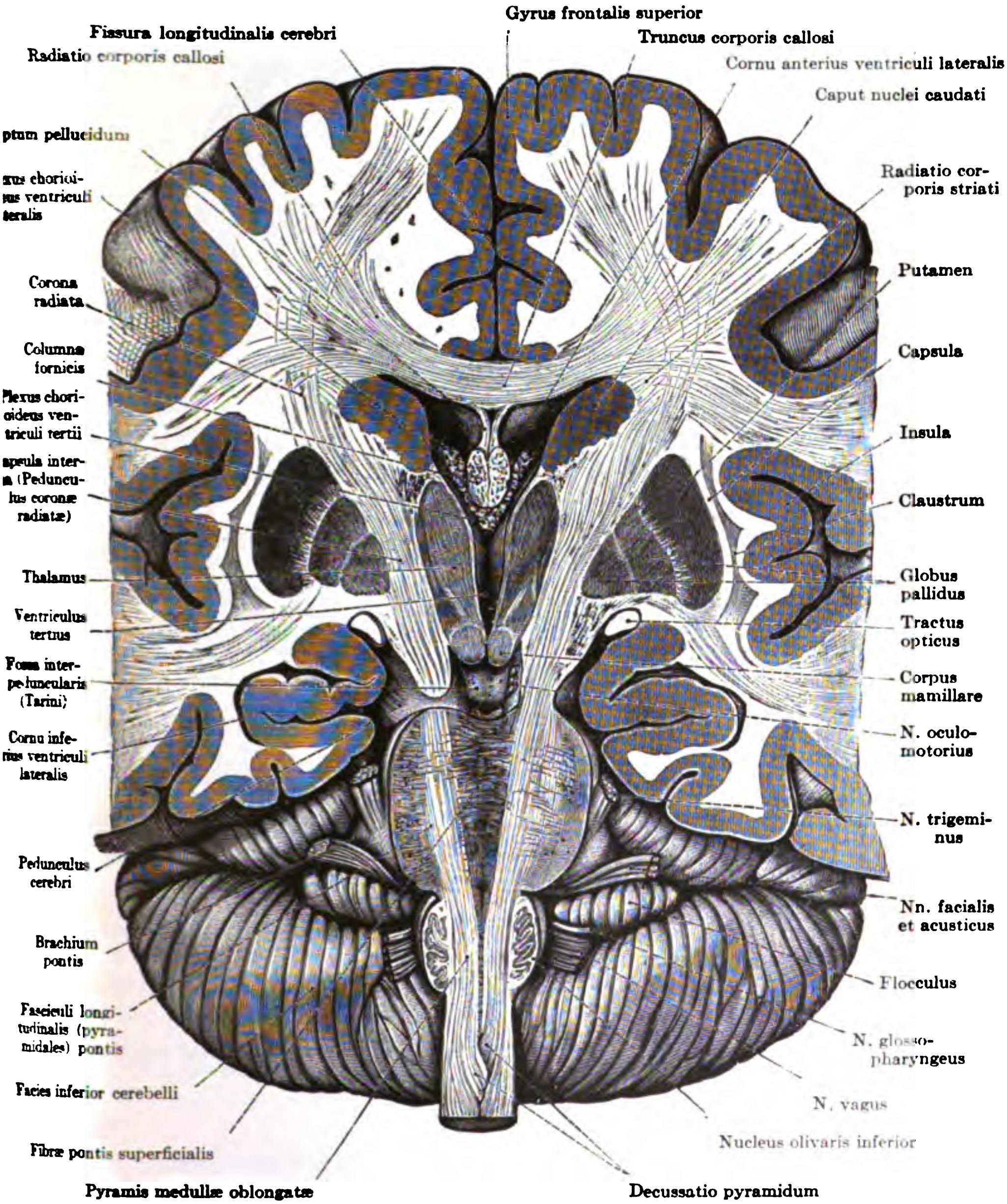

FIG. 61.—THE PYRAMIDAL TRACT. (After Toldt.)

The *tracts* of the second motor neuron are in the foot; these run side by side for a short distance (Figs. 42, 43, 46) but soon diverge. and only a comparatively small number of them continue caudalward. The others ex-

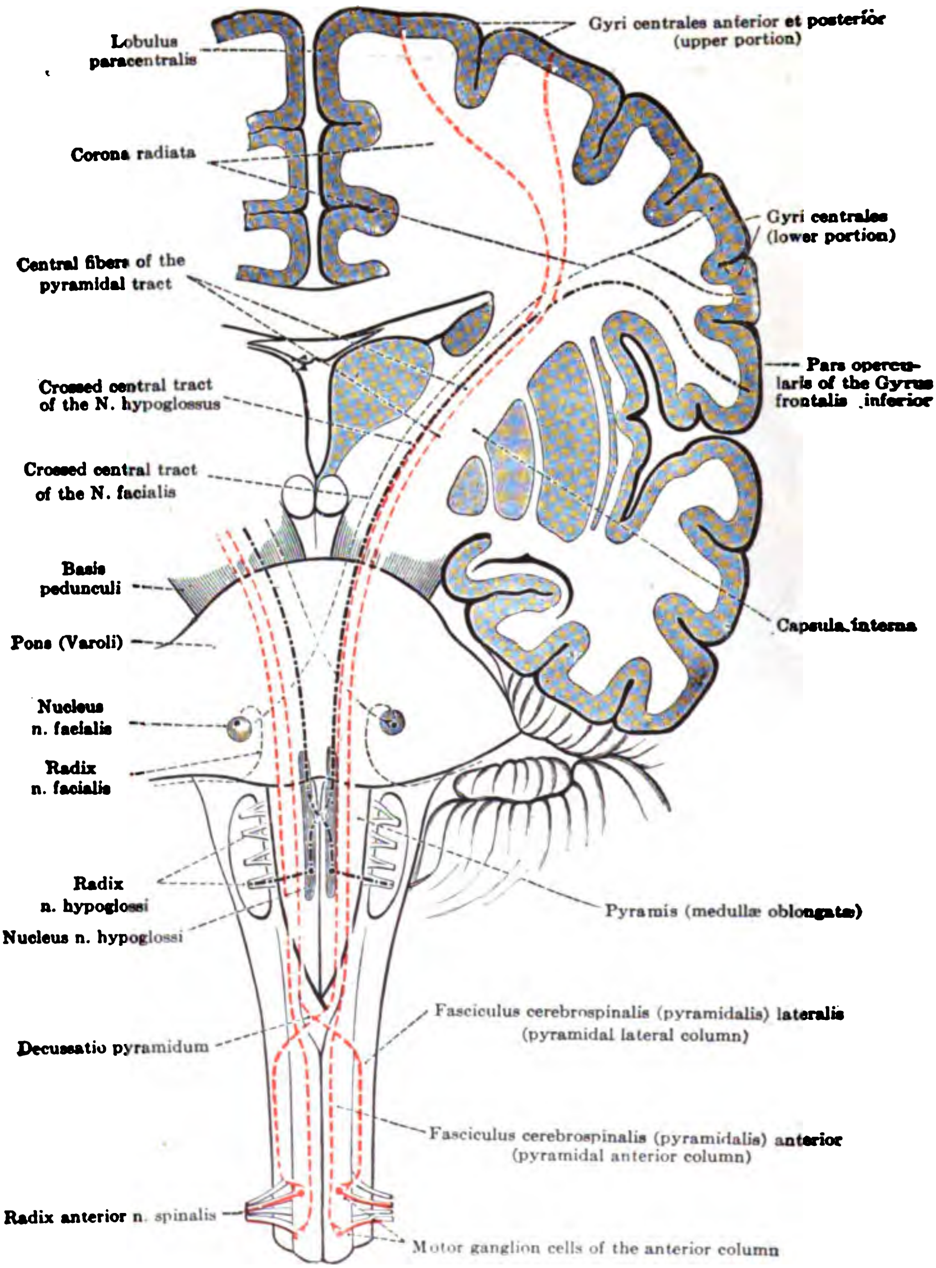

FIG. 62.—THE PYRAMIDAL TRACT (RED) BESIDE THE TRACT OF THE HYPOGLOSSAL AND FACIAL NERVES (BLACK). (After Toldt.)

tend no deeper than the area of the pons (Figs. 42, 43, 51, 61) and these are mostly transverse fibers which pass from one hemisphere of the cerebellum to the other and also cover from below those fibers which had previously centered in the cerebral peduncles. *The majority of the motor fibers* from other peduncles (Fig. 62) are lost in the pons. We are familiar with only a part of this divergent tract. By analogy, as well as by direct histologic observations (Spitzka), it appears reasonable to assume that these tracts diverge only in the pons from those which are situated deeply because they unite with the cerebral nerve nuclei in the mid-brain and medulla, and surround these cells with their own terminal arborizations; at present we know the tracts of only the facial and hypoglossal nerves (Edinger). At all events, at the *upper end of the pons* where the peduncles are inserted *a thick bundle*

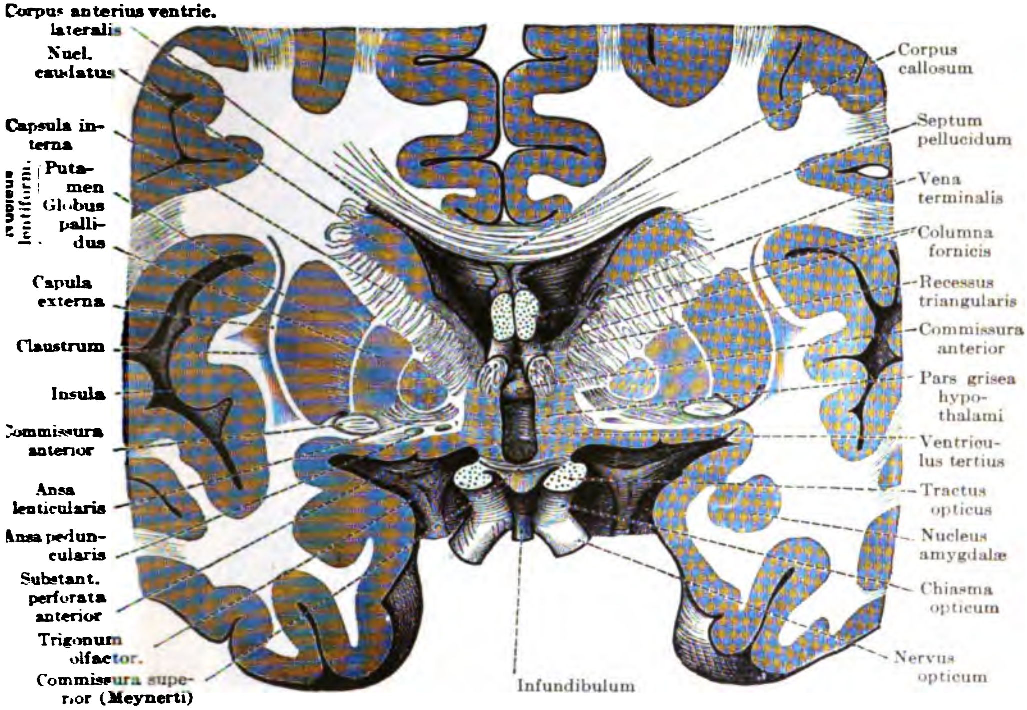

FIG. 63.—FRONTAL SECTION THROUGH THE HEMISPHERES BEHIND THE OPTIC CHIASM. (After Toldt.)

passes dorsally from the foot to the *tegmentum,* and this after previous decussation in the raphé may carry the motor neurons to the cerebral nerve nuclei, particularly to both of the above mentioned nuclei. Other fibers of the peduncles which do not form this bundle, and which extend no further caudally, are lost in the numerous *nerve-cells of the pons* which lie between the fibers passing through the cerebellum (the fibers of the middle peduncle of the cerebellum).

The remainder of the ventral portion of the crus cerebri which passes transversely through the pons as a *closed bundle* (Figs. 61, 62) and then extends caudalward is called the pyramidal tract. This unites at the posterior

border of the pons in the form of the so-called *pyramids* at the base of the medulla (Figs. 42, 43, 61, 62), and subsequently with the so-called *pyramidal*

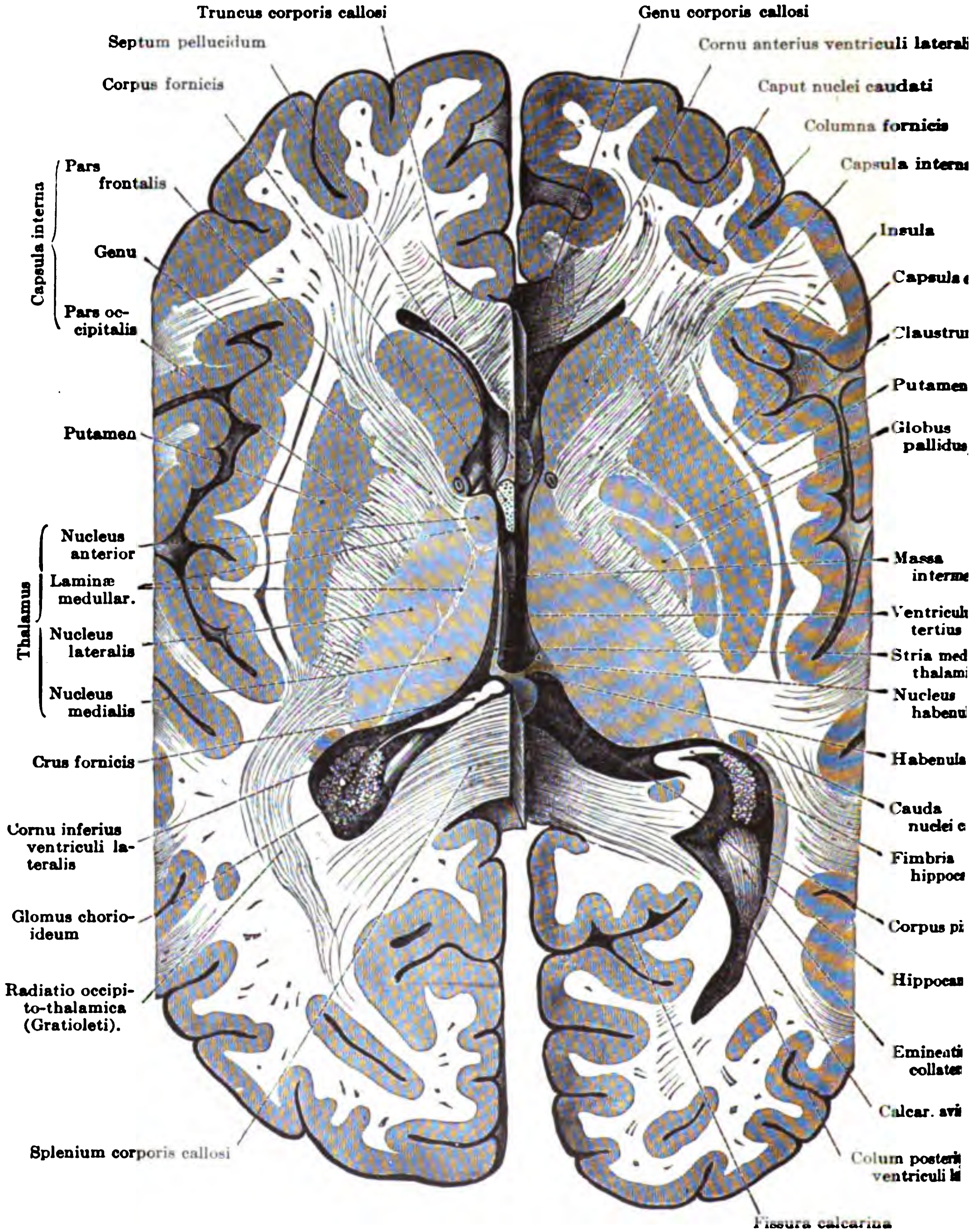

FIG. 64.—HORIZONTAL SECTION THROUGH THE HEMISPHERES (SOMEWHAT DEEPER UPON THE RIGHT SIDE). (After Toldt.)

decussation below the point where the lower olives are laterally attached. The majority of the fibers in this tract cross to the other side, and simultaneously extend to the ventral surface on that side of the cord, which they continue to traverse as the *pyramidal lateral column tract* (Fig. 37, *b*). In man a few of these fibers do not cross but remain in the anterior columns, the so-called Türk's bundle (Fig. 28, *i*); subsequently they also pass downward into the spinal cord. In the spinal cord itself these fibers of the pyramidal tract pass from time to time to the *large motor anterior horn cells* with their terminal filaments (Figs. 38, 65). The fibers for the lateral column pass directly into the gray substance, those for the anterior columns only after the fibers of the anterior commissure have crossed. It is therefore the property of the second motor neuron (Fig. 62) *to terminate on the side of the central nervous system opposite* to that in which it originates, and subsequently to surround the nerve-cells of the first motor neuron which, on its part, continues its course through the spinal cord and, to a great extent, through the brain.

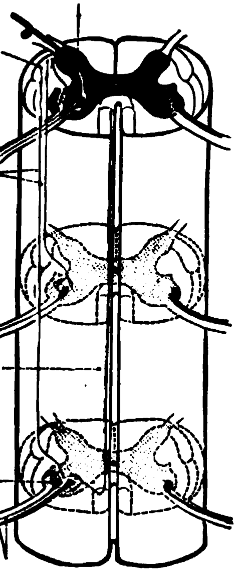

Fig. 65.—Course of the Second Motor Neuron in the Spinal Cord; Diagram. (After Toldt.)

In reviewing this description of the path of the second motor neuron in the spinal cord it is evident that its course has been quite accurately determined; in the brain, however, its termination near the origin of the cranial nerve is still to be found.

Recently it has been demonstrated that the pyramidal tract in the lateral columns of the spinal cord, which had previously been regarded as an entity, contains a *special variety of fibers*, discovered by Monakow, and, according to the investigations of Rothmann, this promises to be a valuable addition to our knowledge of the processes of motion. This tract, designated as Monakow's bundle, extends backward and gradually diverges from the pyramidal tract in such a way that in the pons and in the ventral portion of the crus cerebri it enters the tract of the tegmentum, crosses near the corpora quadrigemina, and is finally lost in the region of the red nuclei. Corresponding to this we have also a special motor neuron for the second division, the cells of which in all probability also originate in the cerebral cortex; but concerning this we have no absolute knowledge.

B. DISEASES OF THE MOTOR TRACT

Although nerve-cells and processes actually form a histologic and biologic unit, nevertheless it is not absolutely necessary that the neuron theory should simultaneously stand or fall with this, and that the entire neuron should

invariably and uniformly be attacked by disease. Of course, when the nerve-cell is destroyed we may quite properly assume that the processes originating in it also immediately degenerate. But inversely, when there is destruction of one or several processes anywhere in its course and consequent interruption of function, we cannot assume that the cell itself and the central portion of the nerve process must likewise undergo degeneration. On the contrary, this is true only of the peripheral end; cell and process form a histologic and physiologic entity. Hence, as we maintain that every cell may continue to live if the damage which it sustains does not injure too great a part of its body, so the nerve-cell also may retain its properties if not too large a portion of its processes, that is of itself, is destroyed, and this is especially true whenever subsequent restitution is possible. Naturally if the degeneration of the nerve fiber is very extensive, and particularly if this be permanent, the cell itself will suffer. This has been proven by pathologic histology, as well as by experiment.

The observation is frequently made that a nerve tract is primarily only partially diseased, and later wholly degenerates; this does not conflict with the neuron theory. On the contrary, such diseases of the nerves are the strongest proofs that an entire neuron may be attacked or several individually.

Such *primary diseases of neurons*—after the foregoing we are interested only in the motor and sensory, other neurons having been as yet but little investigated—may quite properly be designated as *neuron diseases*, and may be differentiated from other affections of the nervous system which are caused secondarily by the vessels, or by the supporting tissue, or by the effect of traumata and tumors affecting the nerve tracts.

I shall attempt in the following to select from the whole series of diseases of the nerves those neuron diseases with which we have become familiar, and to portray these as such. It is an initial effort, and this must excuse its incompleteness.

I shall begin with *neuron diseases of the motor tract*, chiefly from a histological standpoint, without minutely discussing their clinical aspects. The subject of sensory neuron diseases will follow the description of the normal sensory neuron.

There must be no misapprehension if a *comparatively small number* of diseases follows the preceding theoretic explanation. On the contrary, I must state that we are only groping in the dawn of scientific research, and that this subject still contains many dark problems. Above all, autopsies and investigations with approved modern methods are most necessary; for the great abundance of material, the diseases often running an exceedingly slow course, do not permit the clinical and larger institutions to investigate these as distinct diseases because many carefully observed cases die outside the hospital from intercurrent affections, and thus afford no opportunity for the autopsy which is so necessary for the scientific elucidation of affections of the nerves.

(1) DISEASES OF THE FIRST MOTOR NEURON

(a) *Its Multiple Affections in the Spinal Cord*

The Spinal Form of Progressive Muscular Atrophy.—Clinically we are all familiar with the characteristic disease which Duchenne and Aran first designated as progressive muscular atrophy, beginning very slowly in the upper extremity, and finally distributing itself to other parts.

Autopsies conducted with the aid of recent methods—notwithstanding the frequency of the disease but few reports are at hand—have shown that a typical neuron disease here forms the basis of the muscular atrophy. The *motor cells of the anterior horns* of the spinal cord (probably with their dendrites) and the *nerve fibers* originating from them (*through the anterior roots*), even the atrophied paralyzed muscles, are primarily degenerated. In place of the absent or atrophic nervous substance proliferated glia tissue appears, and the walls of the vessels are thickened. The affection is therefore not a primary disease of the muscle, but a degeneration of the neurons supplying the muscles, with secondary atrophy of the muscles.

(*b*) *Multiple Motor Neuron Diseases in the Brain*

(*i*) **Chronic Progressive Bulbar Paralysis.**—This disease of the spinal cord is closely related to that of the medulla oblongata which exclusively affects the cells of the motor nuclei and their fibers in this region; therefore the first motor neuron. The elective affinity for the motor tract which this process here displays is the more marked because the sensory nuclei and their tracts often lie in the immediate vicinity, yet are not implicated. Here also we find, as in the preceding form, *destruction of the motor nerve-cells*: *Degeneration of the motor cerebral nerves and disappearance of their root fibers.* In place of the absent nervous structure there is a proliferated glia tissue. The disease of the spinal cord sometimes changes into the bulbar form so that the entire peripheral motor tract is involved.

(*ii*) **Progressive Ophthalmoplegia.**—In this disease the process chiefly attacks the motor nerve nuclei of the medulla oblongata, while the nuclei of the muscles of the eye with more frontal situation are exempt or are only secondarily implicated.

On the other hand, there is a quite rare affection in which the more frontal nerve nuclei are predominantly involved, especially the nuclei of the *oculomotor* and the *trochlear nerves* in the mid-brain, occasionally also those of the *abducens*, and in very severe cases there is a partial implication of the nucleus of the *facial nerve.* Both of the latter, as is well known, belong to the anterior divisions of the medulla oblongata.

Here we are dealing with a lesion of the *anterior portion* of the first motor cerebral neuron, and therefore this affection, clinically named *progressive ophthalmoplegia*, may be included among the neuron diseases.

(*iii*) **Circumscribed Diseases in the Area of the First Motor Neuron.**—The affections previously mentioned are diseases of the spinal cord or the brain which implicate a great number of neurons. The more *diffuse* affections are contrasted with those which occur in a *circumscribed* area, in an isolated nerve nucleus, or which implicate only the motor conduction tract belonging thereto. Among those of the *brain* are *isolated nuclear paralyses*, and of *the spinal cord those paralyses* designated as monomuscular, which, however, are in part not of peripheral origin but central, their origin being in a corresponding portion of the anterior horns of the motor nerve, the actual focus of the disease.

There is a great paucity of literature on this subject, not because the cases are rare, but because, for reasons above mentioned, they have not been studied.

There are no cases of undoubted disease of the spinal cord which may be regarded as central monomuscular paralysis or primary affection of the neuron; of course, secondary disease following tumors and inflammatory processes of the membranes of the brain, and particularly after injuries, is often reported in literature.

Idiopathic nuclear paralyses have, on the contrary, been histologically determined in the brain. The oculomotor nerve furnishes an example of such a circumscribed neuron disease, which is frequently seen clinically, and more rarely anatomically. Here we do not consider the *progressive* form of paralysis mentioned above, but that dependent upon a different etiologic base (migraine, syphilis, etc.), and which is often of benign type.

Other nuclei of the nerves of the muscles of the eye, especially the abducens, occasionally suffer from similar attacks.

Recently the neuron diseases of the spinal accessory have been studied, but the material for investigation is exceedingly scant, and additional researches are necessary.

No further description of the histologic picture is necessary; its prominent features remain the same: Disappearance of the nucleus and fibers in the entire course of the neuron.

(2) DISEASES OF BOTH MOTOR NEURONS

Atrophic Spastic Bulbospinal Paralysis (Senator); (Amyotrophic Lateral Sclerosis, Charcot).

We as yet know little of primary diseases which attack the second motor neuron exclusively, either in its entire extent or in any part of its course. Naturally, the most common and best known are *secondary* lesions and interruptions of continuity in various areas of these tracts (see below). The histology of recent cases of acute polioencephalitis, the counterpart of poliomyelitis, which is most often observed in children, has not been sufficiently studied for us to pronounce it a neuron disease.

Charcot has described under the name of *spastic spinal paralysis* an affection with a characteristic symptom-complex, and, because of these symptoms, has postulated a primary degeneration of the *pyramidal tracts* of the spinal cord; therefore, according to our conception, the tract of the second motor neuron; wherefore, if the neuron theory be otherwise correct, it must also include a degeneration of the motor cells of the cerebral cortex belonging thereto, a process which has as yet been too little studied.

So far no conclusive case has been anatomically investigated—the case Strümpell reported also showed disease of the posterior column; instead of the looked-for changes, the necropsy revealed very different conditions (multiple sclerosis, combined system diseases, tumors, meningitis). *It still remains a mooted question whether there really is an isolated affection of the entire second motor tract.*

But we recognize distinctly a not very rare disease which was first graphically described by Charcot; its anatomical course conclusively proves the existence of disease of the neurons. Charcot gave to this the anatomico-clinical designation of *amyotrophic* (myatrophic) *lateral sclerosis*. Senator has denominated it by a purely clinical term, *atrophic spastic bulbospinal paralysis.*

Histologically, the affection in its full development reveals the following conditions (Fig. 66):

(1) **Primary degeneration of the first motor neuron** similar to that of progressive muscular atrophy and chronic bulbar paralysis.

(2) The same **primary degeneration of the pyramidal tracts** which was postulated by Charcot for spastic spinal paralysis, but which has not yet been proven.

The implication of the *motor cerebral cortex*, that is, of the cells in which the motor tract originate, has not yet been certainly demonstrated and requires confirmation; it is questionable in how far disease of these greatly disseminated cells may be histologically determined by our ordinary methods. At all events, it is not too far fetched a hypothesis to regard these cells as implicated in the process. Thus we have a picture which most clearly portrays the symptoms of disease of both motor neurons.

FIG. 66.—TRANSVERSE SECTION THROUGH THE SPINAL CORD IN ATROPHIC SPASTIC BULBOSPINAL PARALYSIS.

In enumerating the diseases of the first motor neuron, I stated that our knowledge of these circumscribed affections is very limited, and we are absolutely ignorant concerning the changes of this kind in the second neuron. We know of no histologic alteration in a cortical area which can be referred to an isolated and circumscribed affection of a definite muscle or muscle group, which would produce the symptoms of a spastic monomuscular paralysis. Perhaps in the future we shall acquire more definite knowledge concerning this.

C. THE SENSORY TRACT

(1) THE FIRST SENSORY NEURON

It has been embryologically determined, especially by the investigations of His, that the centripetal tracts in the spinal cord and brain which we designate as the distribution of the entering posterior roots and of the sensory cerebral nerves are at first adjacent and external to the central organ, and that after only a definite period of development do they become a part of the spinal cord. This makes it clear that even after full development the nerve-cells belonging to these fibers are situated outside of the central nervous system; such are the *cells of the spinal ganglia* (Fig. 29) in which the sensory cerebral nerves resemble the spinal ganglia, the included *ganglia nodules,* this knowledge being also based upon the experimental severing of the posterior roots. Here the following ganglia must be included (Fig. 73): In the vagus and the glosso-pharyngeal nerve the jugular and petrosal ganglia; in the auditory nerve the spinal ganglion (cochleæ) and the vestibular branch of the same, the vestibular ganglion; in the trigeminal the Gasserian ganglion; in the optic nerve the ganglia of the retina; in the olfactory those of the mucous membrane of the nose. The nerve centers external to the central nervous system are the origin of the peripheral centripetal, apparently the entire, sensory tract. They are, therefore, the cells of the first sensory neuron,

and the posterior roots of the spinal cord and the sensory cerebral nerves are the nerve processes belonging to it.

The *cells* of the centers just mentioned differ markedly in their structure and in the nature of their arborizations from the motor cells. In the first place, they are deficient in dendrites; indeed, it is not certain that the cells of the spinal ganglion possess dendrites. Furthermore, they are usually not angular and polygonal like the motor cells, but are round or oval, like vesicles. A covering of connective tissue with nuclei protects them from injury in their peripheral position. The branching of the nerve processes is also characteristic of the sensory neuron.

Near its origin this divides into *two parts*, one of which by a longer or shorter route soon passes to the sensory end organ where it terminates in various ways, while the other proceeds toward the central nervous system, and there pursues a course which is soon to be described.

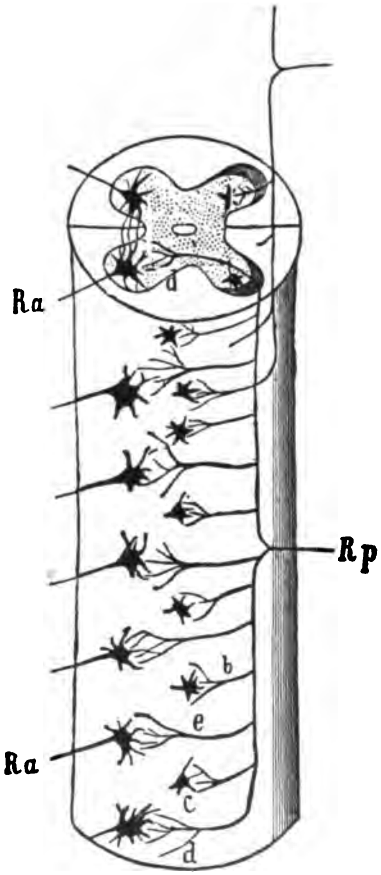

Fig. 67.—Diagram Showing the Branching of the Dorsal Roots. (After Ramón.)

(*a*) *The First Sensory Neuron in the Spinal Cord*

We shall first trace the course of the nerve fibers of the first sensory neuron in the spinal cord. The nerve process passes only a few millimetres from the cells of the spinal ganglia when it divides into a *peripheral* and a *central* branch (Fig. 29). The peripheral branch, after leaving the spinal ganglion and uniting with fibers of the anterior root, passes the processes of the first motor neuron in the spinal nerve (Fig. 40) between the vertebral canal and the periphery, where it separates from the motor fiber and extends to the skin and the other organs which the sensory nerve supplies. After forming a dense net in the cutis by the division and branching of its fibers, it terminates in various end-apparatus (which need not here be further described (Fig. 29).

The *central* process (Fig. 29) within the posterior roots passes to the spinal cord. We shall now trace the *somewhat complicated* course of these sensory posterior root fibers in the spinal cord.

Every posterior root after entering the spinal cord divides into *two* groups of fibers (Figs. 67, 68) each of which pursues a different course. The group of thicker fibers (Fig. 70, *rpi*) passes to the *posterior column*, the other passes to the *posterior horns* (Figs. 67, 69). Immediately after entering the spinal cord (in the angle formed by the junction of the periphery of the posterior

columns and the posterior horns) the fibers separate into two branches, a *short descending* and a *longer ascending branch* (Figs. 68, 69). These last-named branches pass toward the brain as far as the medulla oblongata (see below). During their course they constantly give off collateral branches to the gray substance, particularly those which penetrate the same side of the posterior horn to the cells of Clarke's columns, or by the posterior commissure upon the opposite side (Fig. 70). A few collaterals transversely describe an arc through the posterior horn of the same side, in front of the gelatinous

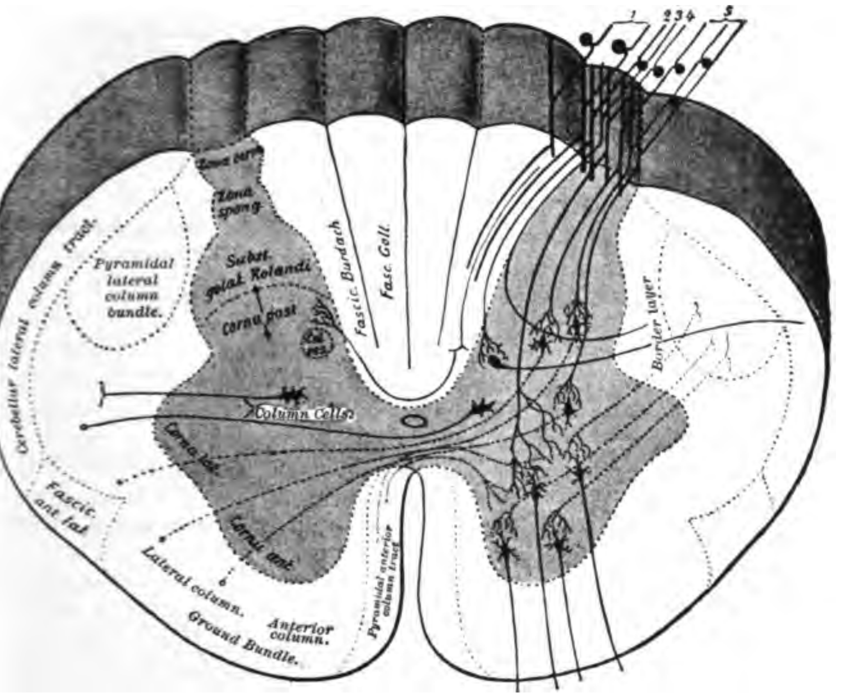

FIG. 68.—DIAGRAM OF A TRANSVERSE SECTION OF THE SPINAL CORD; TRACTS OF THE FIRST DIVISION CONTINUOUS; TRACTS OF THE SECOND DIVISION INTERRUPTED. (After Edinger.)

substance and outwardly into the lateral column, thence into unknown regions (Bechterew) (Figs. 68, 69). The descending roots in the posterior column follow a similar course (Schultze's bundle?) and finally appear to terminate in the gray substance of the posterior horns. Each root as it ascends (Fig. 37, *e*) inserts its fibers laterally beside those which have entered below, thus medially forcing the preceding ones away from the zone of entrance. Thus it happens that *the posterior roots which effected an ingress low down in the lumbar cord are found medially high up in the cervical cord,* in *Goll's columns* Figs. 37, *e*, 38, 68, and 69) while those which entered high up are still in Burdach's column (Fig. 37, *d*). Hence we find that the posterior root fibers and the posterior lateral area, filled by the same, are together designated the entrance zone of the roots.

The *other, more lateral group* of posterior root fibers (Figs. 67, 68, 69, 70, *rpe*) which entered the posterior horn is composed of fibers having a *two-*

fold course. One group passes horizontally through the gray substance directly to the *large cells of the anterior horn* (Figs. 69, 71). The other group, usually

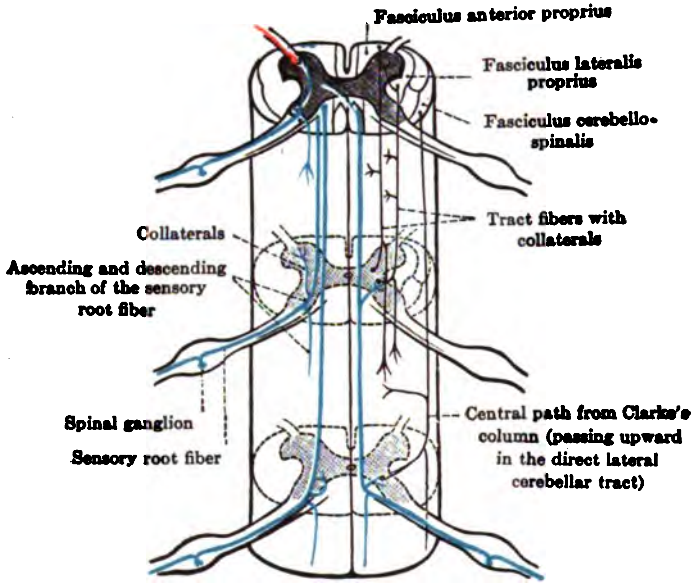

FIG. 69.—DIAGRAMMATIC COURSE OF THE FIRST SENSORY NEURON. (After Toldt.)

thin fibers, divide at the apex of the posterior horn, or perhaps just within it, occasionally in front of the gelatinous substance, into an ascending and descending branch. The finest fibers, which separate as soon as they enter the

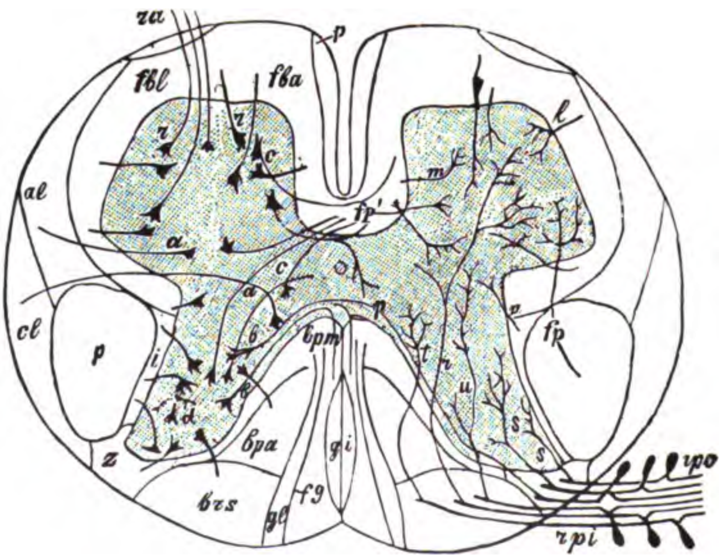

FIG. 70.—DIAGRAM SHOWING THE ORIGIN AND TERMINATION OF THE NERVE ROOTS IN THE GRAY SUBSTANCE OF THE SPINAL CORD AND THE DISTRIBUTION OF THE NERVE CELLS IN THE LATTER. (After Bechterew.)

apex of the posterior horn, form upon transverse section a small area, the so-called border zone of the posterior horn, or *Lissauer's zone* (zona terminalis) (Figs. 68 and 70, *z*). The branches of this group of posterior root fibers also give off numerous collaterals some of which extend anteriorly far into the gray substance while others terminate in the *small cells of the posterior horn* (Fig. 68). The mode of termination of all these processes and collaterals is invariably the same, and occurs by *splitting up*, so that the terminal twigs envelop the cells which they approach, or split up in the vicinity of their dendrites (Fig. 61).

The fibers which ascend to the *medulla oblongata* terminate there. At the beginning of the prolonged cord numerous nerve-cells appear in the posterior column, the *nuclei of Goll's columns* (funiculi graciles) and of *Burdach's columns* (funiculi cuneati) which may be followed to the beginning of the fourth ventricle (Fig. 71). Here all the fibers of the posterior roots split up, and here the first sensory neuron of the spinal cord terminates in a web of cells.

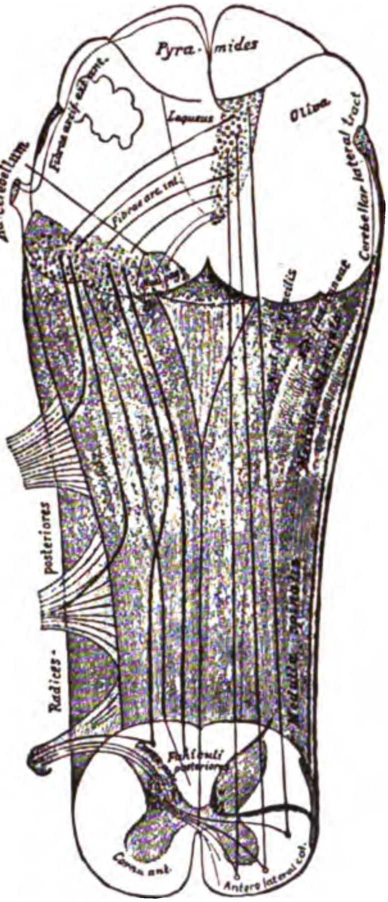

FIG. 71.—DIAGRAM OF THE COURSE OF THE SENSORY TRACTS FROM THE POSTERIOR ROOTS TO THE PROLONGED CORD. (After Edinger.)

(*b*) *The First Sensory Neuron of the Cerebral Nerves*

The intramedullary course of the first neuron of the sensory cerebral nerves which, corresponding to those of the posterior roots of the spinal cord, possess a root for each isolated sensory nerve, is a varying one. In the main, it resembles that of the spinal cord. Its *origin* is in the *peripheral ganglia* (apparently only included in it) (Fig. 73, see above) which, in the nerves of special sense (the optic, the auditory, the olfactory, is situated in the *end organs of special sense.* After they have entered the brain they terminate by *splitting up* around the clumps of nerve-cells which correspond to those nuclei of the funiculi gracilis and cuneati around which the ends of the spinal cord roots divide.

Formerly these nuclei were considered to be the origin of the sensory cerebral nerves. We now know, from the results of Golgi's method, that they *terminate* there,[1] and that from these cells other fibers originate which form a second neuron that we shall discuss later.

[1] In the nerves of special sense (the olfactory, the optic, the auditory) there are also fibers with a centrifugal course and of unknown function whose neuron cells also terminate in the above mentioned cerebral nerve nuclei.

If we remember these important relations of the sensory cranial nerve roots to their peripheral ganglia and to their cerebral nuclei, in *a description* of their intracerebral course we may for *special reasons* disregard the earlier view, that the fibers originate from the nuclei. I shall hereafter follow this plan.

I shall therefore begin with the *sensory nuclei in the medulla oblongata* around which the sensory nerves split up. If we recall the position of the motor nuclei, it will be easy to follow the sensory, particularly their relations to the motor. I therefore refer the reader to what has been said above, and to Figs. 72 and 73.

The *trigeminal nerve* has the longest, most extensive termination of all the cerebral nerves. Its nucleus begins far down in the *cervical cord,* even below the origin of the spinal accessory. The *gelatinous substance* of the posterior horns and their cells are the ones with which it unites. In the medulla oblongata the nuclei of the posterior columns and subsequently also other cerebral nerve nuclei force the gelatinous substance and the nucleus of the trigeminal nerve from their dorsal position and inward somewhat ventrally. The nucleus, however, retains its *lateral* position (Figs. 74, 75, 76), therefore remains external to the portions named above, the long motor cells in the medulla oblongata which form the origin of the first motor neurons of the brain. In the region of the *rhomboid fossa* it follows a *deep lateral course* up to the anterior middle of this structure, where the nerve itself from

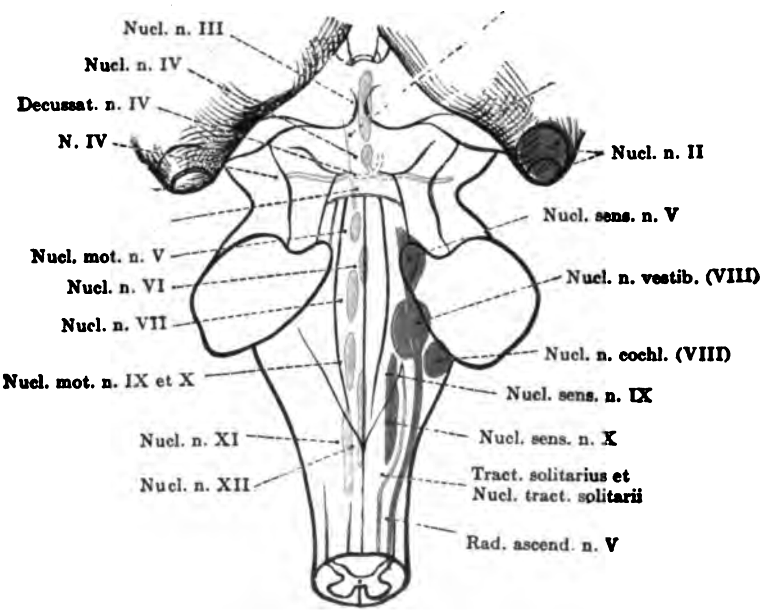

FIG. 72.—THE NUCLEAR ORIGIN OF THE CRANIAL NERVES. (After Toldt.)

without joins the nucleus as a thick bundle (from the Gasserian ganglion) accompanied at first by the motor root which we have already described, and passes from the base obliquely, from without and ventrally inward, and horizontally toward the dorsum through the external divisions of the pons (Fig.

77). After the fibers of the sensory trigeminal root have reached the nucleus just described, like all sensory nerve roots they divide into two branches (see above, the spinal cord roots). The shorter one passes to the nerve-cells near its point of entrance, while the other descends and gradually becomes smaller,

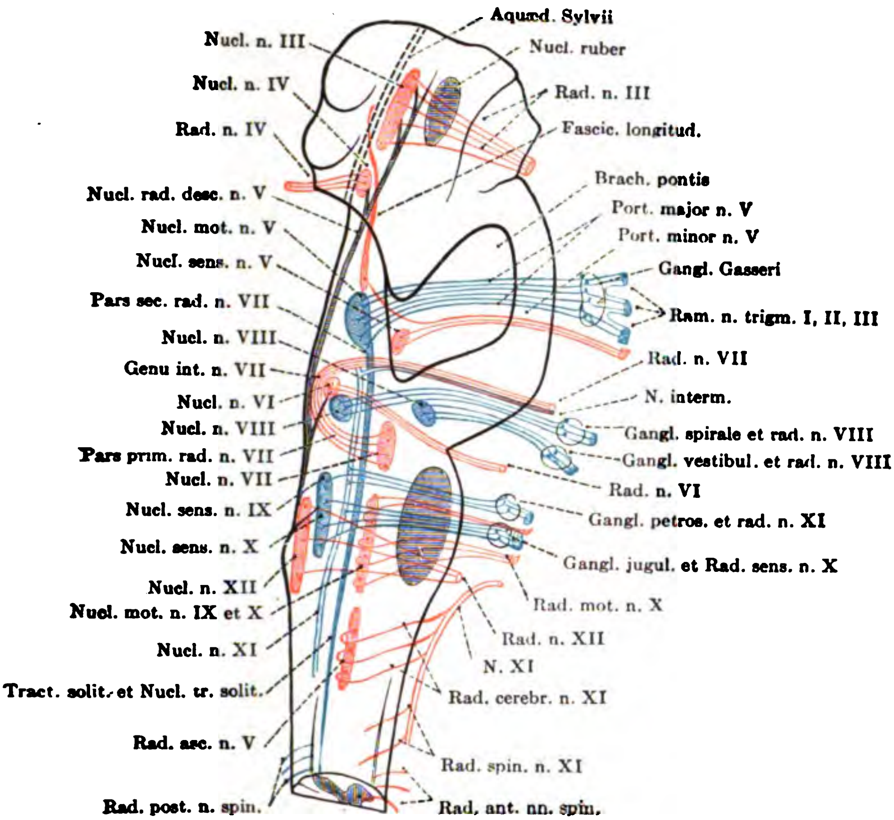

FIG. 73.—THE NUCLEAR ORIGIN OF THE CRANIAL NERVES. (After Edinger.)

supplying all the cells of the gelatinous substance down into the cervical cord (*ascending*[1] trigeminal root) (Figs. 72–76).

Now there is also an *ascending* trigeminal root (Figs. 72, 73, 96, 97, 98). This is distributed about nerve-cells which may be traced to the middle brain. Its most frontal position is in the region of the corpora quadrigemina, laterally from the aqueduct of Sylvius, therefore laterally to the nuclei of the oculomotor and trochlear, and at the same time somewhat toward the dorsum. Thence the nucleus of the descending trigeminal root extends caudally up to the lateral borders of the anterior portion of the fourth ventricle where it approaches the surface and where, by pigmentation of its cells, it becomes visible as the *locus cœruleus* (Fig. 78). There it unites with the *main nucleus of the trigeminal nerve.*

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[1] According to the neuron theory, the designation "descending root" would be the correct one.

We are still in doubt as to the relations of the descending trigeminal roots to this nucleus. It is uncertain whether the fibers of the nerve here split, or

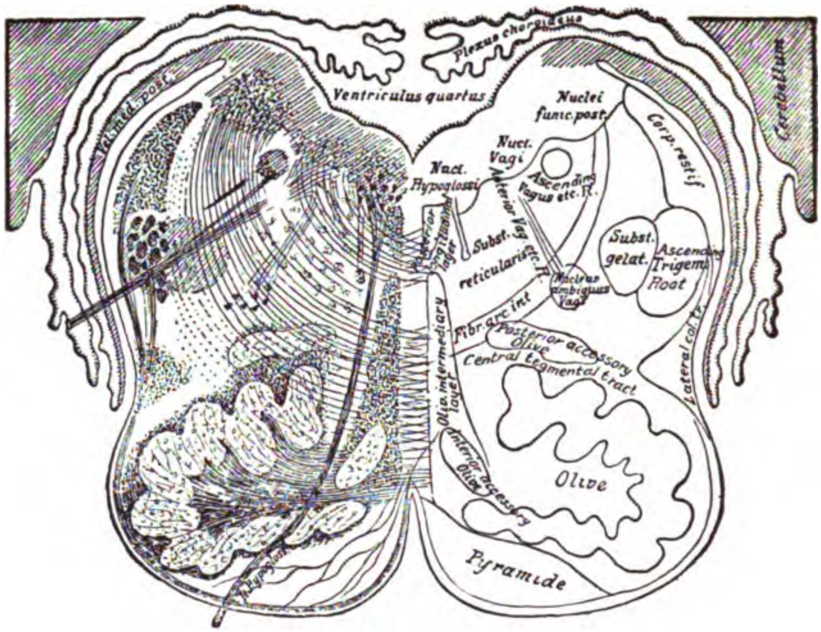

FIG. 74.—SECTION THROUGH THE MEDULLA OBLONGATA. (After Edinger.)

whether they rise at this point, and therefore constitute a descending centrifugal tract. In fact, the nerve fibers of this tract unite with those of the motor, not with the sensory root (radix mesencephalica).

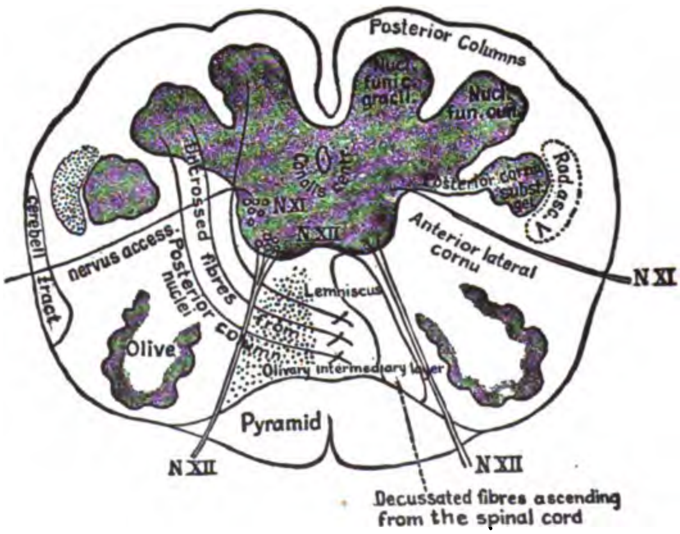

FIG. 75.—SECTION THROUGH THE MEDULLA OBLONGATA AT THE POINT OF EXIT OF THE PNEUMOGASTRIC. (Diagram after Edinger.)

We next consider the nuclei of the *pneumogastric*, the sensory root of which arises from the jugular ganglion combined with that of the glosso-pharyngeal which originates in the petrosal ganglion in the medulla oblongata close behind the auditory nerve (see below) between the olive and the restiform body (Fig. 77). The motor origin of the vagus in the nucleus ambiguus has already been described (Figs. 72, 73, 74). It is situated in that chain of motor cells the position of which about corresponds to the lateral horn of the cervical cord. Dorsally from this, in the position in which the posterior horns were formerly situated, and simultaneously forced to the interior by the nuclei of the posterior columns pressing to the side, is the *sensory nucleus* of the pneumogastric (Figs. 72, 73, 74, 76). It originates in the medulla

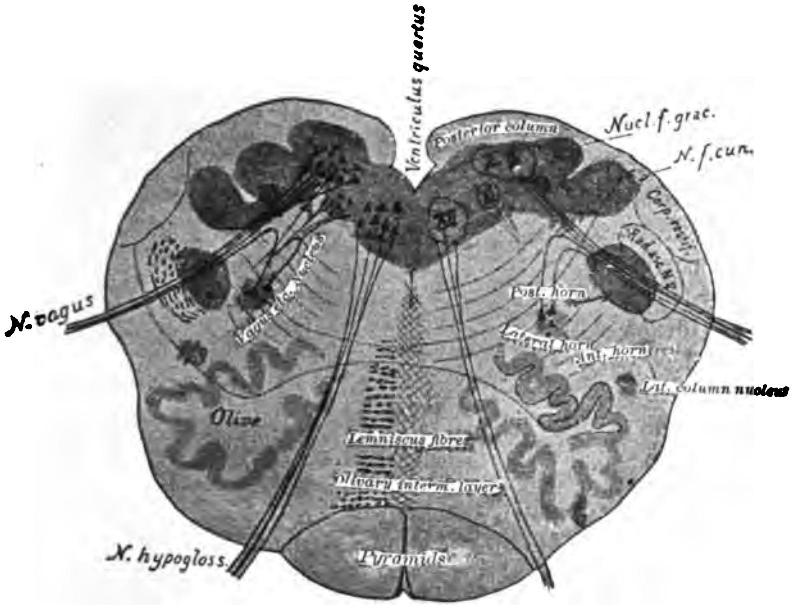

FIG. 76.—SECTION THROUGH THE MEDULLA OBLONGATA AT THE HEIGHT OF THE POSTERIOR HYPOGLOSSAL ROOTS. (Diagram after Edinger.)

about at the point where the olives trend somewhat more ventrally, and passes frontally as far as the beginning of the second third of the fourth ventricle. Its anterior point is, of course, to be distinguished as the nucleus of the *glosso-pharyngeal* nerve. The nucleus of the vagus is situated upon the floor of the fourth ventricle laterally to that of the *hypoglossal nerve* (Figs. 72, 73, 74, 76), and here its *dark pigmented* glistening cells form the *ala cinerea* (Fig. 78).

The accumulation of cells just described does not, however, form the only nuclei of the pneumogastric. The fibers of the vagus root, like the fibers of the trigeminal with their descending branches, the so-colled *ascending root*, unite with cells which, like the ascending nucleus of the trigeminal nerve, may be followed down into the cervical cord, although less far, and communicate with the cell group fibers of the *ascending root of the glosso-pharyngeal nerve*. These cells, like those of the trigeminal nerve and parallel with them,

lie embedded in a *gelatinous substance* with a somewhat more dorsal and more medial inclination and penetrate close to the nuclei of the posterior columns. This ascending glosso-pharyngeal-pneumogastric root also bears the name *tractus solitarius* (Figs. 73, 74, 92, 93, 94).

The *auditory nerve* consists of two nerve roots which pass to the *cochlea* and to the *vestibule* of the ear. *The first branch only* appears to be connected

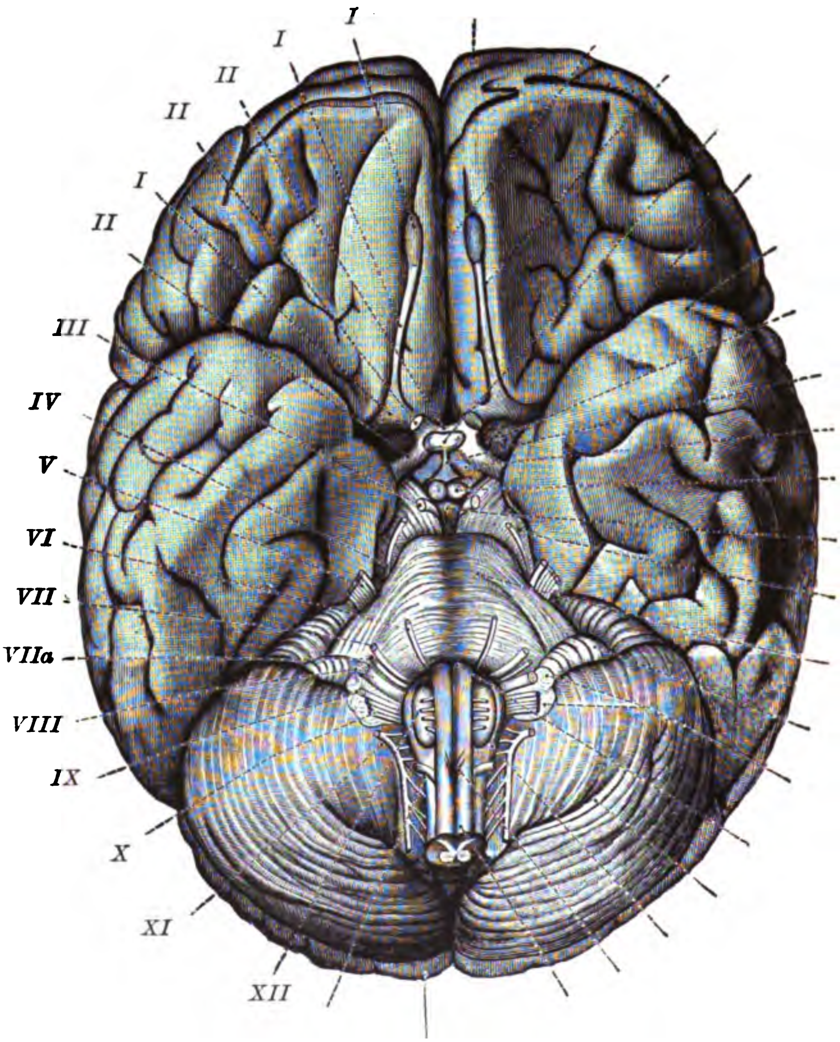

FIG. 77.—BASE OF THE BRAIN AND CRANIAL NERVES.

with the sense of hearing, while the second is probably the chief *nerve of tonus and coördination.* One, the *cochlear nerve,* springs from the cells of the *spiral ganglion* of the cochlea. Like the spinal ganglion cells, the cells of this ganglion also send out a process which soon divides into two parts. The short peripheral end runs to the auditory cells of the cochlea, the other, the

ventral, forms a part of the root of the auditory nerve. The *vestibular nerve* originates from the *ganglia of the labyrinth* and from others which lie in its central course; both its processes are divided like those of the cochlear. The peripheral process extends to the cells of the ampulla, and the central forms the other trunk of the *auditory nerve.* Thus the combined roots of the eighth nerve pass laterally to the central organ alongside of the facial at the posterior border of the pons between the external border of the olives and the restiform body (Fig. 78).

The divisions of the auditory nerve *do not unite in the interior of the medulla oblongata.* On the contrary, the cochlear nerve, whose root fibers

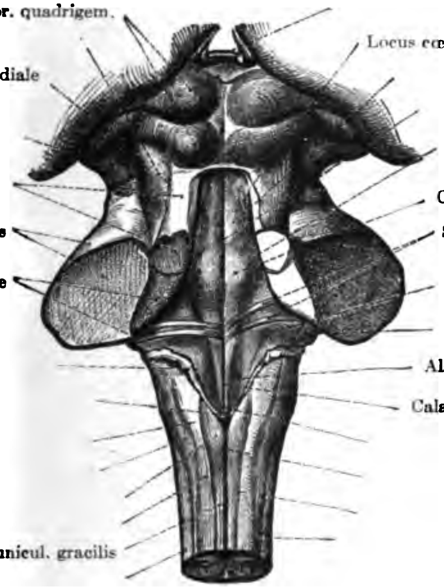

FIG. 78.—MEDULLA OBLONGATA WITH THE FOURTH VENTRICLE AND THE CORPORA QUADRIGEMINA, SEEN FROM ABOVE. (After Toldt.)

branch posteriorly and outwardly from those of the vestibular nerve soon after its entrance into the posterior border of the pons, penetrates the ventral acoustic ganglion (Figs. 78, 79, 94, 95) which upon transverse section is found to be situated in the most lateral region of the pons ventrally and somewhat outwardly from a section through the restiform body, in an area which may be recognized from the flocculus of the cerebellum by a small protuberance; a few fibers of the cochlear nerve pass for a short distance toward the cerebellum and then enter a second small nucleus situated there, the *tuberculum acousticum* (Edinger). The *vestibular nerve* (Edinger, Freud) passes to a second acoustic nucleus, the dorsal (Figs. 94, 95). While the ventral nucleus is external to the other sensory nuclei of the medulla, the dorsal is within the area of the *latter structure* (Figs. 64, 85, 86). It is situated directly upon the floor of the fourth ventricle in its most lateral portion. It runs forward laterally from the anterior division of the nuclei of the glosso-pharyngeal and pneumogastric nerves to the anterior third of the fourth ventricle, so that nearer the front it lies outside of the nucleus of the abducens and the genu

of the facial. The *striæ acousticæ* (see below) are still within its area. The ascending trigeminal root is invariably situated below them ventrally, while the large nucleus has a more frontal situation.

The first neurons of the *organs of sight and smell* are still to be described.

The *optic nerve* which originates from the ganglion cells of the retina is divided into two branches of which the peripheral *goes to the rods and cones*

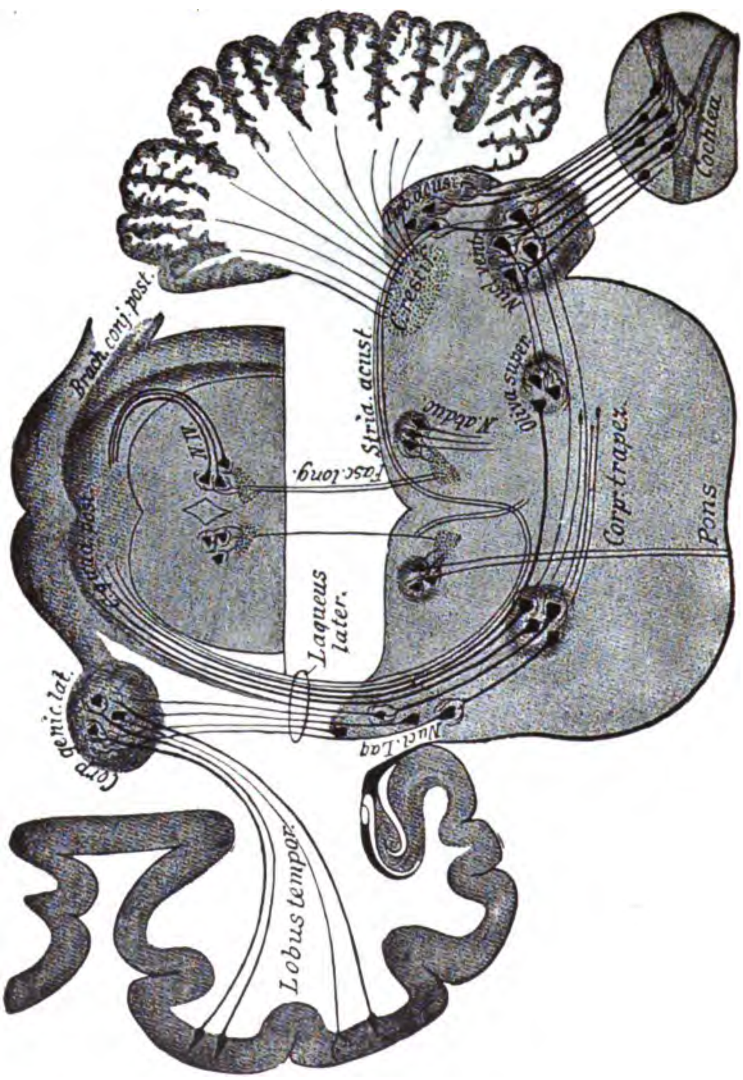

FIG. 79.—DIAGRAM OF THE COURSE OF THE COCHLEAR NERVE. (After Edinger.)

while the central passes toward the brain, and after forming the well-known chiasm at the base of the brain (in front of the tuber cinereum) encircles the cerebral peduncles as the *optic tract.*

The posterior portion of the optic tract disappears under the gyrus hippocampi and the uncus of the temporal lobes which must be lifted if we would

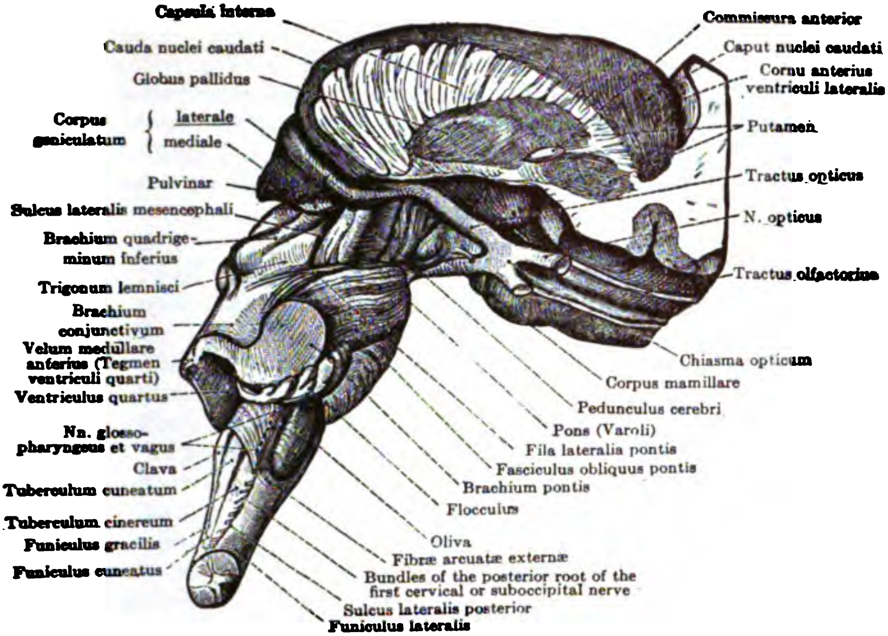

FIG. 80.—REGION OF THE CORPORA QUADRIGEMINA, THALAMUS, AND MEDULLA, FROM THE RIGHT SIDE. (After Toldt.)

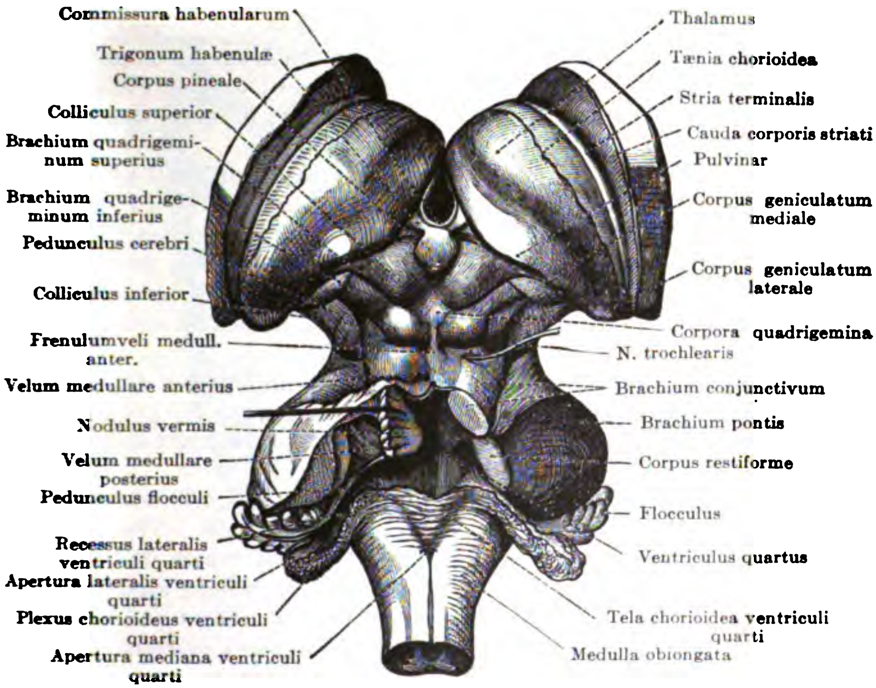

FIG. 81.—CORPORA QUADRIGEMINA, THALAMUS, AND MEDULLA, FROM ABOVE. (After Toldt.)

follow the tract farther (Fig. 43). We then perceive that after the optic tract has passed around the cerebral peduncles (Fig. 80) it sinks dorsally into the roof of the middle brain, the characteristic portions of which are formed by the corpora quadrigemina (Figs. 81, 82). Just before it enters the middle brain it shows upon its lateral aspect a small protuberance, the *corpus geniculatum laterale.* A few of the fibers enter the nerve-cells, but the majority pass on in two directions. Some run through the anterior superior peduncle which leads from the superior peduncle to the internal capsule, and thus enter the *nerve-cells of the superior peduncle itself.* The other enters the *pulvinar and the stratum zonale of the thalamus opticus.*

The *olfactory nerve* receives its neuron cells in the cells of the *olfactory mucous membrane.* Its processes pass as fibers of the nerve of smell through the *lamina cribrosa* at the base of the brain, and there enter the *bulbus olfactorius* (Fig. 77). Here the first division of the tract of smell terminates in cells which are situated in the gray substance in the interior of the bulbus. Much of the interest and importance of the neuron theory centers about the nature of the termination, namely, the terminal filaments which enter into the bulbus, the primary neurons of smell combined with the dendrites of the cells of the second division (see below) in the bulbus. This communication takes place in the so-called *glomeruli,* which form a layer in the bulbus immediately below its external white medullary covering, and which is dimly perceptible macroscopically (Fig. 83). I shall discuss later the complicated structure of the tract of smell.

(2) THE SECOND SENSORY NEURONS

The peripheral neuron is always designated as the first. In describing the second sensory neuron I shall delineate the course of those nerve processes of the cells which we have learned to recognize as the terminal nuclei of the first sensory neuron, and around which they split up.

Before describing the path of the sensory fibers, I must call attention to one point. In the description of the first sensory neurons I stated that many collaterals are constantly given off by the fibers. In the motor tract we recognize the same tendency to unite with the branches of other functionating neurons. In the sensory tract, however, especially in the first sensory neuron, the conditions are complicated by a multiplicity of collaterals and communications.

It will be remembered that, while giving off lateral branches (collaterals), some of the posterior roots of the spinal cord pass through the gray posterior horns, others through the white posterior columns, in a manner which has been described.

The fibers which pass into the posterior columns send collaterals, or even terminal distributions, into the motor anterior horn cells. These are the *reflex tracts* (*reflex collaterals*) (Figs. 68, 69, 84, 85), which unite the first sensory with the first motor neuron (Fig. 88) and which, therefore, have no central sensory continuation. Other posterior root fibers and collaterals of the gray substance communicate with *cells of Clarke's columns* (columna vesicalis); this is particularly marked in the dorsal cord, and takes place at the base of the posterior horn just where the posterior gray commissure arises.

A second sensory neuron does not develop from the cells which here surround it. On the contrary (Figs. 85, 86), the fibers of these cells pass upward in the spinal cord as a neuron of the cerebellum, they enter the lateral column, and emerge at its external periphery as the cerebellar lateral column tract (Figs. 85, 86). Finally, some posterior root fibers from the posterior horn terminate in cells in but a few of which short tracts develop inside of the spinal cord (Figs. 38, 84) (*column cells, association tracts*).

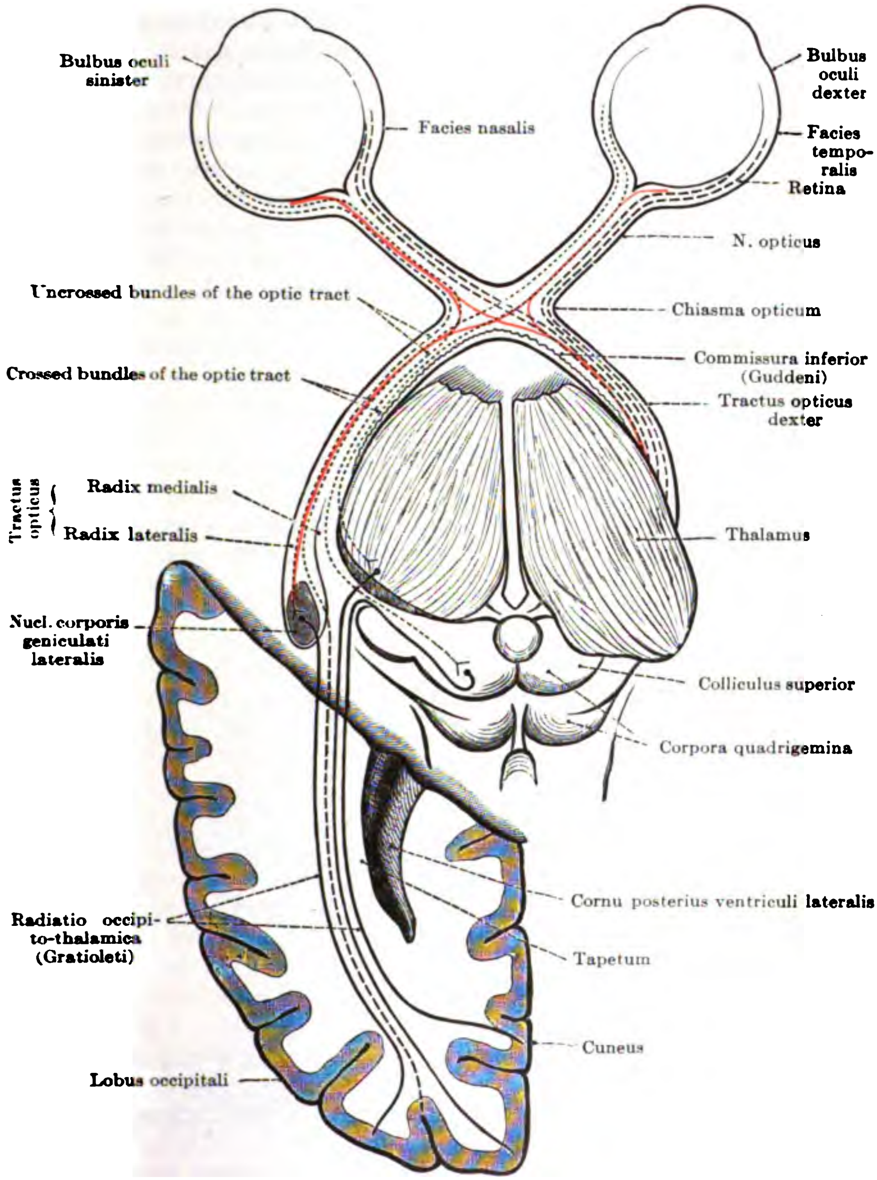

FIG. 82.—TRACT OF THE OPTIC NERVE (FIRST NEURON RED). (After Toldt.)

The ramifications of the sensory cranial nerve roots are similar, in so far as they have been investigated.

The first sensory neuron, with manifold combinations, finally communicates with the actual second sensory neuron. It arises from those nuclei which have already been described as *terminal nuclei* of the first sensory tract, and the course of these fibers we will now follow.

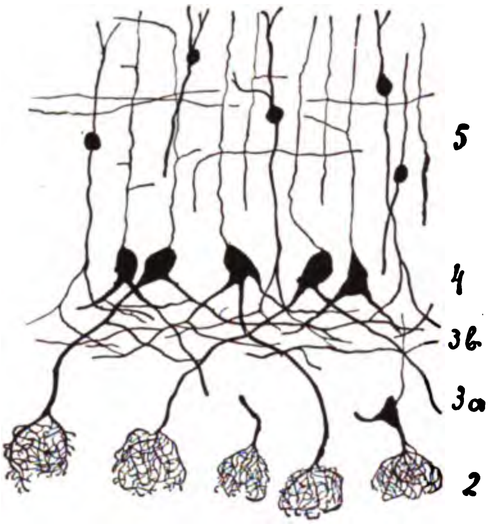

FIG. 83.—THE BULBUS OLFACTORIUS OF THE MOUSE. (After Obersteiner.)

The cells from which the second sensory neurons originate are found in large numbers in the posterior horns of the spinal cord. Their processes pass transversely through the gray substance, first anteriorly into the anterior commissure, and *subsequently upon the other side* (Figs. 84–86) they enter the *white substance of the lateral columns*, and pass through that structure which we designate the *lateral column basic bundle*. Thence they pass upward into the medulla oblongata. In consequence of the alteration in the parts due to pyramidal decussation they make a bend toward the middle, and pass between the lower olives which lie at either side of the median line (Fig. 86).

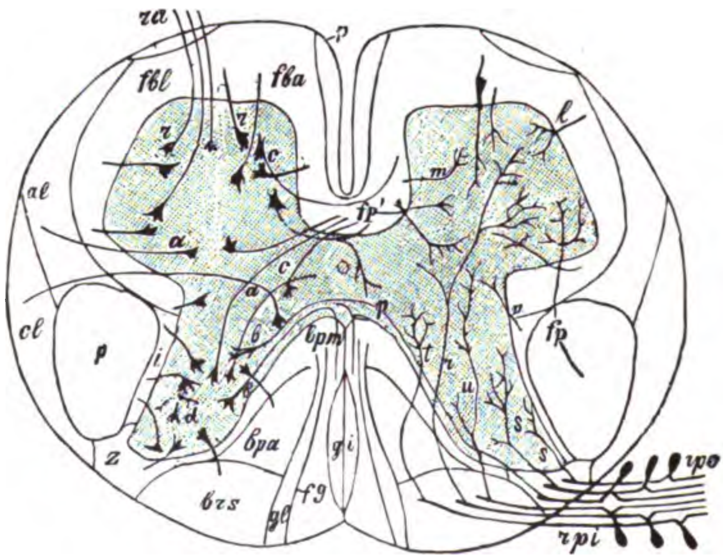

FIG. 84.—DIAGRAM OF THE ORIGIN AND TERMINATION OF THE NERVE ROOTS IN THE GRAY SUBSTANCE OF THE SPINAL CORD AND THE DISTRIBUTION OF THE NERVE-CELLS OF THE LATTER. (After Bechterew.)

Here they receive the name lemniscus (or laqueus, formerly olivary intermediary layer) and pass on to the frontal region.

Before following the path of the lemniscus we must return to those *posterior root fibers* which ascend in the *posterior columns of the spinal cord.* As has been mentioned, they extend to the medulla oblongata where they terminate in numerous cells which arise at the height of the pyramidal decussation, the nuclei of Burdach's columns and of Goll's columns (Figs. 53, 87, 91, 92). These nuclei may be traced as far as the beginning of the fourth ventricle. After this the fibers in the posterior column gradually become more sparse, and finally disappear completely.

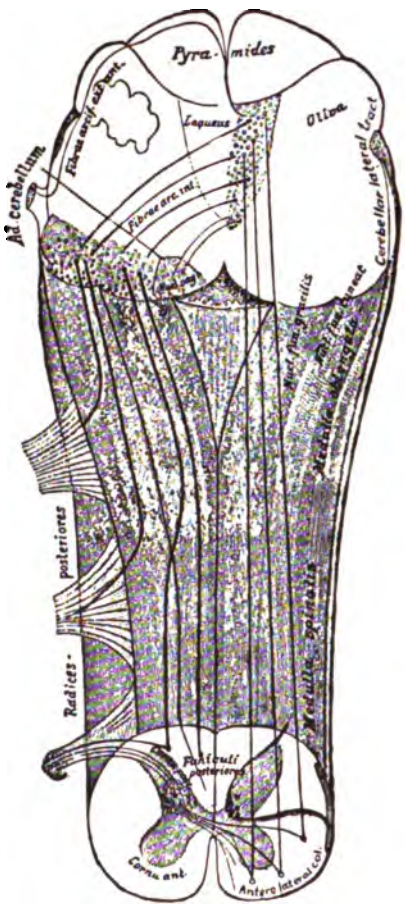

FIG. 85.—DIAGRAM OF THE COURSE OF THE SENSORY TRACTS FROM THE POSTERIOR ROOTS TO THE MEDULLA. (After Edinger.)

From the *cells of these nuclei* nerve fibers originate which form the *sensory neurons of the second division* (Figs. 85, 87). They pass to those fibers which extend from the lateral column of the spinal cord to the median line at the so-called lemniscus (olivary intermediary layer). In order to reach these fibers those from the dorsal side of the medulla must reach a zone situated more ventrally and medially, and thus they pass in an arc ventrally and inwardly from the nuclei of the posterior columns. At the same time *they all cross the median line* in the "raphé," unite with the lemniscus upon the opposite side, and with this they ascend. Until these fibers of the nuclei of the posterior column enter the lemniscus they are called *fibræ arcuatæ internæ* (Figs. 85, 91, 92). Thus we find a *second sensory crossing* above the pyramidal decussation. All sensory fibers of the second neurons, like the motor, find their way to the brain by crossing over. Those which pass from the lateral columns of the spinal cord into the lemniscus without crossing have passed *through the anterior commissure of the spinal cord from the posterior horn of the other side*, while the fibræ arcuatæ internæ cause the crossing of the sensory tract from the posterior columns which until then had not been made. Hence the second sensory neuron of the posterior roots of the spinal cord ascends to the medulla oblongata in the *lemniscus on both sides of the raphé.* We must trace this through the pons and the middle brain.

The fibers of the *second sensory neuron of the cranial nerves* now unite, and, the lowest, the nucleus of the pneumogastric nerve (Fig. 87) whose cells

form the second neuron, sends its fibers through the raphé to the most dorsal region of the *crossed* lemniscus. The same is true of the nucleus of the *glosso-pharyngeal nerve.* (Probably the ascending roots of the glosso-pharyngeal and vagus send crossed fibers into the lemniscus tract; no positive knowledge concerning this is at hand.)

The *relations* of the *auditory nerve* are more complicated. These arise at the height of the pons in the median portion of which, above the pyramidal

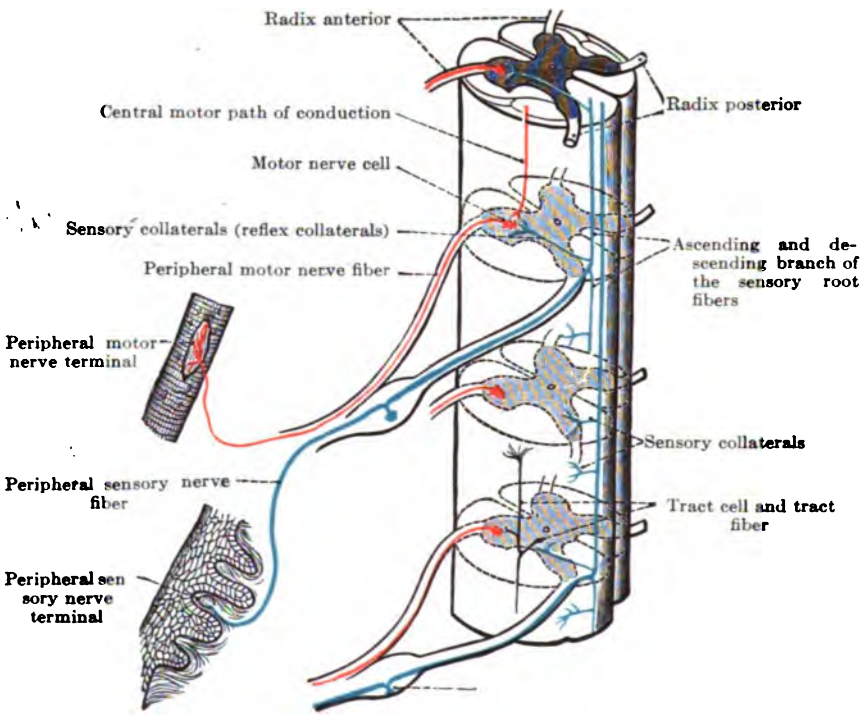

FIG. 86.—DIAGRAM OF THE MOTOR AND SENSORY CONDUCTION TRACTS (REFLEX ARCS). (After Toldt.)

tract, the lemniscus tract passes upward. The nuclei of the acoustic nerve have been described (Figs. 88, 89, 94, 95). The ventral nucleus, situated ventrally and laterally near the entrance of the nerve, contains some of the second neuron cells. These fibers pass horizontally and medially through the pons between the pyramidal and lemniscus tracts, and cross to the other side. This tract is called the *corpus trapezoideum* (Figs. 88, 89, 95). Large numbers of nerve-cells are here deposited. Directly above them dorsally and immediately below the nucleus of the facial nerve is a small collection of ganglia, the *upper olive,* into which large numbers of these fibers penetrate.

Many fibers of the corpus trapezoideum appear to split up around their own cells as well as around the cells of the upper olive, upon the same side as well as after crossing to the opposite side.

From the nuclei of the corpus trapezoideum and the upper olive other

fibers pass to the *nerve of hearing* where they mix with the remaining fibers of the corpus trapezoideum in so far as these have not united with cells, and subsequently as the lateral *lemniscus* (see below) they pass to the middle brain (Figs. 87, 96).

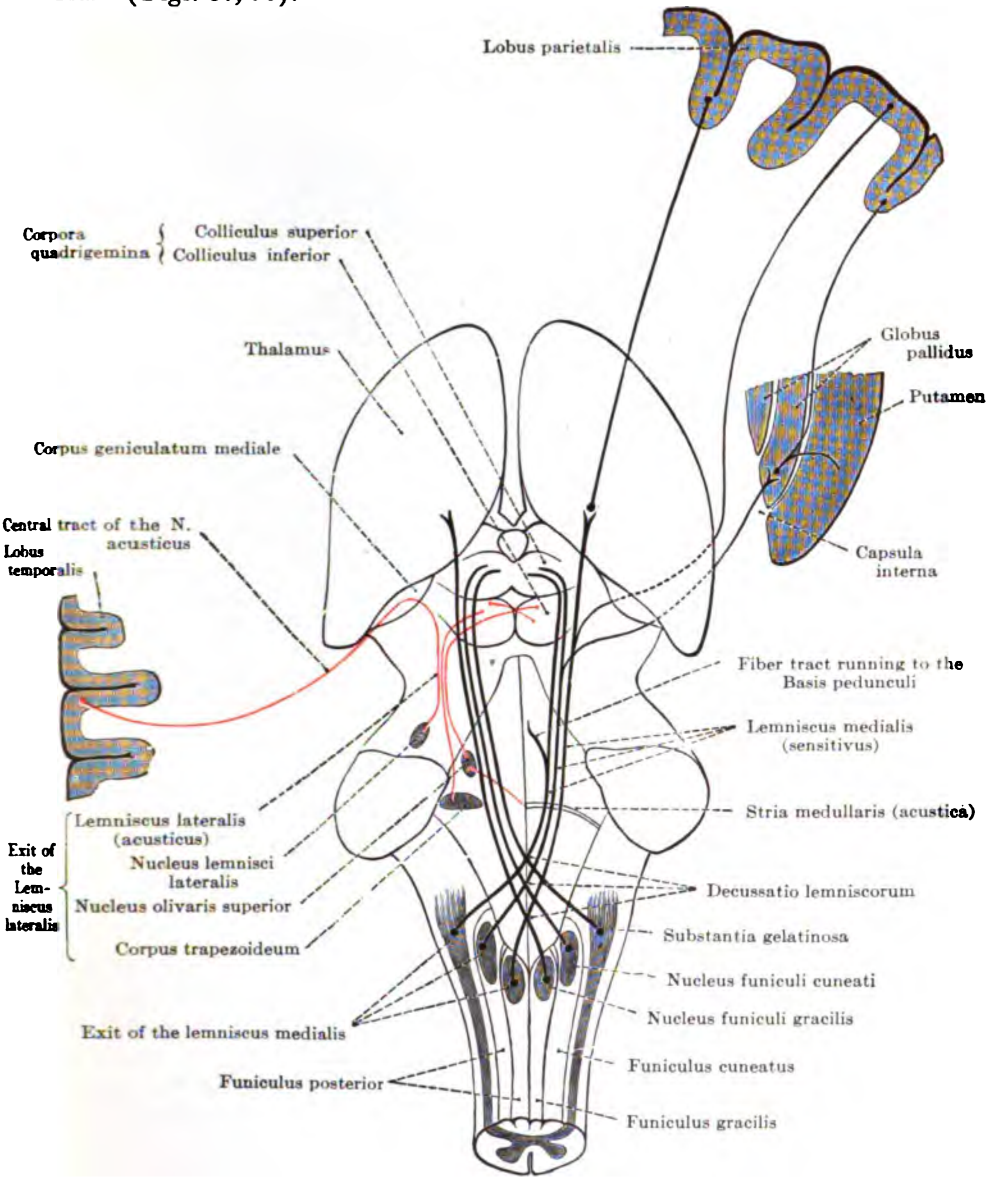

FIG. 87.—DIAGRAM OF THE LEMNISCUS TRACT (THE MEDIAL IN BLACK, THE LATERAL IN RED).

The *tuberculum acusticum* of the nerve of hearing sends its nerve fibers dorsally around the corpus restiforme into the *striæ acusticæ* of the rhomboid fossa (Fig. 94). They pass through the middle of this, and then sink ven-

trally in the raphé down to the level of the corpus trapezoideum and ascend in the lateral lemniscus together with fibers from the corpus trapezoideum and its nerve nuclei.

The path of the fibers of the *dorsal nucleus of the auditory nerve* (vestibular nerve) is still unknown.

As to the second neuron of the *trigeminal nerve,* i. e., the fibers of its elongated sensory nucleus which pass out centripetally, and which for a long

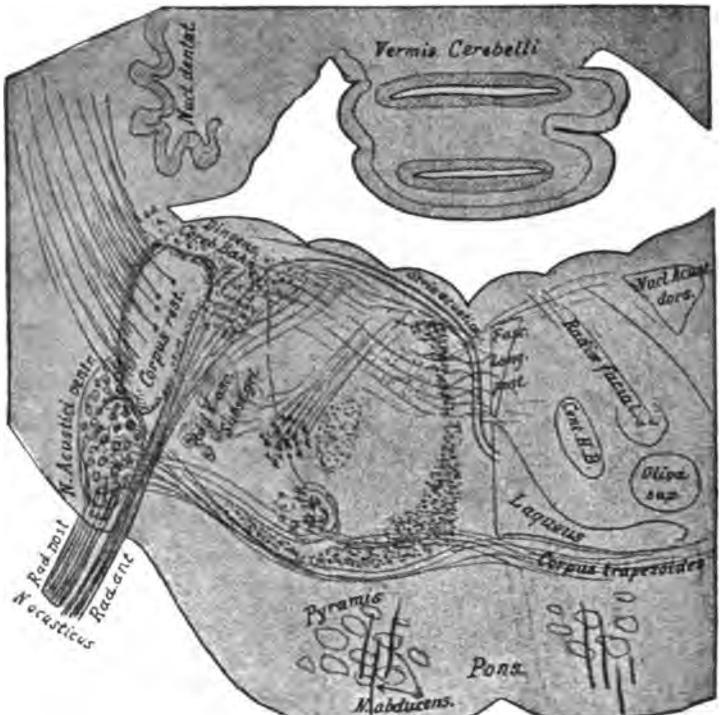

FIG. 88.—ORIGIN OF THE FACIAL NERVE AND ITS SURROUNDINGS, THE FIBERS OF THE AUDITORY NERVE AND ITS NUCLEI. (After Edinger.)

time could not be traced, the subject has been illumined by the investigations of Edinger, Wallenberg and Ramón. On the opposite side of the medulla oblongata they pass upward into a region which is designated as the *substantia reticularis* or as Bechterew's *central tegmental tract* (Figs. 89, 94–96); that is, such a tract has been located in animals. It extends dorsally and somewhat laterally from the median lemniscus up to the middle brain and through this to the thalamus.

In the middle brain, therefore in the region of the corpora quadrigemina, the *lemniscus* appears as the *continuation* of the second sensory neuron of the spinal cord, the glosso-pharyngeus-vagus, the auditory, and probably also of the trigeminal nerve. Here it extends dorsally from the substantia nigra, but ventrally below the decussation of the peduncles, to the cerebellum, and frontally below the red nucleus (Fig. 99), the origin of these peduncles. The

lateral lemniscus disappears in the region of the anterior corpora quadrigemina.

I must now state the *difference between the medial and lateral fibers of the lemniscus.* The medial (upper) lemniscus contains the continuations of

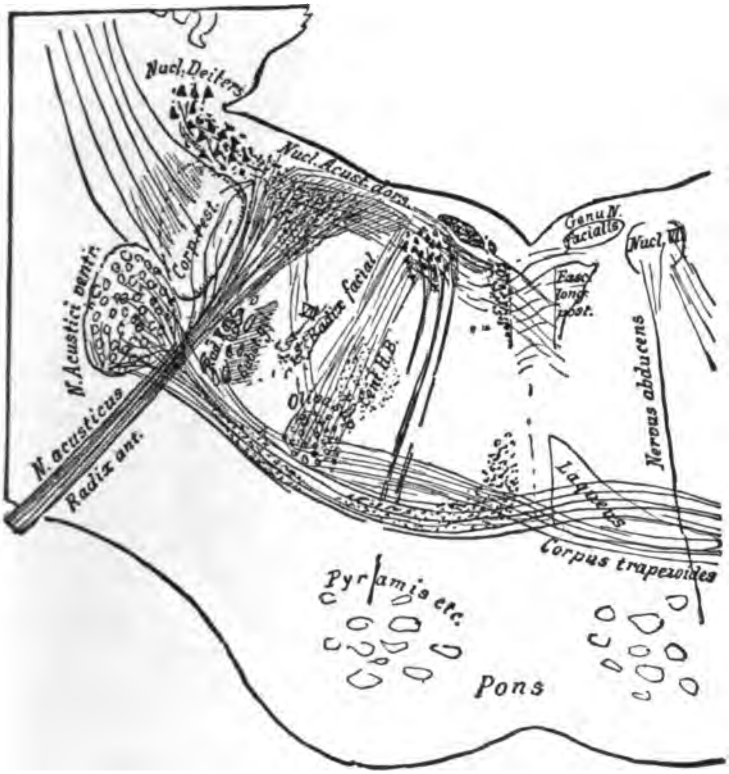

Fig. 89.—Section in the Region of the Origin of the Abducens. The Vestibular Nerve (After Edinger.)

the fibræ arcuatæ internæ, therefore rises from the nuclei of the *posterior columns* (Fig. 87). To these are added the fibers from the lateral column basic bundles of the spinal cord. In the tegmentum it passes below the corpora quadrigemina to the inter-brain, in the thalamus, where its ventral

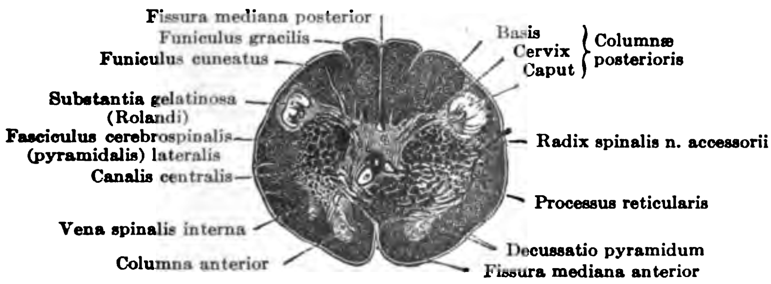

Fig. 90.—Transverse Section through the Pyramidal Decussation. (After Toldt.)

nucleus breaks up; this is, therefore, one of the ends of the second sensory neuron.

The lateral (lower) lemniscus probably contains the fibers of all sensory *nerve nuclei* (glosso-pharyngeal, vagus, acousticus and trigeminal nerves)

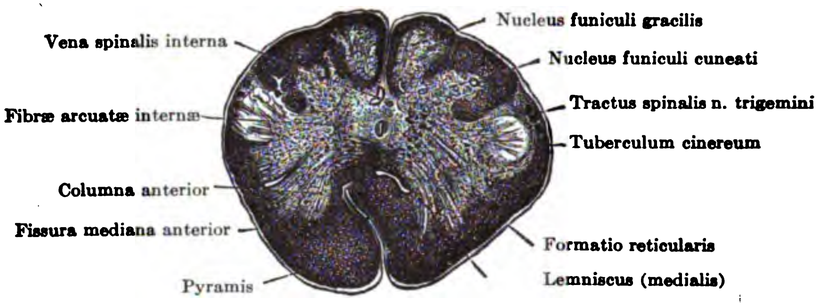

FIG. 91.—TRANSVERSE SECTION THROUGH THE MEDULLA BELOW THE OLIVES. (After Toldt.)

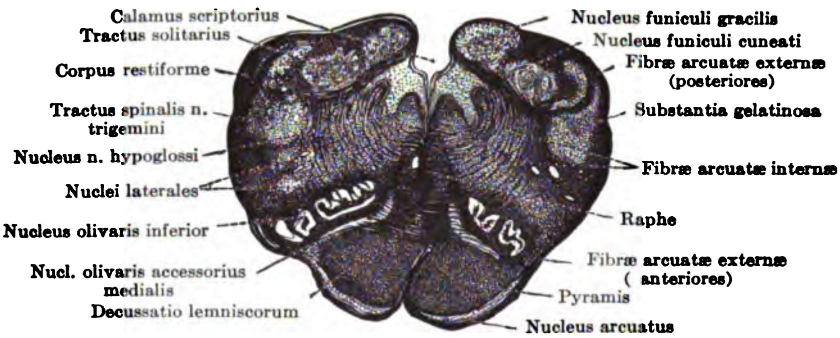

FIG. 92.—TRANSVERSE SECTION THROUGH THE LOWER PORTION OF THE OLIVES AND THE FOURTH VENTRICLE. (After Toldt.)

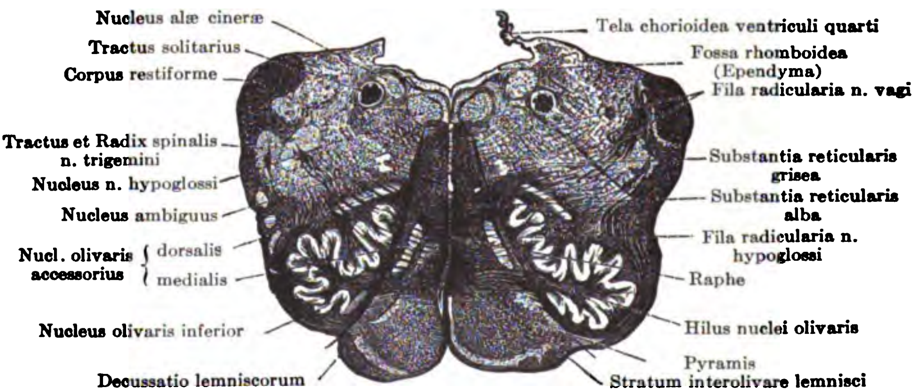

FIG. 93.—TRANSVERSE SECTION THROUGH THE MIDDLE OF THE OLIVE AND THE LOWER PORTION OF THE FOURTH VENTRICLE. (After Toldt.)

from the medulla oblongata. It forms the external side of the tegmentum, and in the area of the corpora quadrigemina it invariably passes in a dorsal

direction until it terminates at various places in the region of the anterior corpora quadrigemina. Its fibers enter partially into both corpora quadrigemina, in part they are arrested beforehand in the ganglia which are simultaneously deposited within it (lateral and upper lemniscus nucleus). A few

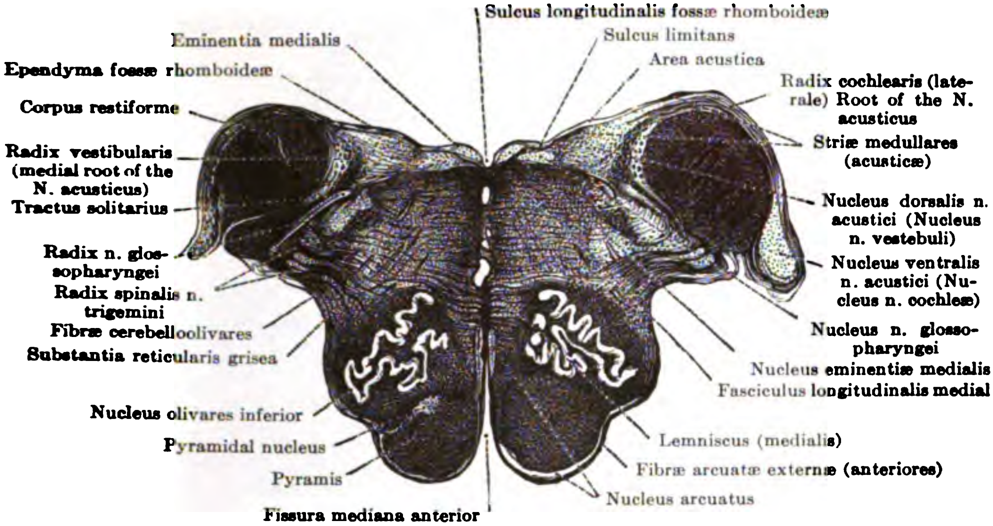

Fig. 94.—Transverse Section through the Middle of the Fourth Ventricle. (After Toldt.)

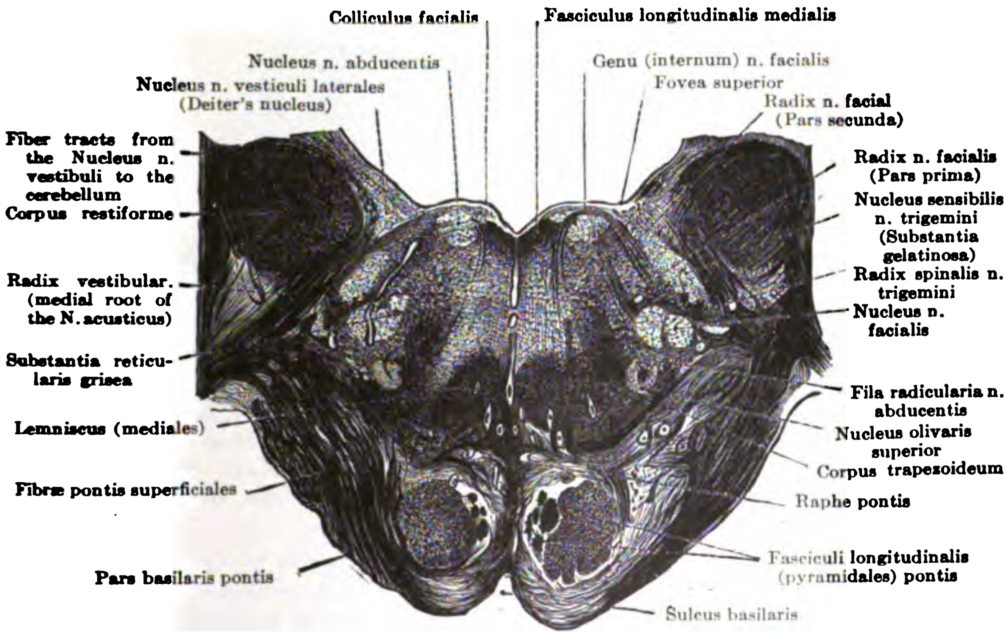

Fig. 95.—Transverse Section through the Lower Border of the Pons Immediately Above the Olives and through the Middle of the Fourth Ventricle. (After Toldt.)

fibers (fibers of the acoustic) terminate in the medial geniculate body. The course of the lemnisci may well be followed in the illustrations from 90 to 99, which clearly portray the general position of the tracts in the trunk of the brain.

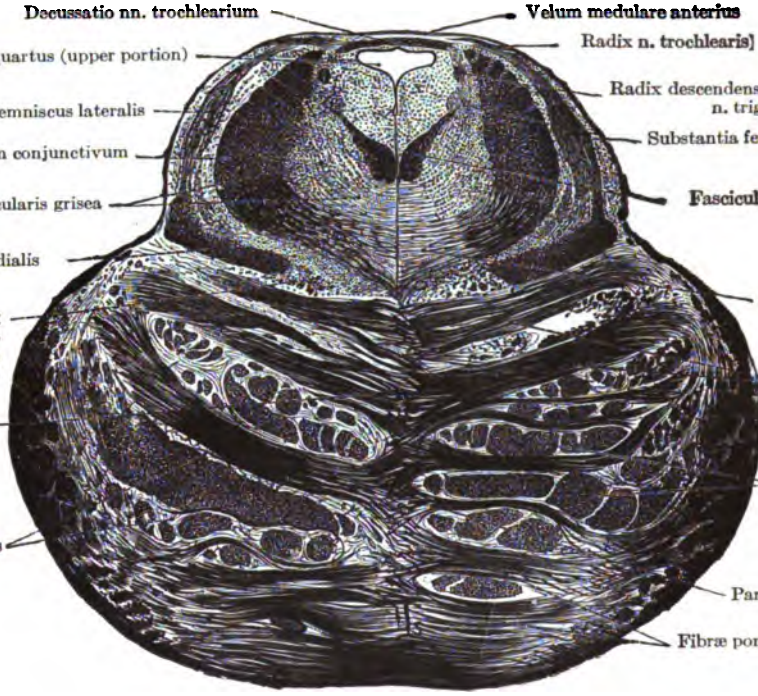

FIG. 96.—TRANSVERSE SECTION THROUGH THE MIDDLE OF THE PONS AND THE ANTERIOR MEDULLARY VELUM. (After Toldt.)

We must now study the continuation of the optic and olfactory nerves after leaving their nuclei. For a reason to be immediately mentioned, I shall omit the secondary optic tract, and shall pass at once to the olfactory. This rises from the cells of the bulbus olfactorius which have already been described as their "glomeruli." From this point the fibers run but a short distance only. They gradually pass into the *gray substance of the optic tract* which immediately joins the bulbus posteriorly and terminate in its cells. Another part appears to run on posteriorly to the point where the tractus olfactorius arises from two roots, one a distinctly visible *white lateral root*, and a medial one which is grayish and can be recognized only with difficulty. This latter enters the cells of the gray substance in this region which is designated as the *substantia perforata anterior* (Fig. 77).

FIG. 96 A.—SECTION THROUGH THE PONS.

The fibers of the lateral root pass on posteriorly into the *temporal lobes.* Later we will again follow them.

In regard to the *secondary optic tract,* we must assume that the external geniculate body, the anterior corpora quadrigemina, and the pulvinar of the thalamus neurons of the second division develop from the three terminal stages of the optic tract, like those in the lemniscus.

The fibrillations of the first terminal stages of this *nerve,* so far as we know at present, pass *directly to the cortex of the brain* without ending in nuclei extending toward the cauda like the fibers of the lemniscus. Therefore this higher optic tract does not correspond to the second, included within the temporal tract, but to that *third sensory neuron* which is now to be described.

(3) THE THIRD SENSORY NEURONS

The complicated mechanism of the sensory conduction tract compared with the motor, which is evident from this description, is explained by the fact that in man as well as in all vertebrate animals there is a *third sensory neuron* in addition to the two neurons we have discussed. The marked devel-

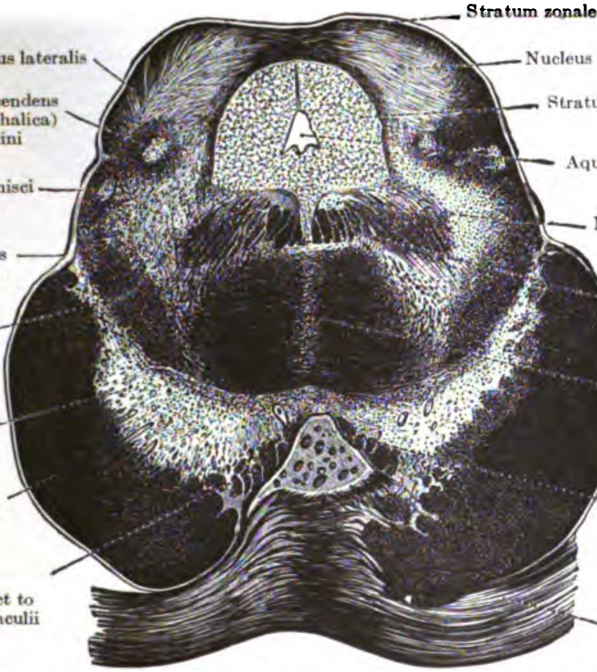

FIG. 97.—TRANSVERSE SECTION THROUGH THE UPPER MARGIN OF THE PONS AND THE POSTERIOR CORPORA QUADRIGEMINA. (After Toldt.)

opment of the anterior brain (cerebrum) in the primates, the higher stage of conscious sensation, are probably intimately related to this. It is scarcely necessary to state that *collaterals* in an unentwined web pass to other centers of other neuron groups with unknown functions, especially in the cortex of the brain itself, and are combined in various ways with others in the sensory tract.

The third sensory neuron is combined with the second in the cortex of the brain. The fibers of its origin must be sought in those *nuclei* in which the *lemniscus ends* (Fig. 97) and also at the terminal ends of the primary optic and secondary olfactory fibers which have been described. In the optic nerve the second neurons must be considered the equivalent in function of the third neurons of the remaining sensory tracts (Fig. 82). The

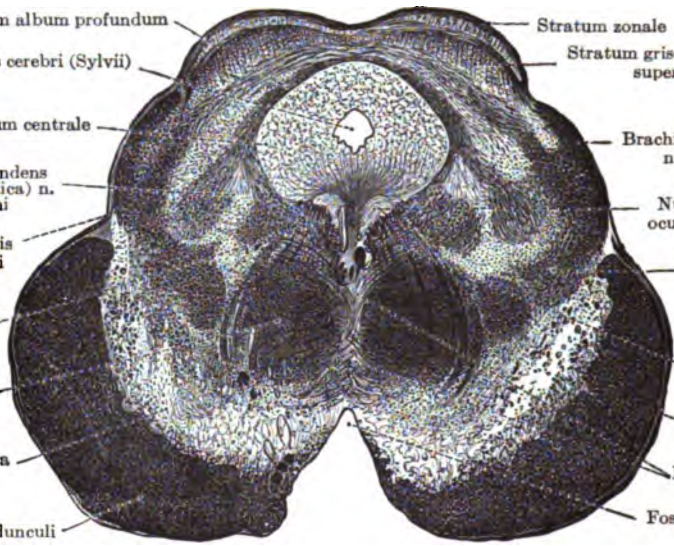

FIG. 98.—TRANSVERSE SECTION THROUGH THE UPPER CORPORA QUADRIGEMINA, THE TEGMENTUM, AND THE CRUS CEREBRI. (After Toldt.)

lemniscus is supposed to carry all of the second sensory neurons from the spinal cord and the medulla oblongata up to the nuclei of the nerves of the mid-brain and to the thalamus. The second sensory neuron of the trigeminal nerve, certainly in the rabbit, is the only one that leads somewhat dorsally and laterally up from the median lemniscus, chiefly through the thalamus; in man this is still uncertain.

The *relation of the lemniscus to the cortex of the brain* must now be outlined. This is brought about by the *corona radiata* fibrillation which has already been referred to. It extends as a large projection tract into the medullary mass of the hemisphere which we designate as the centrum semiovale. The motor cortical tract also runs through the corona radiata bundles.

The path that the sensory tract pursues from the terminal nuclei in the middle brain and in the inter-brain to the cortex is, therefore, a devious one. Its extent over the cortex is much greater than that of the motor tract. Hence we find united here the fibers of the lemniscus which come from the nuclei of the mid-brain (Figs. 98 and 99) (medial lemniscus nucleus and nucleus of the corpora quadrigemina, geniculate body). They pass *through the so-called regio-subthalmica in the tegmentum under the thalamus* (Fig. 63), *obliquely outward and upward into the internal capsule*, and there terminate (Fig. 60) close behind the motor tract.

Fibers from the nucleus of the optic, from the *geniculate body*, from the

anterior corpora quadrigemina and the *thalamus* are added to this tract originally coming from the nuclei of the *medulla oblongata.* The *optic tract* and the *auditory tract* are in the posterior part of the internal capsule (see above) behind the remaining sensory bundles. The *central olfactory tract only has a different course.* Frontally, however, the sensory fibers diverge in various directions from the internal capsule of the cortex of the brain. The majority of the fibers pass longitudinally upward into the corona radiata fibrillation of the parietal lobes, and this is designated as *tegmental radiation.* It was formerly believed that the sensory fibers in this cortical region of the tegmentum (tegmentum, Figs. 98, 99) of the cerebral peduncles passed uninterruptedly into the medulla. We now know that they traverse the tegmentum for some distance but not unbrokenly, for their course is interrupted by the

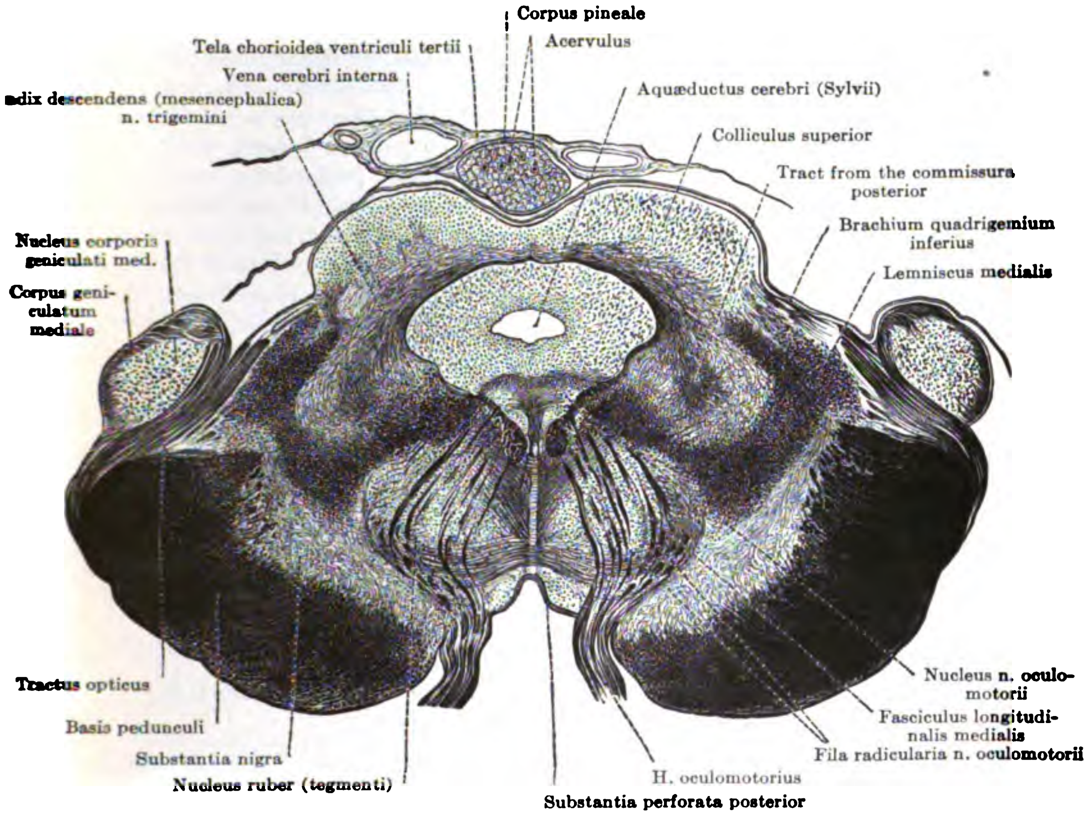

FIG. 99.—TRANSVERSE SECTION THROUGH THE UPPER CORPORA QUADRIGEMINA IN ITS MOST ANTERIOR PORTION. (After Toldt.)

ganglia of the middle brain and inter-brain. These fibers, which pass into the *cortex of the parietal lobe,* probably extend to the same region where the motor centers are also found. According to Ramón they there encircle the small pyramidal cells of the cortex. Doubtless these combinations form other cortical territories which are as yet unknown.

The *fibers* of the *optic nerve* follow another path. Like the optic radia-

tion, they run backward from the posterior capsule into the occipital lobes (Fig. 82). All of the *auditory fibers* appear to reach the temporal lobes and to distribute themselves through its cortex (Fig. 79).

The termination of the *olfactory radiation* still remains to be described but we will not discuss its peculiarities. Although the olfactory tract in man is comparatively atrophic, it nevertheless retains the complicated structure of the fiber of those animals which have been designated macrosmatic, and in which the sense of smell is distributed extensively through a number of lobes. The olfactory tract becomes complicated by centrifugal fibers which the central neurons of other tracts of special sense (the auditory and, probably, also the optic), and perhaps still other sensory cranial nerve tracts, appear to possess. I shall make no attempt to describe these tracts as Edinger and Bechterew have portrayed them in detail.

As to the centripetal fibers, we must assume a complicated direction of the higher neurons of the olfactory tract; this has been lately made the subject of studies which are not yet complete. It has been stated that a secondary olfactory tract is known to extend from the bulbus to the tractus olfactorius and the substantia perforata. From this point a *tertiary tract* arises. It passes in a double path to the cortex of the brain, particularly to that part which we designate the hippocampus convolution; first, through the lateral root of the olfactory tract, secondly, from the substantia perforata anterior through the *anterior commissure to the hippocampus convolution* of the opposite side. Now there are fibers in this lateral root which pass directly from the bulbus without touching the tract or its cells or other cells, chiefly as a *secondary tract, to the hippocampus major* of the same side.

The other relations of the cortex of the hippocampus are very complicated, even that of the coördination fibers and association fibers with other parts of the brain. Without minutely discussing the subject I wish to mention that the fibers coming from the corpora mammillaria to the hippocampus major, particularly those connected with the psalterium, are especially implicated, that some of these fibers communicate with the thalamus and with the tegmentum of the middle brain; briefly, that the center for smell has been traced beyond the cortical tracts to the most distant regions of the brain, especially in animals.

D. DISEASES OF THE SENSORY NEURONS. TABES DORSALIS (LOCOMOTOR ATAXIA)

So far as it is known we have seen how very complicated is the histologic structure of the sensory tract in comparison with the motor. Its functions also are more numerous and more complicated. There are several qualities of sensation. These must be conveyed through the same conduction tract, and on this point we are unable to decide whether, according to the nature of the stimulation, the same fiber is capable of developing various kinds of sensation, or whether in every sensory tract there are special fibers for different sensations. All sensory nerve tracts convey various sensations. This is not only true of sensory neurons, *sensu strictiori*, but also of the neurons for hearing, for sight, and for smell. In the auditory nerve we find the tract for equilibrium, in the optic there are tracts which permit the estimation

of distance, space, etc. There are fibers which act reflexly upon the nerves of accommodation and the pupil (oculomotor, sympathetic). In the tract of smell, in many animals at least, there are numerous but as yet unknown varieties of sensation.

Thus *diseases of the sensory tract* produce greater disturbances than those of the motor. Tactile sense, the sensation of pain, of temperature, and coördination of movements, for example, are conveyed by the first sensory neurons of the spinal cord (v. Leyden), thence they are transmitted further by neurons of the sensory tract, and are brought to consciousness. How manifold must those symptoms of disturbance be which affect only the first sensory neuron! The various sensations in disease are not even perceived in the same way; on the contrary, even with apparently similar histologic changes, we see that in one case this, in another a very different sensation, is experienced.

This sufficiently explains why we know so much less of neuron diseases of the sensory tract than of those of the motor tract.

We have fairly accurate knowledge in only one disease, an affection of the *first sensory neuron,* and this is *tabes dorsalis.* According to present opinions (v. Leyden, Pierre Marie, Redlich, Rosin) it is a diffuse disease of the fibers of the first sensory neuron; i. e., of *those tracts* above all which enter into the *posterior roots of the spinal cord,* also of the roots of the *sensory cranial nerves* which, it is true, are as a rule *less intensely* implicated. All the fibers of this neuron are not diseased in tabes but invariably more or less of them, and we must trace their degeneration until they split and form the nucleus of the second neuron. The view that tabes is a disease of the posterior roots was maintained by v. Leyden at a time when the structure of the spinal cord was by no means so well understood as it is to-day, and when the neuron theory had not been formulated.

Since tabes is, above all, a degeneration of the extramedullary and intramedullary posterior root fibers of the spinal cord, we find in the severe forms of the disease, i. e., according to the number of posterior roots and their fibers which are diseased, degeneration in Burdach's columns following the entrance and course of the posterior root fibers, and in Goll's column following the fibers which penetrate more deeply. We also find a decrease in the fibers of the *posterior horns,* in the region of the *reflex collaterals,* and in the fibers of Clarke's columns following the course of the posterior roots. *Optic atrophy* is often added, also *trigeminal disease* (affection of the jaws), gastric crises, laryngeal crises, and impairment of the sense of taste, etc., all of which are to be attributed to diseases of the root of the vagus and of the *glossopharyngeal* nerves.

Tabes is a primary degeneration of the nervous tissue without inflammatory implication of the intermediary tissue and the vessels. In place of the degenerated fibers glia tissue with its cells and vascular adventitia appear. This leads to so-called sclerosis. Proliferation of vessels, hemorrhages, round cells, granular cells, etc., are entirely absent. In cases of pure tabes we find upon transverse section of the spinal cord merely degeneration in the posterior columns and the disappearance of fibers in the posterior roots (Fig. 100). With the naked eye we recognize the characteristic *gray discoloration* (Figs. 101, 102, 103, 104) in the posterior column; in the stained preparations

nerve fibers are absent in these areas and a dense glia tissue, the nuclei of which are not greatly increased, takes their place (Fig. 105). This glia tissue seems to lose its structure only in very old cases, when it appears to be transformed into true connective tissue. Glia staining by Weigert's method shows in these areas that the character of the tissue is unchanged.

It is difficult to diagnosticate disease of the cells of the posterior root fibers, for here we can consider only spinal ganglia cells, in the brain the ganglion of the vagus root, the trigeminal root (Gasserian ganglion), and the optic nerve-cells in the retina, etc. No disease of the spinal ganglion cells has as yet been found in tabes. Neither atrophy, nor destruction,

FIG. 100.—POSTERIOR ROOTS (*a*) AND ANTERIOR ROOTS (*b*) IN TABES.

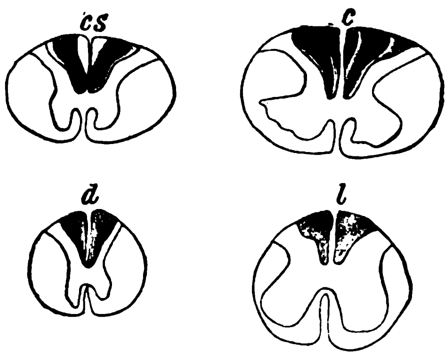

FIG. 101.—TABETIC DEGENERATION.
cs, Upper cervical cord; *c*, posterior cervical cord; *d*, dorsal cord; *l*, lumbar cord.

FIG. 102.—TABETIC DEGENERATION IN THE LUMBAR CORD.

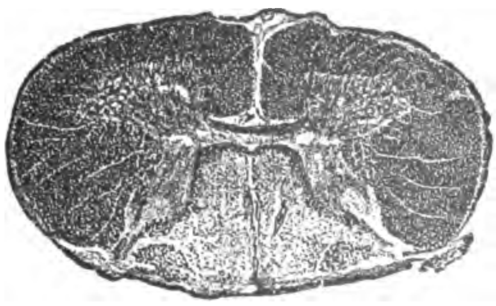

FIG. 103.—TABETIC DISEASE OF THE CERVICAL CORD. (After Oppenheim.)

FIG. 104.—TABETIC DISEASE OF THE LUMBAR CORD. (After Oppenheim.)

nor change in Nissl's granules has been positively determined; of course, the basic substance has not been minutely investigated. There are authors who believe that tabes does not originate in a neuron but at the point where the posterior root enters from the pia (Obersteiner and Redlich).

For the present we must withhold a positive opinion regarding these conditions; we have no methods with which to determine accurately whether a nerve-cell is histologically and functionally sound or not. The basic substance, too, which Apáthy and Bethe maintain may be divided into a fibrillary structure, has never been sufficiently examined pathologically on account of a lack of approved methods. According to the best authorities, the retention of Nissl's granules does not absolutely prove the normal composition of the cells, as its destruction proves disease.

Fig. 105.—Posterior Columns in Tabes, Greatly Magnified.

Tabes is the only *pure* neuron disease of the sensory conduction tract with which we are as yet familiar; and no disease of the sensory neurons of the higher divisions is known.

Histologically we are still in ignorance concerning the circumscribed and partial sensory diseases. Clinically, however, these are more or less familiar. The number of paralyses, especially of irritative conditions, of the sensory neurons is very great, and to these must be added the various forms of peripheral neuritis. But we have no accurate insight into the anatomical substratum. We believe the cells of the spinal ganglia to be the neuron cells of the peripheral sensory nerves, but we know nothing of their implication in the previously mentioned pathologic conditions. It is interesting to note that in cases of *herpes zoster* changes have recently been observed in the spinal ganglion cells, but, as stated, the entire subject has been too little investigated for us to discuss it in this brief article.

E. OTHER KNOWN TRACTS IN THE CENTRAL NERVOUS SYSTEM

(1) IN THE CEREBRUM AND PEDUNCLES OF THE BRAIN

The motor and sensory tracts are the ones with whose anatomical course and function we are most familiar, but they are not the only ones with which we are acquainted. Of some neuron systems we do not know the function, of many others the exact cellular origin, nor has their termination as yet been sufficiently traced. These are tracts which belong in part to projection systems, partly to coördination, and partly to association systems.

I shall begin with the most important of these which are situated in *the cerebrum*.

Here a number of fiber bundles unite the distinct and detached sections of the cerebrum, and then traverse extensive areas in the white substance

of the hemispheres. The numberless short tracts which run from one convolution to another or run within a convolution form a network of association fibers which cannot be disentangled.

First we must mention those most important association bundles which unite areas of the same hemisphere with each other (Fig. 106). The *fas-*

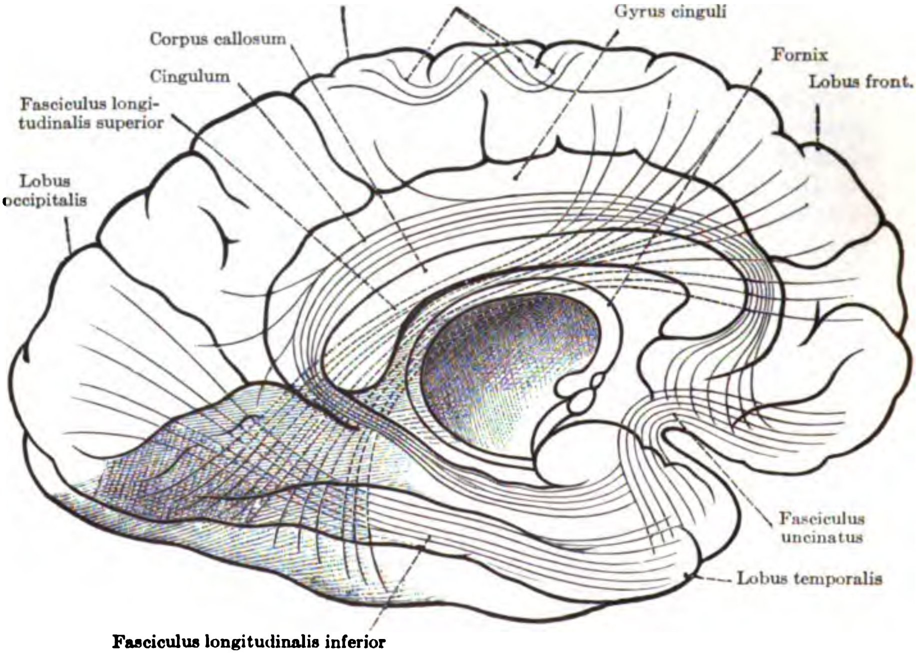

FIG. 106.—ASSOCIATION BUNDLE PROJECTED UPON THE MEDIAN SURFACE OF THE HEMISPHERE. (After Toldt.)

ciculus uncinatus runs from the lower external border of the frontal lobe to the anterior portion of the temporal lobe. The *fasciculus longitudinalis inferior* connects the temporal lobe with the occipital lobe. The *fasciculus arcuatus* extends from the posterior portion of the temporal lobe to the frontal lobe and also to the anterior portion of the parietal lobe. The *fasciculus fronto-occipitalis* runs from the frontal lobe to the occipital lobe. The *cingulum* runs above the corpus callosum in an arch from the cortex of the hippocampus major of the temporal lobe, and reaches that area of the frontal lobe where the fasciculus uncinatus is inserted.

Furthermore, we know there are important fiber masses which unite the areas on one side of the brain with those of the other side. They are called *coördination fibers*, and we assume that they serve the functions of both hemispheres for the purpose of coördination. The corpus callosum (Figs. 107, 108) is the structure which unites the greatest number of these fibers. With the exception of a few longitudinal bundles it consists almost entirely of coördination fibers which emerge from one hemisphere and radiate into the other, closely uniting in the median line, and forming a compact mass.

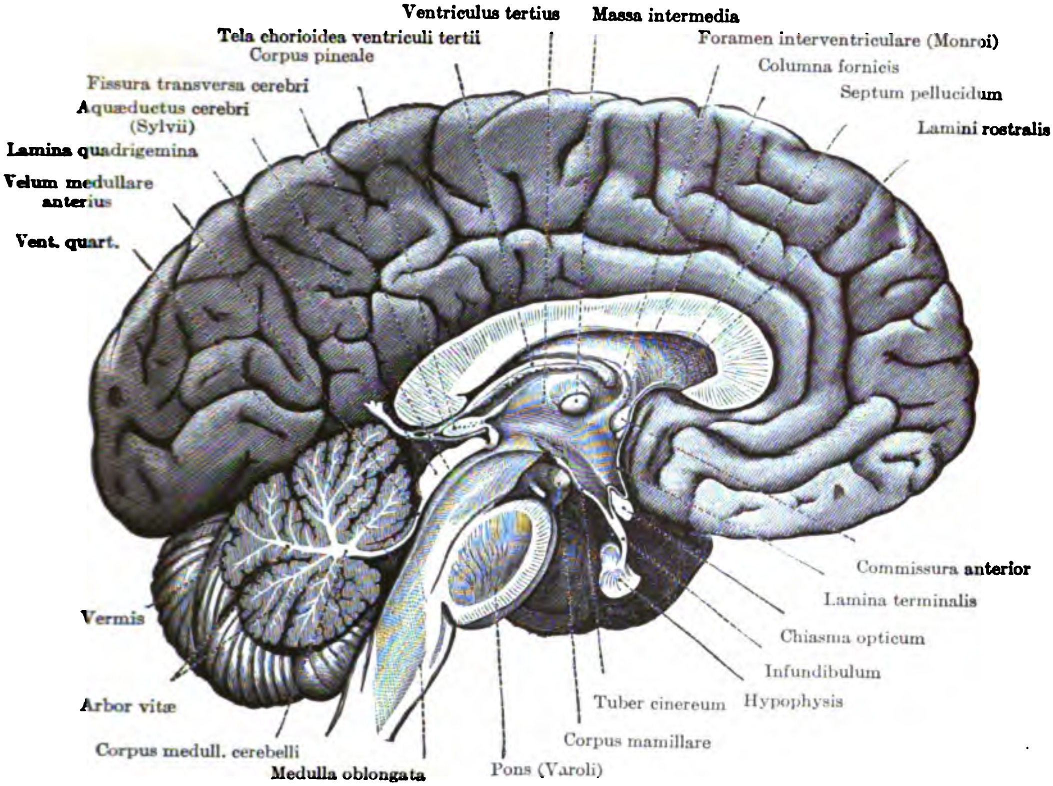

FIG. 107.—MEDIAN SAGITTAL SECTION THROUGH THE BRAIN. (After Toldt.)

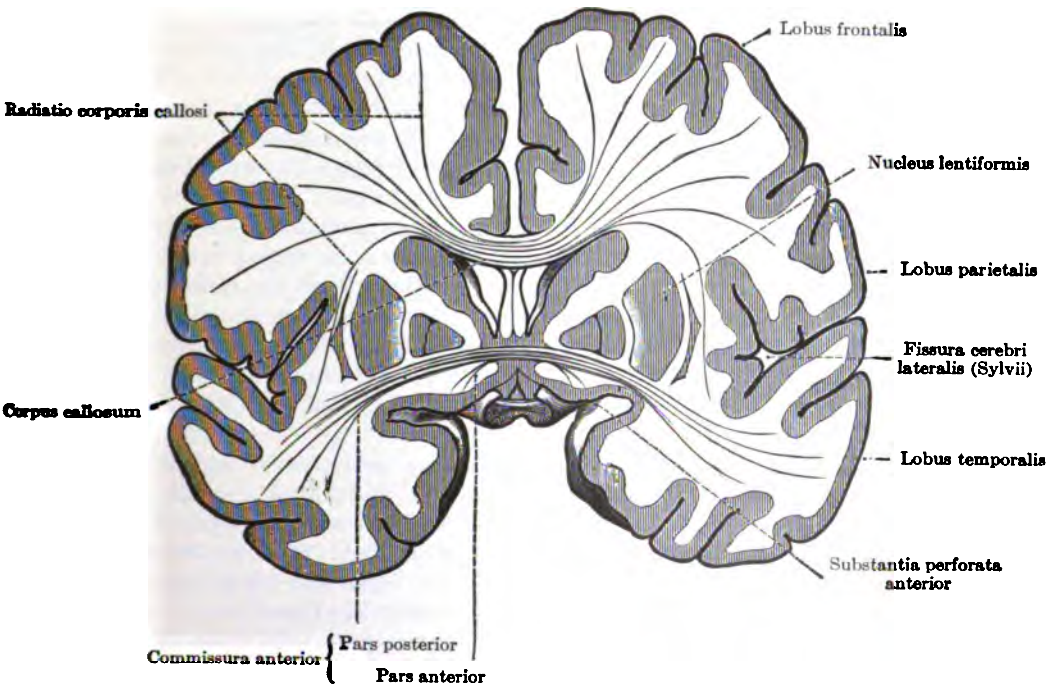

FIG. 108.—THE MOST IMPORTANT COMMISSURE FIBERS. (After Toldt.)

If we trace the radiation of the corpus callosum into the hemispheres we see it distributed to various regions of the cortex; the anterior portion passes over the anterior division of the lateral horn to the frontal lobes; fibers of the corpus callosum which pass to the occipital lobe encircle the posterior horn from above; these are called the forceps major; others which do not run so far backward as laterally pass to the outer side of the posterior horn in the temporal lobes, and are known as the forceps minor.

Some of the coördination fibers of the cerebrum do not pass through the corpus callosum but into the anterior commissure. We know from the macroscopic anatomy of the brain that this passes from one side to the other quite anteriorly in the ventral portion of the third ventricle, in front of the crus of the fornix and under the septum pellucidum. It lies at the same time under the head of the corpus callosum. In the hemispheres the fibers radiate below the corpus striatum, quite close to the floor of the brain, and above the substantia perforata anterior, also posteriorly and downward as far as the posterior parts of the temporal lobes. Here they join the cortex of the hippocampus major, and appear to form a coördination system for the sense of smell. A few fibers apparently enter the tractus olfactorius (olfactory lobe). There are a few other small commissure systems (commissura mollis and posterior, lyra Davidis) the importance of which I shall not discuss.

Besides the association and projection fibers of the cortex of the cerebrum there are numerous *projection fibers* in the corona radiata, and these run caudally to the trunk ganglions (*nucleus caudatus* and *corpus striatum*), to the *thalamus* (inter-brain), directly downward to the middle brain (*corpora quadrigemina*), or to the *medulla oblongata* and the *spinal cord.* The second motor neuron and the third sensory neuron are known to be important parts of these projection fibers, but the origin and course of many of the fibers from this tract are still unknown and their function still obscure.

I shall now leave the fiber systems of the cerebrum, and pass to the consideration of tracts in the caudal portions of the brain.

The trunk ganglia (lenticular nucleus) and the thalamus are connected by a closed tract, the lower part of which is surrounded by the internal capsule. This is the *ansa nuclei lentiformis.* It forms the *three links of the lenticular nucleus*; its function is still unknown to us.

I must also call attention to the *fasciculus longitudinalis posterior* (Figs. 94–98), a bundle of longitudinal fibers which may be traced caudally from the region of the corpora quadrigemina far down into the medulla. It is invariably situated in the sagittal plane of the *lemniscus tract,* extending dorsally beyond it as a closed round bundle. During its course it constantly gives off fibers to the cranial nerve nuclei, and appears to form for these an association center, made necessary by their complicated and numerous coördinate functions.

In conclusion I must mention a net-like layer (fibers and nerve-cells combined) which may be followed from the most caudal portion of the medulla through the pons to the tegmentum of the cerebral peduncles. This tract, formerly designated the substantia reticularis (Figs. 91, 93–96), has been characterized by Bechterew as the *central tegmental tract.* Its function is unknown. In the medulla and pons it usually lies laterally and above the lemniscus; it disappears in the region of the corpora quadrigemina.

(2) IN THE CEREBELLUM

We now come to the histology of the *cerebellum*, a neuron system of great extent and important in its unity. The course of its fibers has been only partially traced, and its connection with other portions of the brain has been more clearly revealed than its own complicated structure and the functions of its cells and columns.

We know that there are *centripetal and centrifugal cerebellar fibers*; we do not accurately know the individual course of these, but the bilateral functions of some of them have been determined.

Extending between the cerebellum and other portions of the brain we recognize the following principal fiber systems: (1) Columns from the spinal cord to the cerebellum (and *vice versa*); (2) From the nuclei of the medulla; (3) Pontal fibers of the cerebellum; (4) Tracts to the middle brain.

We will first consider the tracts which unite with the spinal cord (Figs. 109 and 110). The columna vesicalis of the spinal cord, the so-called *column of Clarke*, around which the fibers of the posterior roots split up (Figs. 67, 84), is already familiar to us; new fibers originate from these cells and pass into the *lateral columns* of the spinal cord, at the border of which they ascend without crossing (Fig. 85). Gradually forming a broader zone, they reach the medulla where they unite with the *corpora restiformia* and form the hemisphere of the cerebellum (Figs. 80, 81, 85), and within its medullary mass upon either side of the *corpus dentatum* they pass to the upper vermis, and split up in its cortex while crossing in the median line.

But we also distinguish a bundle descending from the cerebellum to the lateral columns of the spinal cord. These fibers originate in *Deiter's nucleus* (Fig. 109) of the cerebellum which is situated within the medulla of the hemispheres at the point where the corpora restiformia are inserted.

Finally there is an ascending spinal cord cerebellar bundle, *Gowers' bundle* (Figs. 37, *g*, 38, 109, 110). This lies as a triangle or prisin ventrally toward the cerebellar lateral column tract, joins the anterior column basic bundle, but lies also at the periphery of the spinal cord, passes through the medulla, upward through the dorsal and also the most lateral region of the pons, and in its most anterior division winds dorsally around the peduncles to the mid-brain, then passes through the velum anticum and into the cerebellum in the region of the superior vermis (Figs. 109, 110).

These are the best known spinal cord ramifications of the cerebellum, and now we come to those of the *medulla oblongata*. Almost all of these pass through the *corpus restiforme*. The most important layer is the *cerebellar olivary tract*. This probably arises from the hemispheres of the cerebellum, the fibers passing arc-like *through the posterior olives and the secondary olives of the same side* to the nuclei of *the olives and secondary olives of the opposite side* (Figs. 109, 110).

Some of the fibers which pass from the cerebellum through the corpus restiforme to the medulla reach the *sensory nuclei*, the glosso-pharyngeal-vagus, and the trigeminal. They originate in the middle portion of the cerebellum, probably in the nucleus tegmenti.

Another important fiber tract which originates in the cerebellum is that

of the *peduncles to the pons.* They form that *cerebellar fiber system in the pons* which passes from unknown regions in the hemispheres of the cerebellum to the cortex, first entering the ventral portions of the pons and encir-

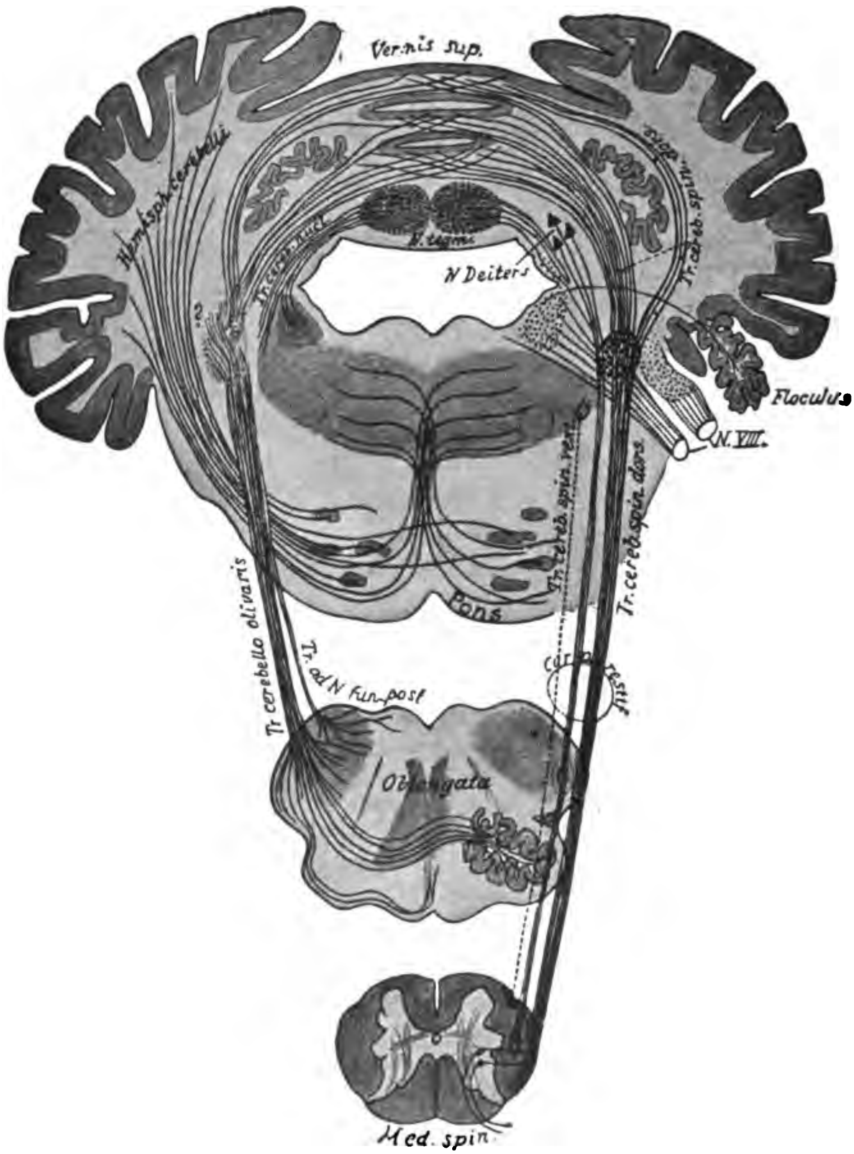

FIG. 109.—DIAGRAM SHOWING THE ARMS OF THE CEREBELLAR TRACTS. (After Edinger.)

cling the closed bundles of the pyramidal tract (Fig. 96). Many nerve-cells in which a portion of the fibers appears to end are here deposited, while, on the other hand, fibers of these cells ascend to the cerebellum, probably in the same tract. Some of these fibers, however, extend to the raphé of the pons,

which they partially cross, and then pass dorsally to the region of the *tegmentum* where they may be traced toward the cerebrum.

The communication of the cerebellum with both nuclei of the *auditory*

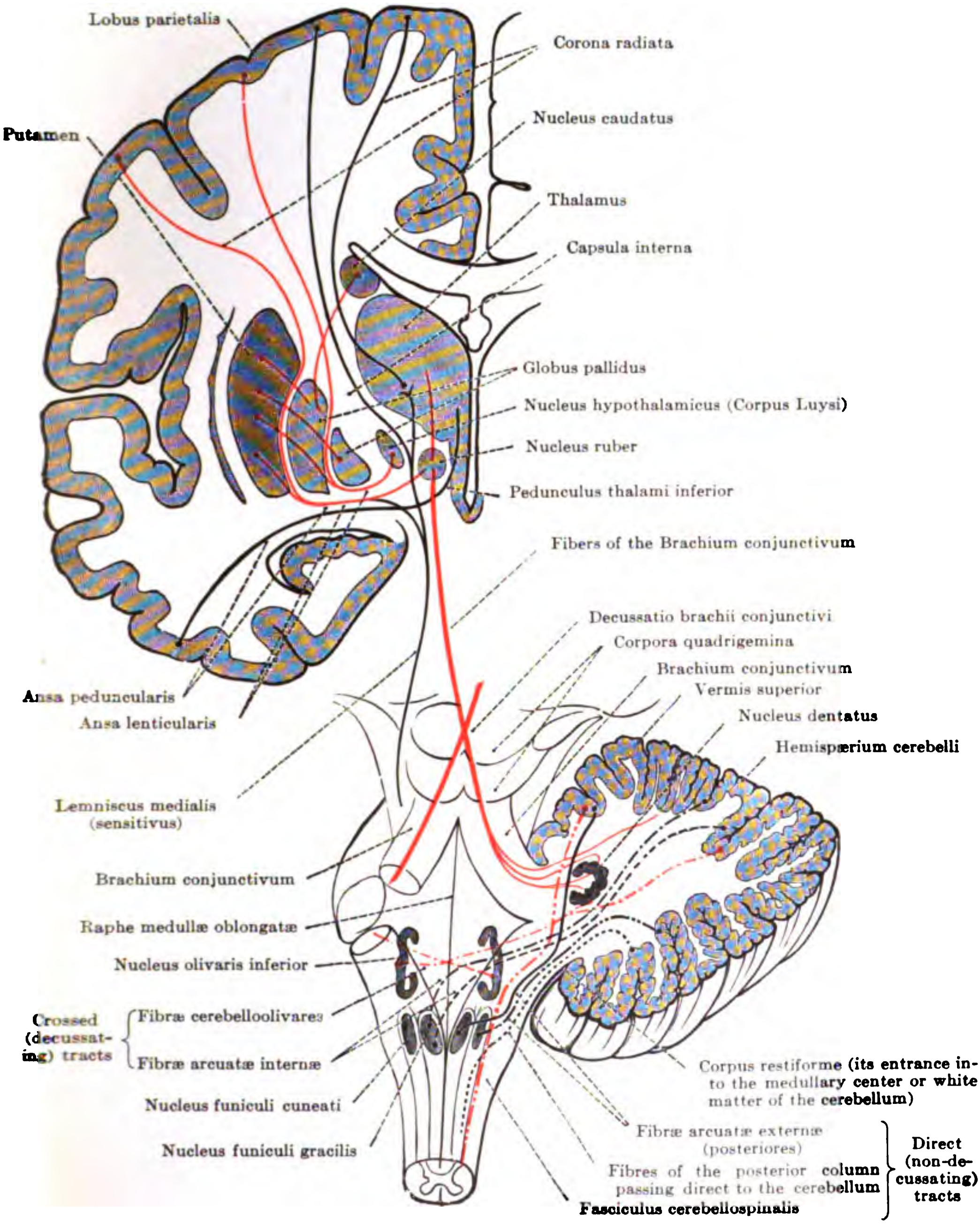

FIG. 110.—CEREBELLAR TRACTS. (After Toldt.)

nerve deserves special mention. The origin of these fibers in the cerebellum is unknown.

Another important communication is that of the cerebellum with the mid-brain by means of the *anterior cerebellar peduncles* (Figs. 97, 98) passing to the corpora quadrigemina. Its fibers arise from the *nucleus dentatus* of the cerebellar hemispheres, traverse the tegmental region of the mid-brain, cross from the lemniscus layer dorsally and ventrally to the posterior corpora quadrigemina (Fig. 98), and terminate in the *red nucleus* (of the tegmentum) in front of the anterior corpora quadrigemina (Fig. 99).

This forms a brief description of the many direct communications of the cerebellum with the middle brain and the spinal cord, and also with regions lying between these which contain very important nerve centers. The function of these many communications is uncertain. Some serve for coördination and assist in maintaining the equilibrium brought about by cerebellar combinations.

As the portions of the brain which must still be considered are united in various ways (collaterals, posterior longitudinal fasciculus) the *union* with the cerebellum must be regarded as a *second,* perhaps more important, one.

There appear to be no *direct tracts from the cerebellum to the cerebrum,* but the peduncles *indirectly* communicate with the pons and the corpora quadrigemina, just as tracts of the cerebrum enter into their terminations in the tegmentum.

Our knowledge of the course of the neurons within the cerebellum is very meager; of many fibers we know neither the origin nor the path they pursue. But we may hope that the future will reveal this.

I take this opportunity to mention a few peculiarities in the *structure of the cerebellar cortex* which differs markedly from the cortex of the cerebrum (Fig. 111). Here three layers may be differentiated; the external *molecular layer* which is deficient in cells and the *internal granular layer* are separated by a narrow band in which a *large number* of peculiarly formed cells are arranged like a palisade. The cells in the middle layer are called Purkinje's or bottle cells. They are characterized by an extraordinarily large number of dendrites which are distributed chiefly in the molecular layer while the nerve process passes to the medullary mass of the cerebellum, in its passage often giving off numerous collaterals. In the molecular layer we find the terminal ends of the neurons whose origin is unknown, the fibers of which rise from the medullary layer and through the granular layer to the cortex. The scant cells of the granular layers themselves, at least in great part, are connected with the dendrites of Purkinje's cells; their nerve fibers and collaterals come in contact within the body of these cells. The granular layer, which was formerly not regarded as a nervous organ, is composed of numerous polygonal cells with a few short dendrites and a nerve process which extend upward into the molecular layer and there divide into two branches and become entwined. Besides these small cells of the granular layer

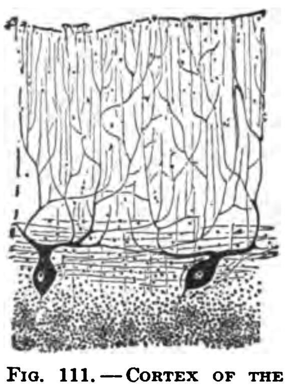

FIG. 111.—CORTEX OF THE CEREBELLUM. (After Obersteiner.)

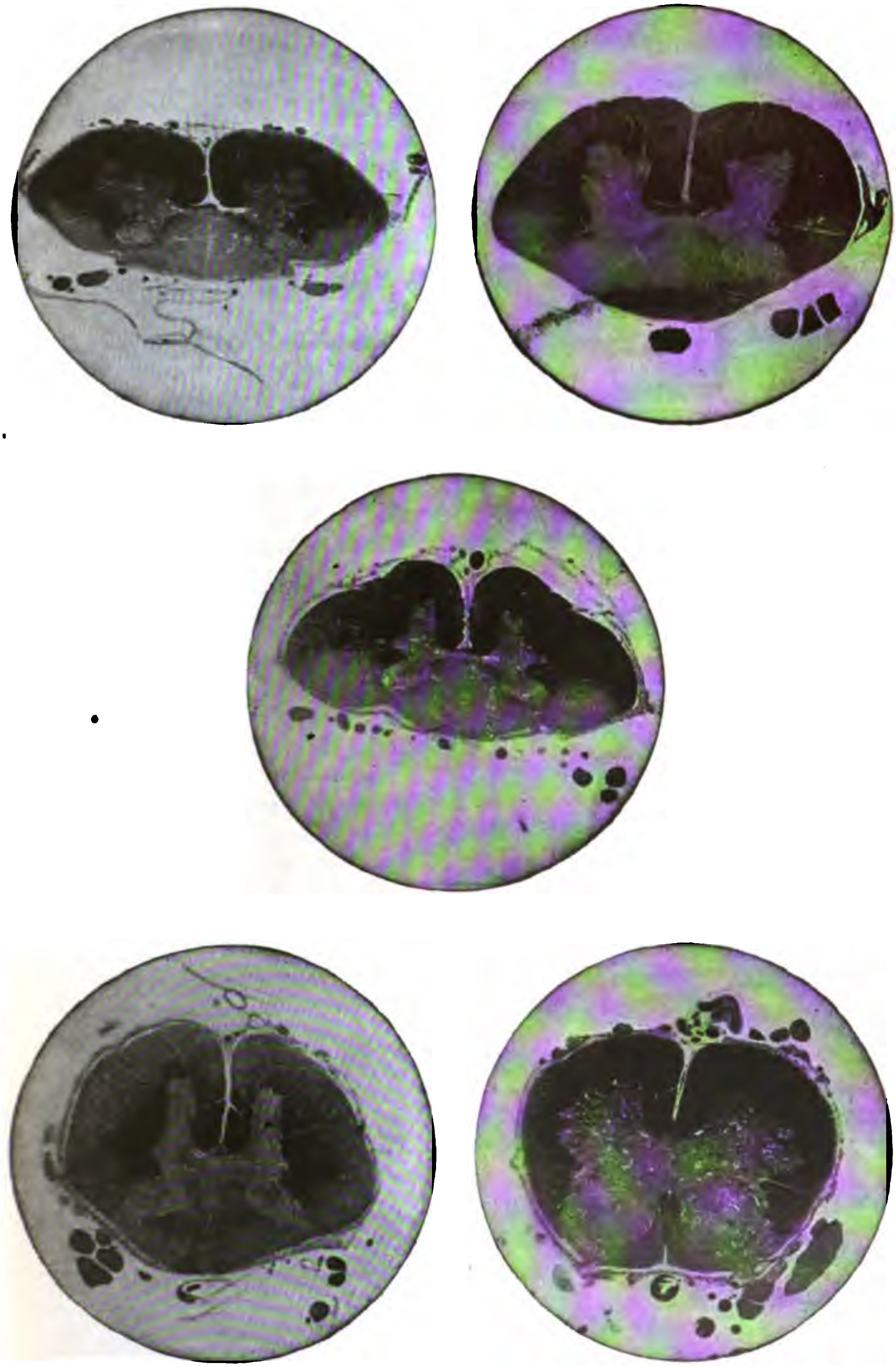

FIG. 112.—FRIEDREICH'S DISEASE. (After P. Marie.)

there are a few large ones whose dendrites pass upward into the molecular layer, but the nerve processes within the granular layer after running a short distance split up around many cells. Neuron fibers from the medullary mass of the cerebellum, whose origin is not yet known, end in the granular layer. We are chiefly indebted for our knowledge of the finer structure of the cortex of the brain to the investigations of Ramón and van Gehuchten, and this knowledge has been acquired by the Golgi method.

F. NEURON DISEASES OF THE CEREBELLUM. FRIEDREICH'S ATAXIA

We know as yet of no diseases in which the tracts which lead to the cerebellum or originate from it are alone affected. Such pure neuron diseases of the cerebellum cannot arise independently, because the cerebellar tracts are most intimately connected both anatomically and functionally with other spinal and cerebral fiber systems, and in pathologic cases all must be affected.

There is, however, a disease which implicates a definite cerebellar tract; namely, that situated in the *periphery of the lateral column tracts* of the spinal cord. This disease is *Friedreich's ataxia*; it is hereditary, and manifests itself by marked ataxic disturbances, yet, even when the patella reflexes are absent, sensation is not decidedly impaired.

Anatomically, this disease produces degeneration in three different column systems. We find destruction of fibers in the *posterior columns* (but not in the roots or in their course), in the *pyramidal tracts*, and in the *cerebellar lateral column tracts* (Figs. 112, 113). It is possible that this degeneration in the posterior columns takes place in the area of the association tracts, the course of which we know to be shorter. As has been stated, the posterior roots and their zone of entrance are normal, probably also their entire course. The disease in the course of the pyramidal tracts apparently does not affect the motor fibers since muscular spasms and increased reflexes constantly appear here. It is still a mooted question which must be settled by further researches which fiber tract is destroyed; I must call attention to Monakow's tract which unites with the pyramidal tracts and descends in the lateral columns. There is certainly degeneration of the *cerebellar lateral column tracts* and the *cells of Clarke's columns* which form their origin. Since various authors (Senator and others) believe in a primary implication (congenital atrophy) of the cerebellum, we may assume disease of the tracts of the cerebellum. We may hope that, in the future, thorough histologic investigations of this rare disease may give us more accurate information as to the origin and relation of the cells connected with the degenerated fibers.

FIG. 113.—FRIEDREICH'S ATAXIA.

G. COMBINED SYSTEM DISEASES

There is till another group of diseases of the spinal cord in which the anatomical finding closely resembles that of Friedreich's ataxia. These cases have been designated *combined system diseases.* The name may be deemed fitting only when those areas in the columns of the spinal cord in which definite tracts run are affected. In the *posterior columns,* Goll's columns in toto, Burdach's columns alone were found to be partially degenerated, in the *lateral columns* the region of the pyramidal tracts, but usually, as in Friedreich's ataxia, the cerebellar lateral tracts, not rarely the region of Gowers' columns, and in the *anterior columns* Türk's bundles in the immediate vicinity are often implicated (Fig. 105). But these degenerations are not strictly limited to the previously mentioned tracts, and in each of the individual cases now frequently observed the seat of the disease varies, at one time this, at another time that, column being implicated. The clinical symptoms vary exceedingly; they resemble those of other spinal cord diseases, and may be as vague as the anatomical localization. Hence, the anatomical starting-point of the disease remains obscure, and we are in doubt whether a diffuse leukomyelitis chronica, perhaps starting from the vessels, or a primary diffuse disease of the gray substance is the cause. We cannot to-day positively assert that the affection is a neuron disease, as is maintained by some authorities. We may point by analogy to similar degenerations of the white substance, not in columns but, on the contrary, occurring in foci, particularly in severe anemias and in certain intoxications. Here the assumption of processes which start from the vessels and only secondarily lead to degeneration of the nervous substances is much more reasonable.

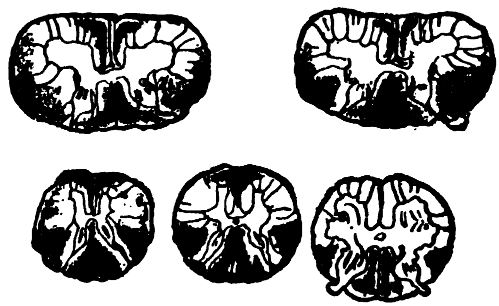

FIG. 114.—COMBINED SYSTEM DISEASES. (After Oppenheim.)

V. OTHER DISEASES OF THE CENTRAL NERVOUS SYSTEM

I must now consider the pathological histology of those affections of the brain and spinal cord which do not depend upon the primary disease of *one nerve tract* or of one or more neurons, which, therefore, are not limited in their extent but are found in any area regardless of the nature and function of the nerve elements there situated. The cause of such diseases is *acute or chronic inflammation* which may occur diffusely or in foci within the central nervous system, or which may attack the membranes, and secondarily implicate the nervous substance; at times we are dealing with *degenerative processes* which may originate in the vessels, or from the interstitial tissue and may develop in various areas. Finally, *tumors* or *injuries* may simultaneously implicate the various nervous elements.

All the tracts which are damaged by diffuse or circumscribed (focal)

disease of the nervous system must show *degeneration* beyond the focus since that portion of the tract immediately degenerates which is cut off by the pathologic focus from the neuron cells belonging to it. Therefore, degeneration will follow the direction of the affected tract either peripherally from a *descending* focus or centrally from an *ascending* one.

It is evident that secondary degenerations always occur in primary foci of the central nervous system, and these are often extensive if long tracts are interrupted. At the same time the study of such secondary degenerations after primary focal diseases is invaluable, not only for the full comprehension of paralyses present but also for the recognition of the structure of the central nervous system. In a certain sense they correspond to experimental sections to which we owe much of our knowledge of the course of fibers.

In the following I shall attempt to describe the most important *inflammatory* focal diseases while I must refrain from considering tumors and injuries, since these would lead us too far afield. *Secondary degeneration* following inflammatory foci, tumors and injuries, in so far as its course is known, will be discussed.

A. ACUTE MYELITIS, ENCEPHALITIS, AND ACUTE SOFTENING

Acute myelitis presents manifold clinical pictures. Acute encephalitis, which has been less accurately studied, occasionally produces most unlike symptom-complexes according to the seat of the affection and whether it is circumscribed or diffuse; for an accurate portrayal of the different pathologic types text-books upon nervous diseases should be consulted.

The *histologic picture* to be described is uniform. In most of these processes two characteristic changes are invariably found in acute myelitic foci. First, the invariable alteration in the *walls of the vessels* which is conspicuous in the histologic picture; the dilatation and the abnormal filling of the vessels which not infrequently causes the smallest branches to rupture, the exudation of cells and lymph from the same, and the proliferation of "round cells" from the vascular wall and the glia tissue. Occasionally the *degenerative changes of the nervous elements* in the region of the focus are of great histologic interest. The acute inflammatory changes in the nervous apparatus do not differ essentially from those of other organs.

Certain individual differences are evident. These are mainly the consequence of the soft *consistence* of the tissue, and this we refer to the presence of myelin, a substance readily dissolved and absorbed. Added to this are deviations from the conditions perceived in other organs which are often evident at some distance, and produce secondary alterations in the nervous tissue.

The consistence of the tissue frequently leads to *softening.* Every extensive myelitic and encephalitic focus in the white substance may be reduced to a pappy consistence so that in making microscopic preparations it is difficult to keep the destroyed elements in position even by careful embedding in hardening substances. When the processes begin acutely we find in the white substance in the area of greatest inflammation a *disintegrated field* in which only the tissue elements of greatest resistance, the vascular walls, and the glia tissue (fibers and cells) are visible, although they may be displaced. In the main, however, we find here myelin granules, swollen and broken up

axis cylinders in irregular arrangement which cannot always be recognized as such. *Hemorrhages* readily occur in these softened tissues, the greatly distended vessels finding no external resistance rupture, and the effusion of blood is rapidly distributed throughout the necrotic, softened, and destroyed tissue.

The *cell proliferation* in the surroundings of the vessels is of a somewhat different character in the white substance of the nervous system from that in other organs. We usually find the body of such cells enormously swollen and completely filled with remains of tissue, which, because of their softer consistence, are more readily absorbent than the necrotic elements of other organs. Therefore, these cells are filled with a granular substance which becomes black on the application of osmic acid and this is to be regarded as altered myelin. In contrast with normal myelin this blackens with Marchi's fluid (see below), stains deeply with sudan red, scarlet or other dye-stuffs acting on fat, which is not the case with normal myelin. Such cells are called *granular cells.* According to the softness of the tissue they are often found quite remote from the vascular wall in which they appear to originate. They push forward in rows, palisade-like, to the border of the diseased area.

Stress must be laid upon the fact that these granular cells are all mononuclear—not rarely the nucleus is still present—and it is noteworthy that cells are present in the inflammatory diseases of the central nervous system which *cannot be regarded as wandering cells* of the blood but as *proliferated cells of the tissue,* especially of the adventitia of the vessels, and these, like the multinuclear phagocytes of the blood, have loaded themselves with granules of myelin and other shreds of tissue. They are apparently identical with the "round cells" in the inflammatory foci of other tissues, but are more dense, and are visibly more distended with tissue masses than others.

The farther from the center of the focus that we search for normal tissue, the more numerous do we find the retained nerve fibers to be in the white substance. At all events, we note in these areas numerous nervous elements in a transitional stage between the normal and the pathologic. In an unstained condition, or with ordinary methods of staining, these transitions are not readily perceptible, but in the Marchi method, which makes the granular cells very distinct, we possess a fine means of determining the *recent destruction of the medullary sheaths.*

Marchi's Method.—Here a brief statement of the importance of Marchi's method may be of interest. During the process of its destruction the chemical composition of the myelin apparently changes, for in Marchi's fluid, which consists of three parts of Müller's fluid and one part of a one per cent. hyperosmic acid, normal myelin sheaths are unstained; at most they change to a pale gray color. In beginning acute degeneration the nerve sheaths behave differently. Here they are stained black, i. e., the myelin is found in deep black granules around the colorless axis cylinder, and the globules are destroyed; this can be best seen in longitudinal sections. The medullary substance which apparently shows chemical alteration now stains like the substance of the granular cells with which it is perhaps identical, also like the dense granules which appear in the lipochrome of the nerve-cells, to which I previously referred.

But if the *gray substance* has been attacked by acute myelitis, this struc-

ture also suffers and shows marked pathologic changes. The most important, although not always the most obvious, is the *degeneration of the nerve-cells,* but, because of the firmer consistence of the tissue, no conspicuous softening is present. In intense myelitis the nerve-cells are either entirely destroyed or to such an extent that, robbed of their processes, they are detected as clumps which can no longer be differentiated from other cellular products of inflammation in their midst. Occasionally, however, their configuration is apparently normal and at first conveys the impression of a certain degree of atrophy, unless *staining* according to Nissl shows them to have been completely destroyed in the interior. The body of the cell is filled by a fine granular dust instead of the usual coarse granular substance, and this no longer stains intensely, the nucleus passes away from the center to the side of the cell, and only the lipochrome maintains its appearance and position as the most resistant substance. That the *fibrous net of finer nerves in the gray substance* is destroyed by the inflammation is shown by preparation *stained* according to Weigert's method, or with triacid (Rosin), or with van Gison's method, for we recognize a *massive formation of new cells* particularly in the surroundings of the vessels. These cells, however, are not so large as the granular cells in the white substance, for there are no masses of detritus in the myelin. Nevertheless their origin is the same as that of the adventitia of the vessels. *Dilated vessels* were everywhere observed and *hemorrhages* were numerous.

In the recent foci of acute inflammation the *glia tissue* is only passively implicated. Where the inflammation is most severe much of it is softened although even there some fibers show greater resistance than the nervous tissue; in the mass of tissue detritus we readily find retained glia nuclei and fibers. The glia is usually quite well retained in the peripheral areas of acute inflammation even when there are hemorrhages, granular cells, and dilated vessels. The meshed spaces also are apparently in good condition when the nerve tubules of the white substance have disappeared, that is, have degenerated. Often this network, instead of being filled by nervous elements, is made up of *granular globules* or well retained red blood-corpuscles from hemorrhages.

Thus we have the *histologic picture* presented by acute inflammation of the white and gray substance of the central nervous system. Microscopically only the picture of acute inflammation is recognizable; the clinical variations which appear during life are not seen; and the manifold and varying etiologic factors which permit the differentiation of different forms of acute myelitis form no part of the histologic picture.

From the microscopic appearance it is difficult to differentiate between acute inflammation and *acute softening* such as is produced by nutritive disturbances on the part of altered vessels, by occlusion or rupture of the same, or by toxic agents.

Clinically the differentiation is generally easy. The anatomical picture is invariably the same, and in inflammation also we have the histologic appearance of softening. Both dilatation of the smallest vessels and hemorrhages appear in the focus of softening, apparently because the broken up tissue exerts no power of resistance upon the vessels which are usually friable, and thus the *vis a tergo,* the internal pressure, immoderately enlarges the caliber

and finally causes the wall to rupture. *Granular cells* also are in the surroundings of the focus. But the quantity of mononuclear inflammatory cells in the vessels is more scant, there is no hyperemia of the large arterial vessels, and thus a slight difference is perceptible. Those who are especially interested in the subject are referred to my article upon acute myelitis in which this condition is described somewhat more minutely (*Zeitschrift f. klin. Med.*, 1896).

B. ACUTE POLIOMYELITIS

We now come to a special form of acute myelitis which is *chiefly seated in the anterior horns of the spinal cord* or in the motor nuclei of the medulla oblongata. Its limitation to the seat of origin of the first motor neuron raises the question whether we are not here dealing with a pure neuron disease which secondarily implicates the vessel, and this is the opinion of some authors.

However, the histologic picture makes it clear that no pure disease of the nervous apparatus, as in the previously mentioned disease of the first motor neuron, is present (Goldscheider and others). On the contrary, the nervous tissue is so slightly implicated that it appears questionable whether in this affection the nerve-cells and the processes are not merely attacked secondarily and passively. To this must be added that *not only are the motor cells of the diseased areas chiefly affected, but even those in the area of inflammation* which have other functions than motor are diseased. Therefore, in spite of its peculiar localization this form of most acute inflammation is included among the acute forms of myelitis.

Fig. 115.—Lumbar Cord After an Attack of Acute Poliomyelitis of the Left Side.

The process is disseminated throughout the entire length of the spinal cord, in some areas simultaneously and acutely, but the intensity of the disease varies, and it leads to irreparable changes only in individual areas. Clinically there is paralysis of one or more extremities. Histologically, in recent cases, there is an *acute inflammatory dilatation of the vessels of the gray substance of the anterior horns*, the branches of the anterior spinal artery, and simultaneously a *marked proliferation of round cells.* The process rarely extends into the white substance *beyond the border of the anterior horns.* Here granular cells are not infrequently observed and hemorrhages often occur. In the condition previously described the *nerve-cells* of the affected region are found to be in all stages of degeneration. The motor nerve fibers of the diseased anterior horn cells then secondarily degenerate, and the nerve

fibers disappear peripherally from the anterior roots to the paralyzed muscle. In atrophic paralysis the muscles also degenerate.

If the process is extremely intense, when it has run its course the nerve elements are permanently destroyed and the affected portion of the anterior horn becomes sclerotic. It decreases in size (Fig. 115), is filled with tense glia tissue, and is almost entirely devoid of nerve elements.

It is quite generally known that the disease occurs in earliest childhood, but it is also met with in adults although much more rarely (Moritz Mayer, Duchenne, Erb, F. Schultze, F. Müller, Strümpell). In these cases the anatomical picture is almost identical with that of infantile paralysis.

C. ACUTE BULBAR PARALYSIS

Inflammatory disease in the region of the motor nuclei of the cranial nerve, which corresponds to poliomyelitis of the spinal cord, and bears the name of acute *inflammatory bulbar paralysis*, unlike disease of the spinal cord, is rare and is seen only in adults. In this bulbar poliomyelitis, as in the medullary form, we find the *same affection of the vessels*, the *same cell proliferation*, and the *same destruction of nerve-cells*. Perhaps the alteration of the motor neuron (which alone may be clinically prominent) is more conspicuous because the sensory and other tracts as well as the motor nuclei are situated in its immediate vicinity, and this is regarded by some as a proof that we are here dealing with a disease of the first motor neuron of the brain. However, the above objection may also be urged against this view as well as the fact that only the nuclei in the region of the fourth ventricle are diseased, while those of the mid-brain, the origin of the oculomotor and trochlear, are not implicated.

Clinically, therefore, this rare affection may be readily confounded with affections which run a similar course but are histologically quite different. I shall mention here only acute peripheral neuritis of the cranial nerves, bulbar apoplexy (apoplectic bulbar paralysis), thrombosis of the basilar artery, asthenic bulbar paralysis (Strümpell) (pseudo-bulbar paralysis), and Landry's paralysis, without discussing their histological peculiarities which do not come within the scope of this article.

D. CHRONIC MYELITIS AND ENCEPHALITIS

The affection designated as *chronic myelitis and encephalitis* varies considerably from the preceding picture of acute myelitis and encephalitis. As is well known, this disease often arises *from acute processes*, and much more rarely develops *spontaneously*. It is also well known that chronic processes terminate in so-called sclerosis. So long, however, as this stage is not reached, the histologic picture varies much more than in the acute form. This is due to the numerous and gradual transitional changes from early to later stages of development, and as long as sclerosis is not yet complete this may always be recognized. I have already described acute recent changes, and to this I will add only those which occur when the process is older, or which exist from the onset provided the disease has a chronic beginning.

The more protracted the disease, the more complete the disappearance

of the destroyed nervous substance, the granular cells, and the hemorrhages, and the *more distributed and thickened* the *glia tissue* and the *walls of the vessels.* At first *cells* of the adventitia (mononuclear structures deficient in protoplasm) as well as glia cells (usually containing only a narrow protoplasm body) (triacid stain) are *greatly increased in numbers* and these, at the same time, fill the spaces caused by the absence of nerve tissue. Granular cells are quite scant. Finally sclerosis occurs, not infrequently in a focus in the other parts of which quite recent changes are visible.

Sclerosis of the gray substance may be recognized by the destruction of nerve-cells and those fine fibers which pass through the normal gray substance in so dense an arrangement (with Weigert's stain). In another area there is a thick network of glia with increased cells and thickened vascular walls, the adventitia of which is conspicuously broad and very rich in nuclei. The capillary wall is also thicker than the normal. In the *white substance* sclerosis is readily recognized. Upon transverse section no round cells characteristic of the nerve fibers are to be seen, their places being filled by glia tissue developed from proliferation of the tissues of the normal glia meshes. In this stage a marked proliferation of the glia cells with especially numerous processes is particularly distinct, and we also note those cells which have been designated neuroglia cells (spider cells). In addition there are great numbers of peculiar structures formed somewhat differently and about the size of a leukocyte which are called *corpora amylacea.* They resemble starch granules, are concentrically layered, but do not give the reaction for starch, and have not yet been fully analyzed. Furthermore, a dense *film work of fibers* forms in the white substance, this being at first rich in nuclei which it subsequently loses and then resembles connective tissue, its structure being recognizable only by the use of Weigert's glia stain. Where septa of the pia resembling connective tissue penetrate into the sclerotic focus, they also take part in the proliferation and are distributed still deeper. Occasionally the nuclear proliferation of glia tissue changed in this manner remains for a long time (particularly in the gray substance) in old *well developed sclerotic foci,* and finally reappears. The triacid stain enables us distinctly to recognize these conditions. The vascular wall of the white substance is also markedly involved in the sclerosis; it becomes thickened, as was previously stated, especially in the region of the adventitia.

E. CHRONIC POLIOMYELITIS

As acute poliomyelitis represents a particular form of acute myelitis, so chronic myelitis is typical of another special form, chronic poliomyelitis.

There have been but few thorough investigations of this very rare disease (Oppenheim, Nonne and others), and its curability accounts for the very exceptional occurrence of autopsies. The paralyzed muscles again resume their functions although a few remain weak; we are not fully cognizant of the histologic conditions. But the histologic findings obtained up to the present time very closely resemble those of the acute cases, in which the implication of the medulla oblongata is prominent.

Chronic poliomyelitis is, therefore, not limited to the spinal cord, and the name poliomyelo-encephalitis would be much more applicable.

F. MULTIPLE SCLEROSIS

Multiple sclerosis is a peculiar form of chronic myelitis with typical histologic changes. It usually implicates the entire spinal cord as far as the medulla oblongata; isolated foci are not infrequently found also in the middle and inter-brain. From the onset the disease is chronic and focal. The situation of the foci is entirely independent of the course of the fibers and the structure of the nerve substance, sometimes being only in the gray or in the white substance; occasionally they occur in both, passing from the white to the gray substance. They are distinctly separated from the normal surroundings, show no sign of acute destruction, of hemorrhage, or of granular cells, and are in that stage of sclerosis which still shows numerous cells: *Glia proliferation, vascular thickening, increase of nuclei.* The spinal cord in the medulla is permeated by such foci. With Weigert's glia stain they are perhaps even more prominent than with the medullary sheath stain; for we then often recognize numerous, microscopically small foci which would otherwise not be visible. Much larger foci are seen in the shape of round or angular circumscribed spots irregularly disseminated like defects in the nervous substance, and these appear as if they had been pierced with a stiletto. The chronic myelitic process of this disease is very conspicuous and *does not especially attack the axis cylinder.* This is retained to a great extent, and precludes those secondary changes which we invariably see when the nerve fibers are completely destroyed (Figs. 116, 117).

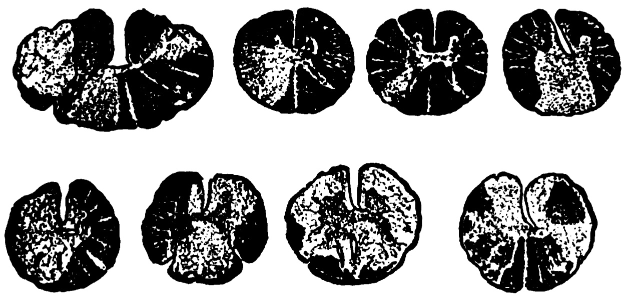

FIG. 116.—SCLEROTIC FOCI IN THE SPINAL CORD, WEIGERT'S STAIN. (After Oppenheim.)

FIG. 117.—MULTIPLE SCLEROSIS.

Those authors are probably correct who designate the disease as *gliosis.* Its origin from the nervous substance may be excluded, but the implication of the vessels is secondary, the seat of the foci by no means corresponding with their course and distribution.

G. SECONDARY DEGENERATIONS

In pathologic processes of any organ certain distant effects are produced, so that the surroundings of parts not directly affected may show certain inflammatory or degenerative alterations. In the central nervous system,

however, *other damage* may be explained by the intimate anatomical relation of the neuron constituents. What Waller once designated as roots of the spinal cord, the anterior of which, corresponding to their course, are centrifugal, and the posterior centripetal, is true of every tract of the brain and spinal cord. Section and interruption of the conduction tract and the consequent separation from the nourishing cell invariably destroy the peripheral portion of the neuron. This degeneration is intense and irreparable provided communication is not restored. Moreover, central disturbances occur, but these, as I have several times stated, are less intense, they can be estimated only by the finer methods, and become distinct only on continued absence of function of the neuron.

The knowledge of the path of secondary degenerations, i. e., the destruction of the peripheral ends of the nervous conduction tracts continuing from

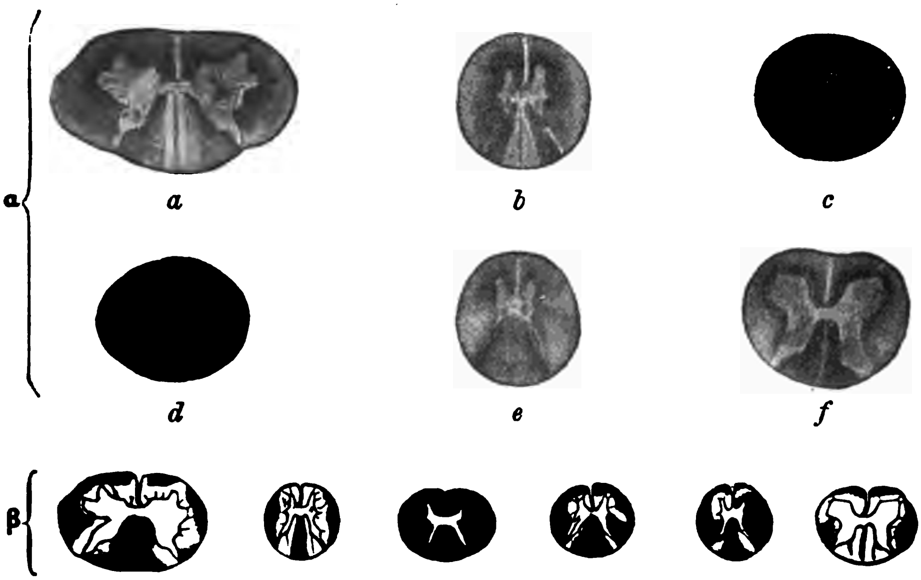

Fig. 118.—Transvers Myelitis with Ascending and Descending Degeneration.
α, Staining according to Weigert; β, triacid stain. The dark areas indicate degeneration.

the point of interruption, is important in many respects; first, because in determining the localization and extent of a disease of the central nervous organs it is important that we should know what portions of the neuron are implicated, and what *deleterious effects* the *primary focus* may have upon *remote regions* of the neuron system. Very often circumscribed disease of the nervous system, particularly in regions where nerve centers or tracts are closely adjacent (medulla oblongata), causes extensive and serious degeneration of entire tracts by damaging many of the neurons situated within the region of the focus.

A knowledge of the course of secondary degeneration is important, too, because not infrequently it enables us to determine *the seat of the secondary focus* which would otherwise be very difficult.

Finally, these degenerations furnish a fruitful field for research. This

unexplored region in the course of the tracts has been persistently studied, and if this investigation is continued with finer methods many other neuron systems, previously unknown, will doubtless be discovered.

Every foci developed in the spinal cord, provided it is sufficiently extensive, leads to ascending and descending degeneration. This is most distinct and intense in foci which affect the entire transverse section.

Transverse myelitis (Fig. 118) furnishes the clearest types of these degenerations, and its course will therefore best illustrate other less extensive foci and slighter degenerations.

(1) SECONDARY DESCENDING DEGENERATION OF THE SPINAL CORD

It is evident that descending degeneration follows in the main the course of the motor tracts in the spinal cord, since, so far as we know, all the motor tracts in the structure of the spinal cord run in a descending direction, i. e., caudally.

True, we also know of a few sensory centrifugal tracts; it will be remembered that all of the fibers which enter the spinal cord (spinal ganglia) from the primary sensory centers divide into two branches, a descending branch which, as a rule, is short, and an ascending one which is often much longer. Therefore, in myelitic foci those descending

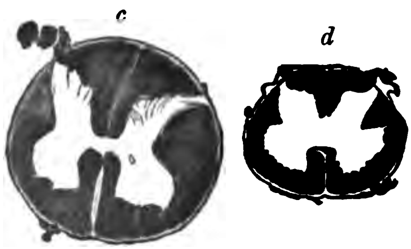

FIG. 119.—DESCENDING DEGENERATION OF A PYRAMIDAL TRACT OF THE SPINAL CORD.

branches whose course is through the region of the focus and thereby disconnected from the center must degenerate. But these fibers are found only in the posterior columns, and even there they form so small a part that they often cause no visible degeneration but sometimes only a complete disappearance of fibers; this we occasionally find described as *Schultze's comma-like degeneration* of the posterior columns, because the degenerated bundles usually (not always!) lie together in the posterior column in the shape of a comma on transsection.

FIG. 120.—BILATERAL DESCENDING DEGENERATION OF THE PYRAMIDAL TRACT.

The principal mass of the descending degenerative fibers is situated in the *lateral* and *anterior columns*. In the lateral column this region is desig-

nated as the *pyramidal tract* (Fig. 118, *e* and *f*, Figs. 119–121). It is usually situated in the posterior internal portion of the lateral column, but does not extend to its border zone nor to the gray substance.

We have also seen that there are two other descending tracts in the lateral

a

b

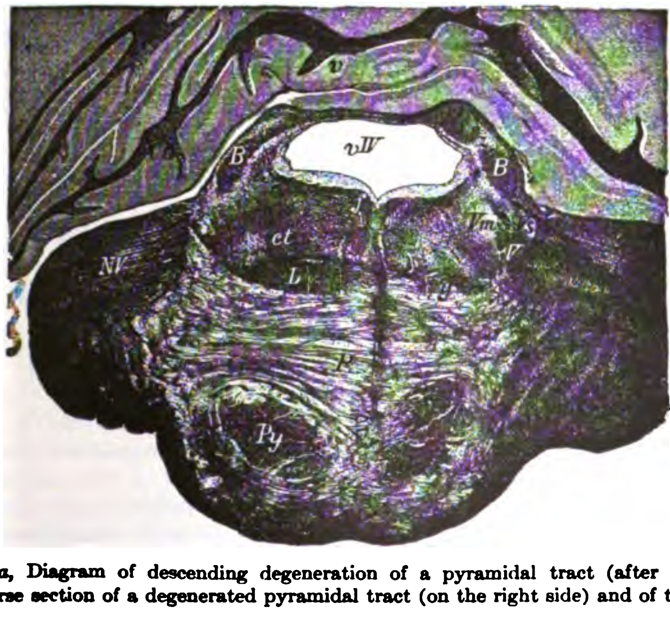

Fig. 121.—*a*, Diagram of descending degeneration of a pyramidal tract (after Edinger); *b*, transverse section of a degenerated pyramidal tract (on the right side) and of the pons.

columns which are subject to transverse lesions. One of these is *Monakow's bundle* which descends from the middle brain in the lateral columns, partly between the pyramidal fibers but somewhat anterior and external to them, and the second is *Löwenthal's bundle* which is situated quite ventrally in the lateral columns and at their margin.

In the anterior columns at either side of the anterior longitudinal space is the remainder of the *uncrossed pyramidal tracts, called Türk's bundle,* which also occasionally shows descending degeneration.

Degenerations of fibers is simultaneously observed, usually in the *anterior* (white) commissure; these implicate continuations of the fibers of Türk's bundle which have not yet crossed.

In this region descending degeneration attacks all fibers which are separated from their cells below the transverse disease, and it extends to their own and their collateral terminal arborization.

Besides the tracts in the neighborhood of the foci which have a *long course* many *short tracts* are interrupted. In recent affections these degenerations are best recognized by the Marchi method. This shows immediately below the foci (as well as immediately above it), the degeneration of enormous numbers of nerve fibers (Fig. 118, *c, d*) in those areas of the white substance which are immediately adjacent to the gray substance, particularly in the lateral columns, and in the anterior columns; it is somewhat less marked in the posterior columns. In the more external areas of the spinal cord, particularly in the region of the anterior, columnar, basic bundle, we observe an intense black staining in transverse sections immediately below the foci. These limited secondary degenerations must be referred to short tracts which run in segments from section to section within the spinal cord and which are designated quite correctly as association systems of the spinal cord.

In conclusion it must be stated that in the inflammatory focus even the motor anterior root fibers arising from the anterior horn cells which have been destroyed invariably degenerate and produce simultaneous paralysis and atrophy of the roots belonging thereto.

(2) ASCENDING DEGENERATION IN THE SPINAL CORD

The centripetal tracts degenerate above the focus. Many of them belong to the *posterior root regions,* and we know that some of the branches of these do not enter the gray substance but mount upward in the posterior

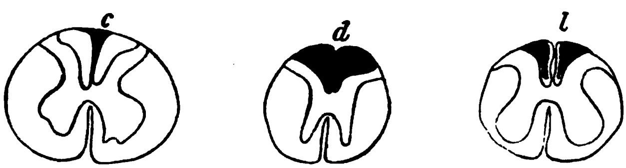

Fig. 122.—Secondary Ascending Degeneration after Disease of the Conus Medullaris.

column. These tracts pass to the medulla in Burdach's and Goll's columns, the former invariably carrying the newly added posterior root fibers, and the latter the fibers which entered lower down. Immediately above the focus in both posterior columns marked degeneration is found; the higher Burdach's

columns ascend the more free they become; finally, if the focus is low enough, Goll's columns only will be implicated (Fig. 118, *a*, *b*, Fig. 122, *c*, *d*, *l*). We have no knowledge of long tracts in the posterior columns other than those of the posterior root fibers.

The *cerebellar lateral column tracts* and *Gowers' bundle* (Fig. 122, *A*, Fig. 123) also degenerate in an upward direction. These columns have already been described. They are situated at the border of the lateral columns, the former behind the latter; but Gowers' bundle penetrates somewhat more deeply

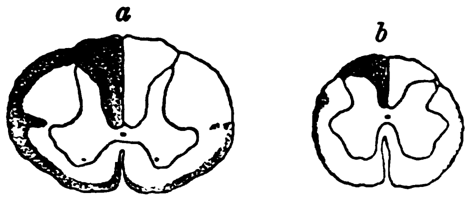

FIG. 122 *A*.—ASCENDING DEGENERATION (RIGHT-SIDED MYELITIC FOCUS).

FIG. 123.—SECONDARY ASCENDING DEGENERATION.

into the interior of the lateral columns. The fibers of the cerebellar lateral column tract originate from the cells of Clarke's columns. *Those fibers of the lateral columns must perish which originate from sensory cells situated on the opposite side in the gray substance of the posterior horns,* e. g., the fibers of the second sensory neuron which in the medulla forms the median portion of the lemniscus and does not cross. These fibers, however, are united into small bundles in the lateral columns, therefore in a transverse interruption are not very prominent. But above the lemniscus layer of the medulla, in the pons and higher, they may be demonstrated as degenerated fibers.

Immediately above the transverse interruption as well as beneath it many *short tracts* are found which have degenerated. These also must be regarded as association tracts.

(3) SECONDARY DEGENERATION IN THE BRAIN

The law of secondary degeneration in the brain is exceedingly difficult to understand. Much study has been expended upon it, and some remarkable facts have come to light which have been utilized in histology and structure. But the conditions are still too obscure and too complicated to be accurately described. The pyramidal tract has been more thoroughly investigated than others. Here I shall merely present a diagrammatic figure (Fig. 121).

H. SYPHILIS OF THE NERVOUS SYSTEM

Many diseases of the central nervous system are attributed to syphilis. In some of these affections, for example, in progressive paralysis, a causal connection can only be determined by statistics, while changes of a syphilitic nature are never revealed in the histologic picture. It is well known that tabes shows histologically the changes already indicated; no signs pointing to syphilitic change have ever been determined.

There are, however, many nervous diseases in which the syphilitic cause

can be demonstrated. The deleterious influence of tertiary syphilis upon the central nervous organs has frequently been revealed anatomically, and in the last decades this has been repeatedly explained and described.


FIG. 124.—SYPHILITIC ENDARTERITIS AND GUMMATOUS INFILTRATION OF THE SURROUNDING AREA.

In such cases a syphilitic endarteritis most often develops (Figs. 124, 125) as well as gummatous disease of the membranes of the brain. For instance, we find in the spinal cord a gummatous arachnoiditis, in the brain a gummatous infiltration of the membranes, especially at the base, which thence distributes itself to the nerve sheaths and to the choroidal plexus. These conditions usually develop slowly and chronically, and produce secondary disturbances in the course of the nervous substance itself, particularly in those areas surrounded or covered by the infiltrated membranes. It is therefore clear that gummatous infiltration of the membranes of the spinal cord, provided it reaches a certain extent, may produce secondary degeneration by compression. Often we find that degeneration (Fig. 126) of the lateral columns has also attacked the pyramidal tracts or the posterior column, in consequence of which muscular contractions, disturbances in sensation, and coördination develop (syphilitic spinal paralysis, after Erb). Microscopically we see scleroses in the lateral and posterior columns due to compression of the roots passing in and out, and these show atrophy of their fibers while the connective tissue of the membranes of the brain is enormously thickened and is permeated by small-celled infiltration.

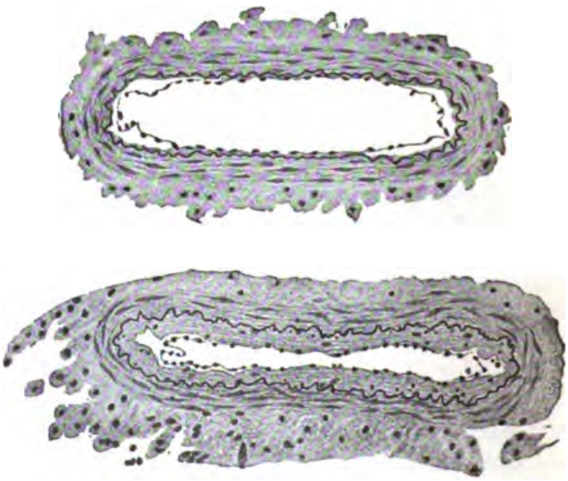
FIG. 125.—THE NORMAL AND THE SYPHILITICALLY DISEASED ANTERIOR SPINAL ARTERY.

This concludes our description of the normal histology of the central nervous system and its microscopic changes. In what has been detailed, it has been impossible to give more than a partial picture, and it was presupposed that the macroscopic and gross anatomical relations of the central nervous system were already familiar

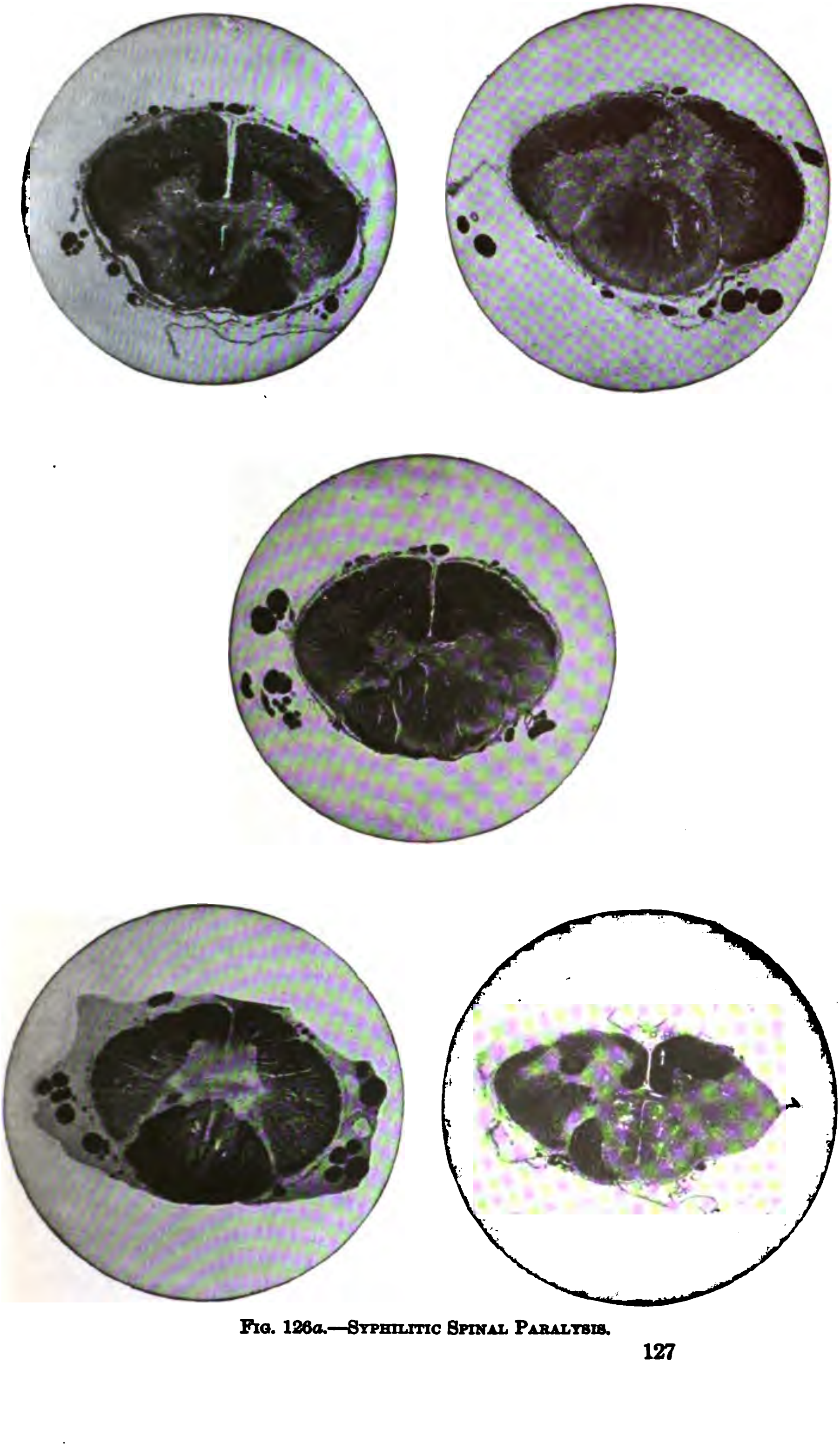

Fig. 126a.—Syphilitic Spinal Paralysis.

to the reader. It is, however, at once apparent that the incompleteness of my description is in great part due to want of microscopic knowledge of the structure of the organs of the central nervous system.

Notwithstanding this, in reviewing the ground gone over, we find much valuable data and abundant stimulation for future researches. The new century advances, and must promote our knowledge of neurology to the same extent as did the preceding century. We hope that scientific research may prove as successful in the future as it has been in the past, and that it may be of four-

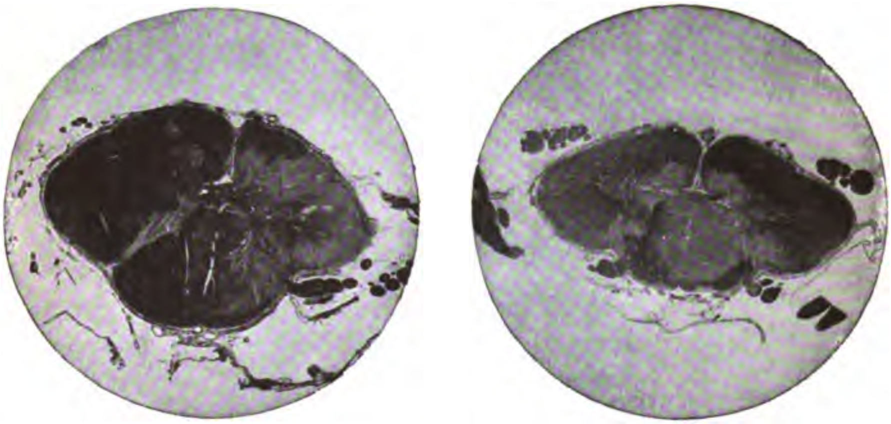

Fig. 126b.—Syphilitic Spinal Paralysis.

fold nature, as follows: (1) Careful microscopic investigation of the diseased parts according to the latest reliable methods; (2) the various methods of embryologic investigation; (3) the comparative anatomical method, especially utilized by Edinger; (4) experimental investigation, which is constantly furnishing such excellent results (section and division). Perhaps in the course of the next hundred years neurologic investigations based upon these methods will unveil in its entirety the complete structure of the central nervous system, of the brain, the organ of all organs.

GENERAL NEUROLOGICAL DIAGNOSIS

INCLUDING THE EXAMINATION OF PATIENTS WITH DISEASES OF THE NERVOUS SYSTEM

By P. SCHUSTER, Berlin

INTRODUCTION

The office patients who consult the specialist for diseases of the nervous system all show a certain peculiarity which distinguishes them from other patients, namely, the ease with which they may be mentally influenced. This fact is generally known, even by the laity, of patients suffering from functional diseases of the nervous system, such as hysteria, neurasthenia, hypochondriasis, etc.—and much more fully by the physician. Somewhat less keenly appreciated by physicians is the fact that this same influence of suggestion is exerted upon patients suffering from organic nervous diseases. It is true, as a rule, that we have less power to control a patient with organic disease of the nervous system than the neurotic, for in the latter the possibility of suggestion is entirely endogenous, and arises from the internal nature of the disease; in the other patients the possibility of suggestion—provided the organic pathologic picture is not obscured by neuroses—is of a more exogenous character, and is generated and strengthened above all else by the consciousness of the patient that he suffers from disease of the nervous system, by the layman's very peculiar conceptions of a nervous disease, and by the consequent action and manner of those about him.

What has just been pointed out as peculiar to all nervous patients, the psychical factor common to them all, perhaps accounts for the fact that the layman more closely associates mental and nervous diseases than does the physician, and that even he commonly combines these diseases.

The increased possibility of psychically influencing the nervous patient, just outlined, is not only of the greatest significance in the general pathologic investigation, but especially so in treatment. For in the psychical peculiarity of our patient there is a mighty therapeutic force, the wise application of which may relieve many of the patient's complaints and difficulties, and the improper use or neglect of which may just as greatly injure him.

This being the case, the physician must endeavor, even at the first examination of the patient, to gain an influence over him, and at the same time to become psychically *en rapport* with him. The physician must remember that with many patients with nervous disease even *the examination represents a part of the treatment*, and that a single incautious question or a careless remark may endanger or frustrate all later therapeutic endeavors.

The careful and patient taking of the history, and a thorough examination of the entire body which should consume as much time as is necessary, often surprisingly smooths the way to treatment. Here we are assisted by the fact that most nervous patients desire very frequent and careful examinations, and rarely give the physician any trouble on account of inconvenience or by their prudery.

If the foregoing is borne in mind we may readily understand that the following description will be merely a diagram or framework representing only the technic and the mode of employing instruments in an examination. And although a hint will occasionally be given as to the motive for undertaking special investigations in the patient, or as to the caution with which certain questions are to be asked, nevertheless nearly all must be left to personal and professional tact, to the human delicacy as well as the personal diplomacy of the physician, so that the barren structure of the technic of examination shall become a living process.

In this description we shall refer only to what is practically most important, and, in order to keep within the scope of this article, shall not drift into details.

HISTORY (ANAMNESIS)

(The extent of the history, the manner of eliciting it, family predisposition, psychoses and nervous affections appearing in the family, previous diseases of the patient, childhood, education, occupation, emotional tendencies, service in the army, development of the last affection, its prior course, trauma, alcohol, syphilis, sexual relations.)

The history is of predominant importance in neurologic examination because in many cases the diagnosis cannot be made from the results of examination but only on the basis of the history. Therefore a certain knowledge of diseases of the nervous system is indispensable if the history is to be at all reliable. The value and utility of the history by no means depends upon its length; on the contrary, useless and secondary details detract from its value. One of the chief dangers in taking a history is that we are liable to wander from the main road into byways and thus we fail to take a comprehensive survey or at least waste much time.

Another source of error consists in believing too implicitly the reports of the patient or his friends, thus forming false conceptions in regard to causal and periodic conditions.

It would, however, be a mistake and most unwise to stop the patient at once in his uncritical report, and insist upon another mode of description. This engenders in the patient a feeling that he is suppressed, that we are not allowing him sufficient time, and that without his report he can form an opinion as to his condition.

Under such circumstances, I advise the following method: The patient should be asked to state his chief symptoms and his reasons for seeking aid, and while doing this we should note his personal peculiarities. After the patient has related what, in his judgment, is most important, the physician should take the reins in hand, for he has already obtained a guide to the special questions which he wishes to ask.

We *first* endeavor to ascertain what diseases, particularly ***nervous*** or ***mental***

affections, have prevailed among the *ascendants, descendants,* or the collateral members of his family. Of non-nervous affections, we must make particular inquiries concerning *gout,* because (aside from other reasons) the success of suitable treatment often favors the fact that the gouty diathesis plays a certain rôle in many functional nervous diseases, especially in migraine. In these patients a history of *tuberculosis* in the family is not very important; much more so is the question of *syphilis* in the parents. But only in exceptional cases, especially when the patients are children accompanied by their parents, will we obtain to this question a definite answer, either affirmative or negative.

According to recent investigations (Peiper) the *blood relationship* of parents appears to be of less significance in the history of nervous diseases than was formerly assumed.

Inquiries concerning alcoholism in the parents had best be reserved until we ask the same questions concerning the patient.

We must enter somewhat more minutely into the question of the possible occurrence of *mental* or *nervous diseases* in the family of the patient, of *peculiarities, idiosyncrasies,* etc. Here we generally learn but little, for either the patients are ignorant and have not noticed these things in their relatives or they conceal their knowledge from the physician—especially of psychoses—from the fear that he may regard them also as mentally unsound. Perhaps, too, they may in this respect unconsciously deceive themselves.

For instance, the pathologic importance of a *case of suicide* in the family is rarely recognized, or if recognized the occurrence is almost invariably concealed. Severe and persistent hysteria in a member of the family is always referred to as "a chronic weakness," epilepsy of near relatives is stubbornly denied, and psychopathic conditions, especially depression, are represented as the normal expression of "much sorrow" or "much care and trouble" and the like. In all examinations of this kind we must therefore attempt to secure accurate reports, because, as I have already stated, questions in regard to the health of the family, no matter how urgently pressed, are answered in the negative; hence, as a rule, it will not require much time to complete the first part of the history.

Subsequently, in the personal history of the patient, we keep the reins in our own hands, and I advise the following method: Request the patient to answer precisely the following questions: (1) *Is the disease for which you consult me the first that you have had, or have you ever before been seriously ill?* If the second part of the question is answered in the affirmative we go on: (2) *Of what nature were the diseases from which you suffered? Have you ever suffered from a disease resembling the present one?*

The answers to these two questions, which should always be asked first, form two fundamental props in the structure of our history. The intermediate framework can be readily filled in subsequently as we must now consider how to put the special questions we desire to ask, and how much time we have left in which to complete the history.

We should now seek for missing links in a certain chronological order, i. e., ask first whether the birth of the patient was normal, whether he had *infantile diseases* and particularly so-called teething spasms, whether the patient suffered from enuresis for any considerable time, whether grinding of the teeth in sleep and terrifying dreams had been observed during childhood,

when puberty began, and what occurred during the *years of puberty.* In women it will frequently be found that the *first menstrual period* was accompanied by transitory nervous symptoms, which not rarely form a miniature picture of the psychosis or neuropsychosis which developed in later years.

The period of time following early youth when a definite *occupation* is chosen, or at least is prepared for, will indicate the *mental development* of the patient, beginning with his school years. But in the history the development of the intellectual side of the mind is even more important than that of the emotional side and of the psychical sphere; for these the *school years* afford a very fine reagent. Neurasthenics and hypochondriacs often tell us that, although they were always well in school, they "always kept to themselves" and did not make friends; epileptics state that when in school they were often "very confused" (*petit mal*) or that they were remarkably stubborn and obtuse. The relatives of hysterical girls sometimes tell us that these girls in their school years often had fantastic ideas, that they were deceitful (pseudologia phantastica), and that even during these early years erotic and sexual manifestations, etc., became prominent. All of these factors must be considered in intricate cases.

Next we take up the *occupation* and its possible *deleterious effects.* Workers in poisons or metals (lead, brass), workmen who are continuously exposed to great heat, and those who perform extraordinarily severe bodily labor must be carefully examined. We must take note of those occupations which are combined with great responsibility, as well as those in which a very strict discipline is enforced (policemen), also that of teachers (music teachers), and all occupations relating to the Bourse (brokers), etc. In all of these, the injurious consequences are of psychical nature. Even the layman knows that these deleterious effects, especially if continuous or *accumulative,* are exceedingly important etiologically and in the history. Of all psychical emotions *care* and *sorrow* are most frequently noted.

Supplementary knowledge of the period of adolescence in the male is sometimes afforded by his service in *the military.* Frequent punishments during this time, misbehavior in an active or passive sense, lead us to consider the existence of certain forms of imbecility as well as other psychical affections.

These questions and their answers give us some idea of the patient's health up to the time when the disease appeared for which he consults the physician. Now the patient can no longer be restrained from a minute report of his present condition, which he will give, as a rule, in a haphazard way, and overload it with minor details. Even in this case he should be allowed to make his report, and should be interrupted only to suppress etiologic considerations, etc., which he is apt to include.

The patient should then be asked the following questions:

(*a*) Whether there have been *prodromes*; (*b*) whether the disease appeared *gradually* or *suddenly*; (*c*) whether the *condition* has remained the same from the onset of the affection, or not. If, either from lack of intelligence and education, or because of mental depression, excitement, or fear, or for any other reason, the patient does not comprehend these questions, the physician himself had better take the matter in hand, and put a few concise questions to the patient which can be answered briefly and to the point.

I recommend this method—unpleasant as it often is for some patients—for many reasons. Almost always it quickly gives us an idea of the development and the previous course of the disease.

Before proceeding to specialize the symptoms of the patient, which we have recognized from his report, and to inquire into his present condition (*status præsens*), three exceedingly important points must be investigated in the history of every patient with nervous disease: *Trauma, alcohol, and the sexual relations, including a possible syphilitic infection.*

Of these three points the patient will usually—at least when this is positive—mention *trauma* voluntarily; for trauma is often of exaggerated importance in the opinion of the layman. I do not mean to state that it is unimportant for the physician to know that such a thing has occurred; on the contrary, we know that many neuroses, particularly hysteria, neurasthenia, and hypochondriasis, may follow injuries or even mere psychical trauma. In organic nervous affections trauma is of less significance, although according to recent experience this condition appears to have been under-estimated. Hence we should never fail to inquire into trauma, even though other etiologic factors may be reported by the patient. On the contrary, when, besides trauma, another cause, such as chronic metallic poisoning, hereditary predisposition, alcoholism, or the like is present, trauma pathogenetically plays an especially important rôle.

A second point always to be investigated is *alcoholism.* If the patient who consults us is a travelling salesman for a wine house, a bartender, a waiter, a driver, or follows any other occupation in which, according to experience, the habit of drinking is almost unavoidable, or if the patient's face reveals the well-known signs of chronic alcoholism, the question of his daily consumption of alcohol will soon be settled.

We often obtain a history from our patients of the better class without effort. It is sometimes very difficult to induce the working classes, especially women, to acknowledge the abuse of alcohol, but here the difficulty may be bridged over, especially for the workman, if the physician remarks that, with such arduous labor he probably has to refresh himself often by a draught, or he is often forced by association with his fellows to drink in the course of his occupation. If we show that we realize the conditions, we can often arrive at the truth.

Women often regard any question concerning the use of alcohol as an insult. Therefore this subject must be led up to by tactful questions, such as asking whether the patient uses much tea or coffee, and then, perhaps as a supplement, whether wines or spirits are used in addition, and finally the question concerning alcohol may be very delicately put. The symptoms of many women patients whom we suspect of alcoholism—pain, attacks of weakness, fainting and the like—pave the way for us to ask whether they resort to some form of alcohol to relieve these conditions, and as soon as alcohol has been mentioned we may make more minute inquiries.

After settling these questions, we must consider specific infection and the sexual relations. In men this usually causes little embarrassment, but we should not shrink from putting our questions in detail, i. e., we should not simply ask whether a patient has had syphilis, but whether he has had a chancre, eruptions upon the skin, etc. If we merely inquire about "syphilis," many

patients who have suffered from simple gonorrhea will answer the question in the affirmative. With married women we may inquire concerning abortions, and on questioning the possible syphilis of the husband, we may obtain a clue to specific infection. Sensible women do not object to the direct question, whether or not they have been infected by their husbands. It is naturally a very delicate matter to make inquiries of a young girl concerning syphilitic infection, and unless we have powerful reasons for suspecting this infection, it is wiser not to allude to it. Of course, if unmarried women directly or indirectly admit sexual intercourse, we do not hesitate to inquire into this subject.

Just as in the case of syphilis, we must individualize in questioning patients as to important *sexual relations*. In men we may inquire openly as to masturbation; in girls and women whom we suspect of onanism, it is well delicately to veil this question. For example, we may inquire whether there is not sometimes an itching of the body, particularly of the genitalia, which becomes so distressing that it leads to scratching and rubbing. Patients are often willing to acknowledge the habit when contracted under a legitimate motive. Sometimes, however, we meet with very sensible and intelligent patients—and a case of this kind shows how much more easily a physician can manage *educated* patients than others—who spontaneously report, either negatively or affirmatively, in regard to masturbation. It is singular, although readily understood, that male masturbators very often are more likely to conceal this evil habit from the physician than females.

Masturbation in many cases is of less etiologic than symptomatologic importance; for onanism is frequently not a cause—as the layman is inclined to believe—but a symptom of disease. For this reason it is of little significance if—as often occurs—we elicit no history of masturbation. In those cases in which it is of practical or *therapeutic* importance for the physician to know whether the patient is addicted to masturbation or not, he generally obtains this knowledge during the course of the treatment from the patient's spontaneous answers to his questions. When the patient fails to divulge this habit, although it is of scientific interest yet it is usually of little practical importance, particularly in the treatment. For, in this case, the patient does not appear to occupy himself—his negative condition shows this—in a pathologico-hypochondriacal manner with the sexual sphere. Were this otherwise, conventional considerations and the restrictions consequent upon admitting onanism would soon be overcome by his psychical depression.

Before leaving this subject I must add that, in discussing these conditions with the patient, we must be careful not to moralize or use expressions which indicate a wish to exercise censorship. Therefore, such expressions as "evil," "bad habit," "pollution," etc., are not to be employed, but we should apparently consider the habit as a light matter, treating the condition *en bagatelle*. This applies also to other sexual conditions, sexual abstinence, sexual abuse, etc., which must be discussed with the patient. To me there appears to be no doubt that sexual abstinence in patients sexually competent, particularly if not voluntary but enforced, may have a deleterious effect (perhaps chiefly psychical). On the other hand, too frequent cohabitation and the so-called *congressus interruptus* must be considered to have a somatic, injurious effect. All of the sexual conditions which here come under consideration are probably

functional diseases, usually of neurasthenic or hypochondriacal nature. I have with reason discussed the details of these sexual conditions which the history discloses, for they are of decisive importance, and demand our deepest scientific interest in the complete understanding of numerous pathologic conditions.

My chief reason for devoting so much time to the sexual relations is the perhaps greatly exaggerated importance which many nervous patients themselves attach to these conditions, and on consulting their physicians they are likely to detail these. Many nervous patients do not regard themselves as correctly appreciated by the physician unless satisfied that he has investigated and correctly estimated everything connected with their disease, and has thoroughly discussed with them their sexual relations and sexual cares.

THE PRESENT CONDITION (STATUS PRÆSENS)

GENERAL CONSIDERATIONS

(Complaints, general impressions, various types, stigmata of degeneration, conspicuous family resemblance of features in neuropathic patients, facial expression, psychical condition.)

In determining the *status præsens* we again permit the patient to detail his *symptoms and complaints*, but here also we must individualize. If he complains of pain we ask whether this is persistent or appears in paroxysms, whether it is lightning-like, lancinating, or boring, whether there are free intervals (root pain), whether it always implicates the same area, etc. Minute questioning of this kind is especially necessary in headache. Insomnia and similar symptoms must also be carefully investigated, for instance, whether the patient soon falls asleep, whether sleep is frequently interrupted during the night, whether it takes a long time for the patient to get to sleep, and whether his sleep is sound. Often it is only as the examination progresses that we are able definitely to understand the complaints of the patient and to form a true conception of his condition.

In gauging the actual objective condition, we must first consider in a psychic and somatic respect the *totality of the impression* which the patient gives us. This total impression naturally embraces many individual points. The most important of these are the nutrition, the muscles and the bony structure, the carriage, the facial expression, the color of the face, the nature of the patient's movements, the character of the speech, the mental condition, and the intellection, etc. When we combine these individual points, we have a certain general impression of the patient. But only to a certain degree; for the general impression is conveyed not so much by the total of the previously mentioned observations, but by faint characteristics difficult to apprehend and very difficult to define. This will be readily understood after we have examined a considerable number of nervous patients. We often have a general impression of the patient who consults us, but we cannot consider him as representing a definite group of patients. This total impression, in itself, need convey no special diagnostic indication. Thus, in the course of time, a series of typical cases, usually of functional nervous disease, impresses itself upon the mind of the physician, so that at the first glance he readily recognizes the hypochondriac who, with "scientific" exactness and aided by written

reports, rears the structure of his condition stone upon stone so that it furnishes a flawless mosaic of his disease; we recognize the overworked, care-worn, unsteady neurasthenic, and in contrast to him the often stout, ruddy-cheeked, and plethoric angio-neurotic, then the "spiritual," pale neuropathic who always appears to be suffering. The bony, thick-set epileptic with his stupid look and clumsy movements, the tall, thin, pale tabetic with his slight uriniferous odor, the helpless paralytic who at once awakens our sympathy, the pale, choreic child with its fine rococo face, the quarrelsome and disagreeable victim of accident who seeks redress, these are a few of the types from the clientele of the neurologist which give characteristic "total impressions." The general impression immediately conveyed to us by these types does not relieve us from the obligation to investigate for certain *individual* points often found in nervous patients, and known as *stigmata of degeneration.* Among these stigmata are the abrupt, narrow, and high hard palate, the irregular conformation of the teeth, the thick lips, the adherent lobes of the ear, the poor development and slight individuality of these lobes, the retreating forehead, and other irregularities of the face or skull; we must also bear in mind the existence of embryonal anomalies of development, such as hare-lip, superfluous fingers and toes, malformations of the genitalia, anomalies of the skin, such as ichthyosis, abnormal pigmentation and many other things. I desire to call attention to two peculiarities that I have relatively often found in neuropathics, namely, medullary nerve fibers in the eye-ground and a striking similarity in the formation of the face of blood relatives.

I have so frequently noticed among neuropathics a resemblance between mother and child that I am inclined to regard this as more than a mere coincidence.

The neurologist, more than any other specialist, must direct his attention to *the face* and its *expression.*

The contraction of certain muscles of the face, above all, of the frontalis, as well as the implication or non-implication of the muscles of the face in active conversation, or their appearance during emotion, is conspicuous. In some diseases the expression of the face is so typical that we can from this at once make a diagnosis; for example, in paralysis agitans, in brain tumor, and in Graves' disease, etc.

From what has been stated, the physician will have had sufficient opportunity during the examination of the patient to form an opinion as to his mental and psychical condition, his manner of speech, whether he speaks spontaneously or only after questioning, whether conversation is ready and loud or whether he speaks in a low, depressed tone, whether he is quiet or excited, whether he at once understands and correctly answers—these facts in combination will enable us to form some opinion in regard to the psychical and intellectual functions. Should we still be in doubt as to the intellectual integrity of the patient, we may give him easy problems in arithmetic, or ask him to relate some details of his occupation, may test his memory for remote and recent events, etc. If we suspect hallucinations and similar conditions these must be investigated. The preceding will by no means acquaint us with the complete psychical status, but it exceeds the limits of pure neurological diagnosis as well as the limits of our theme.

THE SKULL AND THE VERTEBRAL COLUMN

(Form and size of the head, cicatrices upon the skin, osseous cicatrices, depressions of bone, elevations of bone of periostitic nature, thickening and gelatinous composition of the scalp in tumors, etc., *bruit de pot fêlé*, murmurs, sensitiveness to pressure of the nerve trunks, position of the spinous processes, movability of the vertebral column, manner of testing, examination and suspension, sensitiveness of the vertebræ to palpation, Röntgen photography, lumbar puncture, cytodiagnosis.)

On account of the importance of the nervous central organ to the entire organism special attention must be given to it in examination.

Marked deviations of the head from the normal in size or shape are at once obvious. A diminutive skull and a conspicuously large one are often found in idiocy, imbecility, and congenital nervous anomalies. Rickets and brain tumors often produce great enlargement of the skull.

In the diagnosis as well as treatment it should be borne in mind that an abnormally large skull sometimes indicates hydrocephalus during infancy. Quincke has proven that enlargement of the skull occurs in this way in some persons, occasionally also from trauma, or in consequence of some infectious febrile disease, and may indicate the renewed development of meningitis serosa.

An abnormally long, or flat, or abnormally round skull often has no pathologic significance. But, in contrast with this, the so-called bird-skull develops only in those who are psychically below par.

On inspecting and palpating the skull the first point to be noted is whether there are cicatrices on the scalp. The patients often have forgotten the existence of these, nevertheless, they are occasionally—for example, in epilepsy—of the greatest importance.

We must differentiate sharply between physiologic *depressions* in the skull and the protrusion of the bones, between pathologic traumatic depressions and thickening of the bones.

A physiologic thickening of the bone which is often misunderstood is found at the upper insertion of the tabular part of the occipital bone. Certain thickenings of the cranial bones, often insignificant or but slightly prominent, which may sometimes be pressed in by the firm finger, are of great pathologic importance. They are chiefly situated in the region of the parietal tuberosity and are extremely painful. These thickenings are due to *syphilitic periostitis* which causes the acute headache from which these patients suffer. The swellings are often overlooked; therefore, in all cases of intense headache the physician must search for a periostitis. In rare cases this periostitis to which the headache is due is so slight externally as to produce no palpable swelling. One or several circumscribed areas intensely sensitive to pressure render the diagnosis of specific periostitis certain.

A more diffuse, instead of circumscribed, area of the skull, much less intensely painful upon pressure, is occasionally observed in *rheumatic* affections of the galea, in brain tumor, and in some cases of hysteria.

In palpating the skull of patients with brain tumor I have often observed a symptom which I will now describe, a quite noticeable *thickening of the scalp*, especially of the anterior portions. While the scalp of the normal person is thin, tense, elastic, and almost adherent to the bone, in these patients on palpation it seems as though a serous infiltration, a loose and soft pliable

mass, were under the fingers. The surface of the bone can, therefore, no longer be felt by the palpating finger.

In patients with tumor there is another symptom, which was first described by English authors. Upon lightly tapping the skull in some cases of brain tumor or serous meningitis with the fingers or with the metallic side of a percussion hammer, a peculiar succussion sound is heard, such as is evoked by tapping a window-pane which is a little loose or upon tapping a cracked pot (*bruit de pot fêlé*). This sound may also be elicited under normal conditions in early childhood, and is then of no diagnostic value.

An attempt has been made to auscultate the skull, but with little success. In neoplasms and other organic diseases within the skull, we sometimes hear *murmurs* with the stethoscope. The differentiation of pathologic murmurs in the head from normal ones requires practice.

Among the symptoms still to be considered in examining the skull is sensitiveness to *pressure* over the trunks of the fifth and the occipital nerves.

The branches of the *fifth* nerve are susceptible to the pressure of the finger at the supraorbital notch, at the infraorbital foramen, and the mandibular foramen; the occipital nerve responds at a point between the mastoid process and the processes of the vertebræ. The points enumerated by no means represent the only pressure points of these nerves in neuralgia, nor are they always sensitive to pressure in actual neuralgia. But the extreme tenderness of these points which causes the patient to cry aloud, *in contrast to the painlessness of the surrounding tissues upon pressure*, is to be regarded as true pressure pain of the nerve. Anemics, hysterics and neurasthenics will often tell us that a nerve trunk is sensitive to pressure, but actual tenderness on pressure can be assumed only when the previously mentioned condition is observed.

The examination of the **vertebral column** which encloses the spinal central organ is less difficult than that of the skull. Here we must observe the following points: The normal *position* of the *spinous processes* and the vertebræ belonging to them, the normal S-shaped curvature of the entire column, and its normal passive *movability*. The lateral or posterior deviation of one or several spinous processes is not readily overlooked. This, however, is the case in the absence of lumbar lordosis which sometimes occurs—apparently from reflex causes—in tumors of the vertebræ and spinal cord, and even in hysterical conditions. Its possibility must also be borne in mind in the various forms of arthritic disease of the vertebral column and in ankylosis of the vertebral column.

We test the movability of the vertebral column by having the patient bend forward, backward and laterally as far as possible, and also turn the trunk laterally while the feet are close together. Next we ascertain whether sudden and brisk movements produce pain in the vertebral column. To demonstrate this we strike the patient a light blow upon the parietal region—protected by the hand—or the patient may be caused to bend laterally by a slight blow against the shoulder, or he is merely shaken. If he is sitting, the thorax may be tightly grasped, and the thoracic vertebræ be moved laterally against the remaining parts of the vertebral column. Finally, in doubtful cases, suspension may be practised, or, while the patient is in the dorsal decubitus, his legs may be pulled, or while fixed at the hip-joint they may be suddenly moved. I detail these manipulations because, when there are complaints of

pain in the back, it is often of the greatest assistance in the treatment to know whether a small pathologic focus, a tumor or the like, is hidden in the vertebral column. In such cases we should never be content with a purely neurologic diagnosis.

The *spinous processes* show a tenderness upon pressure and palpation which is generally of little diagnostic importance.

This is found in neurasthenics and hysterics (spinal irritation) often in varying intensity and with a varying localization as to height. It usually does not reach a high degree. If, however, one or two of the spinous processes are invariably painful on pressure and palpation, there is probably a vertebral affection. Here, as in all other doubtful cases, a *Röntgen photograph* is decisive.

As a supplement to examination of the vertebral column we must briefly describe *lumbar puncture* and cytodiagnosis. Lumbar puncture, introduced by Quincke,[1] enables us to determine the composition of the cerebrospinal fluid. Cytodiagnosis, a process originated by French investigators, particularly by Raymond's pupils, represents merely the special examination of the cerebrospinal fluid, namely, the determination of its cellular contents.

Lumbar puncture may be performed in the sitting posture, but, according to the inventor of the method, is best done in the left lateral position. In this position we introduce a slender trocar in the middle line, or somewhat laterally, between the spinous processes of the third and fourth or the fourth and fifth lumbar vertebræ. If, following the first attempt, there is no lessening of the resistance of the soft parts to show that we have penetrated the dural sac, we may probe with the tip of the needle and move it about laterally until it enters the dural sac. Then the mandrin of the trocar is withdrawn, a piece is added which connects it with a rubber tube, the other end being attached to the cannula, and, elevating the tube, we determine the pressure of the fluid, that is, its height in the tube. After this has been done a few cubic centimeters of the fluid are allowed to exude. The amount of fluid withdrawn must not be large (upon the average from 20 to 30 c.c.), for the pressure of the cerebrospinal fluid should not be too rapidly nor too greatly reduced. After evacuating the fluid, the needle is rapidly withdrawn and the small cutaneous wound closed with cotton and collodion. To diminish the pain from the insertion of the needle, the skin should be anesthetized with ether spray or ethyl chlorid. For the technical and other details of this slight operation the reader is referred to Quincke's article on lumbar puncture.

Different instruments have been suggested for lumbar puncture. The small instrument advised by Quincke has always given me good results. The operation in itself is generally quite insignificant, and only particularly unfavorable circumstances can lead to disagreeable results. On the other hand, I cannot deny that the performance of lumbar puncture in nervous patients, who are often hyperesthetic and excitable, particularly in private practice, is not always such a simple matter as it is represented to be by most authors who report instances from their clinics and large hospitals. The most important data concerning the cerebrospinal fluid are as follows:

The specific gravity of the normal fluid varies between 1.006 and 1.007.

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[1] See this volume.

According to Quincke, the pressure within the dural cavity amounts to 40 mm. and 130 mm. water. The pressure may fall with a decrease in blood pressure, and (this is more often the case) it may rise with any increase of brain pressure (tumor, hemorrhage, etc.). The normal cerebrospinal fluid has the transparency of water, is colorless, contains a little albumin (0.2–0.5 per 1,000), and sometimes a substance showing reducing properties with a test for sugar.

A distinct amount of albumin, one per 1,000 or more, as well as a turbid composition of the fluid, indicate recent inflammatory processes in the brain and spinal cord; but a slight amount of albumin and a colorless, clear fluid favor chronic hydrocephalus. Changes in the specific gravity have a similar diagnostic significance. Blood is normally present only in slight amounts, provided it originates from the wound made by the introduction of the needle. In hemorrhages of the brain or spinal cord there is often a marked admixture of blood.

Among the cellular constituents of the normal fluid obtained by puncture are some scant lymphocytes (upon an enlargement of from 400 to 500 about 3 to 4 in the field). The examination and diagnostic utilization of these cellular constituents of the cerebrospinal fluid forms the so-called cytodiagnosis.

For cytodiagnosis we require only 3 or 4 c.c. of the cerebrospinal fluid. Puncture, therefore, if only for the cytodiagnosis, may be performed with a very fine needle. According to French authors the puncture fluid should first be centrifugated for 20 minutes with a rapidity of 3,000 revolutions per minute. On removing the fluid, the sediment is microscopically examined. As previously stated, there are normally but a few small mononuclear lymphocytes in the fluid. The presence of polynuclear and large mononuclear leukocytes favors acute inflammatory processes in the region of the brain or spinal cord, or in their membranes.

If the number of small lymphocytes exceeds the minimal of 3 or 4 to the field, their presence is of much greater practical and diagnostic significance than the presence of polynuclear and large mononuclear leukocytes in the residue. For, according to Raymond's adherents, which other authors confirm, in all syphilitic and metasyphilitic affections of the central nervous system, therefore, in cerebrospinal syphilis, specific hemiplegia, specific myelitis, progressive paralysis, tabes dorsalis, etc., there is a conspicuous lymphocytosis of the liquor cerebrospinalis so that 20 or more cells appear in a single field of the microscope. Of course, the appearance of lymphocytes in the cerebrospinal fluid is not absolute proof of the previously mentioned conditions, for in addition to syphilitic and metasyphilitic affections of the central nervous system they have also been found in quite unlike pathologic processes, in multiple sclerosis, in herpes zoster, in recent hemiplegia, etc. And, what unfortunately almost vitiates this sign, lymphocytosis of the cerebrospinal fluid has also been found in secondary syphilis without any implication of the nervous system. In spite of these disadvantages, lumbar puncture, according to our present knowledge, is a most valuable diagnostic aid.

VASOMOTOR AND TROPHIC DISTURBANCES

Of the nature and occurrence of most trophic and vasomotor symptoms we still know very little. They occur in organic affections of the brain, the spinal cord and the nerves, as well as in various functional diseases, and may implicate all the tissues of the body.

TROPHIC DISTURBANCES OF THE SKIN AND CUTANEOUS STRUCTURES

(Color of the skin, temperature of the skin, "blushing," flushing of the face, urticaria factitia, edema, hemorrhage, disturbances in the secretion of saliva and of sweat, myxedema, scleroderma, glossy skin, ichthyosis, loss of pigment of the skin, herpes, neurofibromata, cutaneous ulcerations, perforating ulcer of the foot (mal perforant), changes in the hair, changes in the nails, changes in the teeth.)

We are most familiar with the vasomotor and trophic disturbances of the skin, for these may be recognized by mere inspection, palpation being rarely necessary.

The *temperature and color of the skin*, its thickness, its greater or less infiltration, and its reaction to mechanical irritation, are observed when investigating for vasomotor disturbances. Under certain circumstances (hysteria and neurasthenia) the skin of almost the entire body assumes a bluish, marbled appearance. In certain peripheral areas, such as the region of the knee, the hand or the foot, a distinctly bluish discoloration is observable, the area is often cool and sometimes moist. In prolonged paralyses—no matter of what origin—this cyanosis with coolness is observed in some of the extremities, or in parts of them. Here circulatory disturbances due to the absence of normal muscle and tissue movements is apparently the cause. In other cases (Raynaud's disease) certain portions of the body, usually the fingers or toes, are paroxysmally very pale and cool, and contain a minimal amount of blood; or, on the contrary, they are exceedingly red, swollen and painful (*erythromelalgia*). Under still other circumstances (paralysis agitans, the climacterium), peculiar fluxions appear with sudden, profuse, arterial distention and flushing of the larger vascular areas of the head or trunk, and disappear in one or two minutes.

Erythrophobia is usually found in congenitally neurotic individuals and the like with compulsory ideas (*idée fixe*). It is a reddening of the face on the slightest possible irritation (usually psychical), of which ordinarily no account is made, and simultaneously with this (on account of the pathologic psychical disposition) there is a distressing fear of blushing. Periodic or permanent unilateral flushing of the face indicates a unilateral disease of the sympathetic nerve. The sudden reddening or pallor of *epileptics*, with or after attacks, and the unilateral flushing or pallor of the fact in *migraine*, need only passing mention.

The physician, while examining a hysteric or neurasthenic and while the patient is undressing, is often astonished to see, directly under his eyes, flaming red flakes develop which are confluent, large or small in size, but not elevated. These are usually situated upon the neck or chest (*vasomotor collar*) and disappear just as rapidly as they appear, usually after the patient has

become composed. Moreover, we find that in these patients copious perspiration is very readily induced, and also the appearance of an urticaria-like eruption after the ingestion of certain foods (such as strawberries, lobsters, etc.), as well as the so-called urticaria factitia (*dermographia*). The latter symptom may be evoked by drawing lines upon the skin with a hard, pointed object. After about a minute these lines appear as red or whitish bands elevated above the level of the skin, and they may persist for hours. In urticaria factitia we are apparently dealing with small serous exudations from the vessels into the tissue, such as occur in inflammations.

True *edema* will also be found, sometimes with cyanosis and extremely cold skin—that due to nervous disturbance. The first form occurs in some varieties of neuritis, in cerebral hemiplegia (particularly of the middle brain), and in similar affections. Hysterical edema, that attributable to an organic nervous cause, is sometimes accompanied by other symptoms of stasis; sometimes, however, it exists as a simple, or hard, or doughy serous exudation under the normally colored and normally warm skin (the so-called *flying edema* of hysteria). In addition to the purely nervous edema, stasis edema due to purely mechanical causes also occurs, for instance, edema of the eyelids in tumors of the brain, etc.

Vasomotor disturbances combined with hemorrhages into the tissue are exceedingly rare. Relatively the most common of these are *mucous membrane hemorrhages* from the genital canal or the respiratory tract in hysterical women. The small hemorrhages into the conjunctivæ or the skin of the face observed in epileptics after an attack are purely mechanical hemorrhages produced by venous stasis during the seizure.

Irregularities in the *secretion of saliva and sweat* belong to the vasomotor disturbances which occur in the structures of the skin. Of course, the dribbling of saliva in paralysis of the facial nerve, in bulbar paralysis, and similar clinical conditions is primarily due to an inhibition of the normal closure of the lips or the impossibility of swallowing the saliva. In certain spasmodic conditions of epileptic or organic nature, the production of saliva appears to be actually increased.

Disturbances in the *secretion of sweat* will usually be found in the form of *hyperhidrosis*, that is, hypersecretion. *Too slight secretion* of sweat and also of the sebaceous glands sometimes appears, the skin being remarkably dry, scaly, and fissured (occasionally in neuritis and in disease of the gray substance of the cord). The diminution of the secretion of sweat is often noticed by the patient; for example, the tabetic refers to the absence of sweating as an important etiologic factor in the development of his disease.

Hyperhidrosis may be distributed over the entire body, especially in functional affections, hysteria, neurasthenia, Graves' disease, etc., and is frequently accompanied by dermographia or nervous and cardiac conditions, feelings of anxiety, etc. Limited to certain parts of the body or to members, hyperhidrosis is also seen in neuritis, in spinal, and in cerebral processes. It is an interesting fact that sweating may occasionally be abnormally induced by certain sensory irritations. For instance, I recall a patient with syringomyelia who, a few minutes after eating a spiced pickled herring, showed active unilateral sweating of the face.

Trophic disturbances of the skin occur partly in connection with the

previously described vasomotor symptoms, but also without these. They are much rarer in purely functional diseases than in the organic.

The normal thickness and texture, the amount of fatty tissue, etc., of the skin, is frequently altered by the influence of trophic disturbances. Under certain circumstances, fat increases to an enormous extent throughout the skin of the entire body, and pain and other general symptoms appear (Dercum's disease, adiposis dolorosa), or the skin of the whole body becomes peculiarly thickened and swollen and resembles edema, but does not pit in the manner characteristic of edema (*myxedema*). In patients suffering from brain pressure with *chronic serous meningitis* I have several times seen a skin condition of the face, the trunk, and especially of the hands, which closely resembled myxedema, but I am unable to explain these changes in the skin (*Neurol. Centralbl.*, 1900, p. 548).

Another change which also implicates wide areas of the skin is found in *scleroderma*. Here extensive areas of the face, the arms, the back, etc., have a peculiarly tense, glistening, and usually very red appearance, and almost convey the impression of being firm, smooth, surface cicatrices. Here the skin cannot be picked up from the underlying structures; on the contrary it appears to adhere to them. If we succeed in pinching up a small fold of skin, it feels between the fingers like a coarse, tough, and inelastic piece of leather.

A similar condition—but limited to much smaller areas of the skin—is the so-called *dermatomyositis*, an extraordinarily rare affection.

If, in the previously mentioned conditions, the skin increases in thickness and toughness, the opposite, atrophy of the skin and of all its structures in consequence of nervous disturbances, occurs under the following circumstances. The best known form of atrophy of the skin is the so-called "*glossy skin.*" In this the skin becomes so thin that by lightly passing the finger over it numerous small folds are formed; it also glistens, is usually of a yellowish color, and easily desquamates. This pathologic glossy skin closely resembles the physiologic atrophic skin of the aged. Glossy skin occurs in neuritis —more rarely in spinal processes.

An abnormal and intense dryness and desquamation of the skin is seen in *ichthyosis*. In neuropathic subjects this disease is frequently seen upon the skin of the leg, particularly in the region of the tendo Achillis, hence this symptom may perhaps be regarded as one of the so-called stigmata. Sometimes the scaliness and desquamation are not great enough to warrant its being called ichthyosis, but, nevertheless, it is sufficient to attract the attention of the examiner and to be regarded as pathologic. The patient usually observes this condition in himself or in other members of his family before his attention is called to it by the physician.

In old cerebral paralyses, especially those of the upper extremity, another peculiar change in the skin is often observed. The *pigment* in the skin of the hand and fingers is decreased. The skin has a rosy, delicate, and childlike appearance, and is smoother and softer than on the normal side.

This cutaneous change is often combined with general atrophy of the bones of the finger and a peculiar tapering of the fingers. A decrease in the pigmentation of the skin is also seen in neuritic processes.

I must still mention a few trophic disturbances of the skin which pro-

duce conspicuous changes, namely, an *eruption* of *herpetic vesicles*, tumor-like neoplasms of the skin (neurofibromata), and ulceration of the skin. The eruption of herpes accompanied or preceded by severe pain, and the vesicles which are usually arranged in rows and which at first contain serous fluid (subsequently pus if in the area of a peripheral nerve) form such a characteristic phenomenon that it cannot fail to be recognized. The previously mentioned circumstances and the occasional occurrence of hypesthesia and hyperesthesia in the area of the affected nerves favor the presence of neuritic processes. As a rule, herpes occurs in the course of the intercostal nerves (a possible disease of the vertebra should then be considered!) but it also occurs in the area of other peripheral nerves.

[It is now pretty definitely proven that true herpes zoster is invariably associated with an inflammatory and hemorrhagic process in the posterior root ganglion. Irritation of the posterior roots or the posterior ganglia in spondylitis, or involvement of the intercostal nerve, may be attended by an herpetiform eruption, which, in the case of the peripheral nerve, is limited to the corresponding cutaneous distribution of the nerve involved.—Ed.]

Neurofibromata of the skin are small nodules under and in the skin which are sensitive to pressure, usually very movable and only discovered when the patient consults the physician for other nervous disturbances than the condition of the skin. Very often, simultaneously with these relatively harmless tumors of the skin, neurofibromata of the same nature form on the anterior and posterior roots of the spinal cord, and by destruction of the root fibers produce severe pathologic conditions. If, on the other hand, we first discover the neurofibromata of the skin, we must be exceedingly careful to investigate the other nervous symptoms of the patient.

The most conspicuous trophic alteration of the skin is caused by *ulcers*.

If no other reason for cutaneous ulceration can be found, trophic nervous disturbance must be thought of, particularly that due to disease of the spinal cord, to tabes dorsalis (mal perforant), or to syringomyelia. In the last named disease the condition becomes complicated, for, besides genuine ulcers not due to trauma, others appear in consequence of a traumatic defect in the skin and sensory disturbance, and the defective tendency of the tissue to heal produces true ulcerations.

Similar to the ulcerations of syringomyelia is a rare variety of ulcers seen in so-called Raynaud's disease, in diabetes mellitus, and in similar affections. Here there is a local *tissue necrosis* due to the spasmodic closure of vessels, and a succeeding ulceration at the point of necrosis.

Almost as common as trophic disturbances of the skin is an implication of tissues adjacent to the skin, the hair, the nails, etc. *Falling out of the hair*, alopecia, sometimes depends upon functional nervous disturbances; in myxedema there is almost invariably a marked alopecia of all of the hairy parts of the body including the eyebrows. A local falling out of the hair occurs in neuritis, although an abnormal growth of hair in the diseased area is more common. That alopecia is often limited to the areas of peripheral nerves is shown by the most familiar form of baldness. In the baldness of a person who, fortunately, cannot be regarded as a nervous patient, only the areas of the fifth nerve are usually bald, the hair remaining in the region

of the occipital nerve. General hirsuties (hypertrichosis) over the entire body is looked upon as a stigma of degeneration.

Grayness of the hair—either general or partial—in neuralgia or other severe psychical conditions is well known to the layman. It is less well known that, in mental diseases and some other nervous disturbances, similar changes may occasionally take place in the color of the hair and its entire composition. Cases are reported in which blond, straight hair became red and curly or dark hair turned white. The texture of the individual hairs is changed only in the last mentioned case. In myxedema, also, the hair which remains upon the head becomes stiff, ropy and completely loses its normal luster. In neuritic hirsuties, the hairs upon the surface of the body are abnormally long and thick.

The symptoms presented by the *nails* of the fingers and toes after trophic disturbances are quite uniform: Abnormally rapid growth, the normal form of the nail changing to a marked curve, the appearance of longitudinal and transverse ridges, a friable condition, and the gradual loss of the entire nail. These changes are usually attributed to the peripheral neuron, and preëminently to the spinal processes (tabes, syringomyelia). They are also occasionally found in cerebral hemiplegia. Some of these changes in the nail occur in rare cases of functional nervous diseases, such as prolonged paralysis or contractures of the individual members.

A symptom seldom observed is formed by the numerous small white spots which in neurotics often cover the entire nail from above downward, and which may appear and disappear periodically. A smaller number of these spots may be observed in almost any person.

In examining the *teeth* we must look for well known defects in growth and development which occur in rachitis and congenital syphilis. In neurologic diagnosis the finding of the characteristic notched semilunar and excavated free border with the peculiar oblique end makes us suspect *hereditary syphilis* (Hutchinson's teeth). Regularity or irregularity of the teeth is also to be observed. Superfluous teeth projecting from the surface of the jaw are not rarely found in nervous patients and neurotics. In trophic disturbances in the region of the fifth nerve, for example in tabes dorsalis, the teeth sometimes fall out singly or several at a time, without much pain. On the other hand, in neuralgia of the fifth nerve, there is neither trophic nor other recognizable change in the teeth, hence I must issue a warning against unnecessary extraction of teeth.

TROPHIC DISTURBANCES OF THE MUSCLES

(Hypertrophy, atrophy, atrophy in the course of the cranial and the spinal nerves, hypertrophy, pseudo-hypertrophy, occurrence of pseudo-hypertrophy, atrophy, simple atrophy, degenerative atrophy.)

Trophic disturbances of the muscles appear as hypertrophy and atrophy, and are recognizable by inspection, by palpation, and by mensuration. The practised eye soon notices—particularly on comparison with the normal side—any abnormal depression or prominence of the skin from atrophy or hypertrophy of the muscular tissue beneath.

The results of mere inspection are confirmed by palpation and mensura-

tion. With a marked development of fat, inspection will not suffice, and the latter methods become necessary. It is quite comprehensible that trophic changes of the deeply situated muscles cannot be recognized at all, and in other muscles can only be determined with difficulty.

The muscular areas most likely to be involved in trophic changes are, as a rule, the following: Along the course of the cranial nerves, the face, the tongue, and the lips. Atrophy of the temporal muscles is recognizable by the marked depression in the temporal region; that of the masseter muscle on palpation of the sunken cheek; atrophy of the muscles on one side of the face (facial hemiatrophy) by the singular appearance of the diseased side of the face which sometimes almost resembles the bare skull.

Only an extreme atrophy of the *lips* is noticeable at the first glance. Usually we must take the lips between the thumb and index finger in order to detect this condition. But in old persons the lips are physiologically very thin.

Atrophy of the tongue is readily determined, whether it be unilateral or bilateral. The tongue appears withered, contracted, and dry; its surface is fissured. The extremely small size of the organ, especially upon protrusion, is pathologic and unmistakable, and in peculiar contrast with the normally opened mouth.

In the neck and nape of the neck atrophy of the *sternocleidomastoid* and of the upper trapezius is most easily recognized. These muscles are readily palpated, and they are so necessary for the proper contour of the neck that their atrophied condition at once attracts attention. Moreover, they are among the muscles which not rarely undergo true hypertrophy (for example, in patients with tic). In such cases, the sternocleidomastoid, even during rest, stands out prominently as a massive rope under the skin.

Hypertrophy of the upper *portion of the trapezius*, recognizable by its enormous prominence, which, even under normal conditions, may be distinct, should only be diagnosticated when the shoulder on the involved side is not higher during rest, for only thus can a permanent contraction of the trapezius (which also causes the muscle to appear more massive) be excluded.

The depression of the *supraspinatus* fossa is readily determined, but it is not so easy to decide whether atrophy of the trapezius or the supraspinatus is the cause. For this purpose a test of the function and an electric examination are necessary.

In atrophy of the *deltoid*, the shoulder viewed anteriorly has an angular appearance (from the prominence of the acromion process and the external end of the clavicle). Posteriorly the stiff and angular external contour of the upper arm is conspicuous. Turning to the *muscles of the trunk*, the determination of atrophy of the middle and lower portion of the trapezius will occasion no perplexity. Atrophy of the *teres major* and the *rhomboideus* is much more difficult to perceive; to determine it in the first mentioned muscle we must when palpating compare the normal with the affected side; an atrophy of the rhomboideus while the trapezius which covers it remains intact, also of the latissimus dorsi, can be assumed only when the palpating finger feels the ribs more distinctly than usual under the superficial muscle masses.

Atrophy or aplasia of the *pectoralis major* on the anterior side of the

trunk is revealed by the presence of an abnormal and ugly groove below the clavicle. Atrophy of the *serratus anticus* is readily shown by the absence of the characteristic zigzag of the muscle in the anterior axillary line. The intercostal muscles, on the contrary, lie so deep that atrophy cannot be detected. In very thin persons only is it possible to recognize *atrophy of the abdominal muscles.*

Atrophy of the muscles of the *extremities* is readily perceptible because these muscles, even during rest, are more susceptible to inspection and palpation than the musculature of the trunk, and, moreover, the many changes in the position of the extremities give us more opportunities thoroughly to examine a definite muscle.

The scope of this article does not permit us to investigate all of the muscles of the extremities in regard to atrophy. In those cases in which mere palpation and inspection are insufficient to enable us to decide whether or not the muscle is atrophic, a knowledge of the special physiology of the muscles will make the diagnosis positive. Many common forms of muscular atrophy may be recognized at the first glance. Among these is atrophy of the *biceps,* of the *extensors* of the *hand* and *fingers,* of the *interossei* and the muscles of the *ball of the hand,* of the *quadriceps,* the *tibialis anticus,* the *muscles of the calf,* etc. In examining for muscular atrophy in the extremities it should be borne in mind that the left upper arm and forearm are usually 1 cm. less in circumference than the right [provided the person is right-handed.—J. L. S.].

Besides atrophy of the muscles I have spoken of *hypertrophy of the muscles* as also a trophic disturbance. True hypertrophy of the contractile muscular substance is rare. It is occasionally observed in the affected members in long standing cases of clonic spasm, for example, post-hemiplegic chorea and the like. *Pseudo-hypertrophy* of the muscle, in which the entire volume of the muscle appears enlarged from the fact that fat has been deposited between the individual muscle fibers, is much more frequent, and appears chiefly as *dystrophia muscularis progressiva.* Here the vulnerable points for pseudo-hypertrophy are the triceps, the infraspinatus, the muscles of the calf, the quadriceps and the gluteal muscles. If these muscles appear to be abnormally large, pseudo-hypertrophy is very probable. This becomes certain if, on palpating the muscle when at rest as well as during extension, we feel a soft and easily movable mass instead of a firm and tense tissue.

While pseudo-hypertrophy of the muscles occurs, as a rule, in only one disease, progressive muscular dystrophy, atrophy of the muscle is noted in various pathologic processes. Disease of any part of the great corticomuscular tract may be accompanied by muscular atrophy. Two special varieties of atrophy produced by the arrest of function or by the intense effect of irritants upon the muscle are so-called *inactivity* and *pressure atrophy.* Inactivity atrophy appears to be closely related to *reflex atrophy.* In this condition the extensor muscles in the neighborhood of a diseased joint undergo wasting—the quadriceps in disease of the knee-joint, the triceps in affections of the elbow—probably because the centripetal irritation of the sensory nerves and reflex collaterals no longer takes place normally but is pathologically conveyed into the anterior horn cells. Our diagnosis primarily depends less

upon classifying the atrophy than upon deciding whether it is "*simple*" or "*degenerative.*"

The division of atrophy into these two groups is pathologico-anatomical. In simple atrophy there is only a quantitative diminution of the muscular mass, in the degenerative form the muscular substance has undergone chemical change, "degeneration." Mere inspection and palpation will never reveal with certainty which form is present. This can only be ascertained by *electric examination.* In simple atrophy there is either no, or merely a *quantitative,* change in contractility; in degenerative atrophy there is a *qualitative* change in contractility which manifests itself by the sluggishness of the contraction, and by the sluggish arrest of the electrical contraction of the muscles caused by direct galvanic muscular irritation. Normally, all electrical muscular contractions take place with lightning-like rapidity.

The diagnostic significance of the character of the atrophy can be appreciated when we consider that degenerative atrophy is produced only by injuries or disease of the motor nerves or the anterior horns of the gray matter of the spinal cord. When we differentiate degenerative atrophy we have a valuable aid for the topical diagnosis. In the cases in which, for any reason, an electrical examination is impossible, *mechanical muscular irritation* is advisable (by means of a sharp blow of the pleximeter upon the muscle mass); with degenerative atrophy this will produce a sluggish contraction. In my experience mechanical irritation is a very poor substitute for electrical irritation, and frequently leaves us in doubt, because in some muscles, even normally, neither the contraction nor its cessation is so rapid as the contraction produced by electricity.

Of the relation of atrophy of the muscles to paralysis I shall say merely a few words. In certain cases, atrophy of a muscle too deep to be susceptible to examination may be concluded from the existence of paralysis even when it cannot be decided by palpation or inspection.

TROPHIC DISTURBANCES OF THE BONES AND JOINTS

(Hypoplasia of the bones, osseous atrophy in neuritis and after injury, changes in the bones and joints in spinal diseases, partial giant growth, acromegalia, intermittent dropsy, hysterical affections of a joint.)

The growth of bone depends upon nervous influences. In *infantile spinal paralysis* the growth of the bones in the affected extremity does not keep pace with that of the normal side. This is often the case in the *cerebral paralyses of children,* although it is not so conspicuous as in poliomyelitis. Sometimes only a unilateral, extremely small shoulder-blade reveals that the affected person has had infantile cerebral paralysis. In this case we are not dealing with atrophy of the bone, but with *hypoplasia,* a retardation of the growth of bone. True atrophy of the bone is usually observed in diseases of the peripheral nerves, with or without preceding injury. For instance, in polyneuritis the peculiarly thin and tapering phalanges of the fingers are remarkable; but *atrophy of the bones* of the extremities is more frequently observed in patients who have suffered from accidents. The latter phenomenon also indicates long existing hemiplegia or similar processes. Other trophic

disturbances of the bones which are often combined with similar disturbances of the joints are less common than atrophy. I refer chiefly to a peculiar change in the consistence of the bony tissue in which the normal firmness is lost and a friability of the bones appears. The joints show a similar vulnerability as well as a tendency to synovial thickening, deposits, and enlargement. Usually after slight trauma—unnoticed because of the coexisting analgesia—or even without a recognizable lesion, in these cases (syringomyelia, tabes dorsalis or similar processes) fractures of the bone, deposits, and thickening of the joints, subluxations and similar conditions appear.

Hypertrophic, as well as atrophic, disturbances take place in the bones. So-called *partial giantism,* in which a foot, a hand, etc., is abnormally large at birth, is formed during infancy or continues to develop, belongs to this category. The abnormal processes of growth which take place in acromegalia within a relatively short time (within months) and which affect particularly the hands, the feet, and the lower jaw, are generally more important. The soft parts also are involved in this abnormal growth of the bone. After seeing and investigating one typical case of this kind, a diagnosis can be made at the first glance.

Before dismissing the trophic disturbances of the joints I must call attention to an exceedingly rare affection, namely, so-called intermittent dropsy of the joint. This usually attacks the knee-joint without recognizable cause, there is neither pain nor reddening of the skin, it persists a few days and then disappears, to recur after the lapse of some time. The nature of this disease is absolutely unknown; it is presumed to be due to an affection of the nervous system.

I advise the greatest caution in making a diagnosis of hysterical swelling of the joints, an affection which has been described by some authors.

MOTILITY

(Muscle tonus, significance of the anterior horn cells of the spinal cord as the motor power station, increase and decrease of muscular tonus, the effect of other areas of the central nervous system upon the power station, the muscles of the body in a position of rest, the muscles innervated by the cranial nerves in a position of rest, the muscles of the face and eyes in rest, the muscles of the upper and lower extremity in rest, position of the pelvis, shoulder-blade, and muscles of the back. Passive movements, spastic conditions, recognition of slight spastic muscle resistance, active and passive contractures, clinical difference between active and passive contractures, influence of the psychical condition upon muscular tension, exclusion of this influence during examination, the value in diagnosis of determining the spastic character of a paralysis, relation of spastic to flaccid paralysis, occurrence of spastic conditions and contractures, Kernig's sign, contractures of paralysis agitans. Hypotonia, cause of hypotonia, clinical symptoms of hypotonia. Active movements, the test of active movements in the region of the cranial nerves, nerves of the forehead, and of the facial branches of the mouth, voluntary and emotional movements of the nerves of the face, paralysis, contracture, and atrophy of the tongue, paralysis in the bulbar sphere, in the muscles of mastication and of the larynx, dysarthritic speech disturbances, stuttering, hysterical stuttering, other hysterical speech disturbances, scanning speech, syllable stumbling, muscles of the nape of the neck and of the back, movements of the arm and hand, adynamia, myasthenic conditions, movements of the leg and of the foot, psychical and resistance movements, psychical and hysterical paralysis, abasia, weakness of the grasp and flexure of the hip in neurasthenia, occurrence of organic paresis and paralysis.)

The investigation of the motor sphere is of course not limited to the examination of *voluntary* movements. But, turning to these, we must first consider the tension of the muscle and combined with this the rest of individual portions of the body as well as their passive movability. Then only will we able to test *voluntary* movement, the coarse development of power and the faculty of muscular coördination.

Subsequently we shall consider involuntary movements and the principal types of spasm.

In the normal individual while awake the muscles of the body are not fully relaxed but are in a certain state of contraction. This slight degree of contraction is called the *tonus* of the muscle. Clinical researches make it appear highly probable that the normal tonus is not an autochthonous muscular phenomenon, but is produced from the spinal cord by centripetal sensory irritation, and is, therefore, a reflex condition. To understand this perfectly it is necessary for us to analyze—although in a diagrammatic manner—the mechanism of the motor central organs which is revealed as a *clinical* postulate.

Here, as the first and most important requisite, we must adhere to the fact that the *sole and actual* source of energy for muscular contractions, i. e., the *motor power station,* is situated in the large anterior horn cells of the spinal cord or in analogous cells of the medulla oblongata and the mid-brain. The physiological intactness of these cells is an absolute prerequisite for the normal function of the *entire* motor apparatus. Clinical facts lead us to the conclusion that the anterior horn cells form merely a power station, or a power station with potential energy, i. e., with the *ability* to discharge power; that, therefore, some *causative* factor is necessary to change the ability to furnish force into an actual discharge of force. Under normal conditions there is always—even in rest—such an active cause; namely, the thousands of irritations ceaselessly passing from the periphery to the spinal cord, and produced by the circulation of the blood, by the chemical processes in the tissues, by the action of the tissues upon each other, by the effect of air and clothing, or by the subcutaneous tissues, etc., upon the skin. All of these irritations transmitted to the spinal cord by way of the reflex collaterals through the posterior roots—to say nothing of the fact that they are transmitted also as conscious sensations by way of the long spinal cord tracts to the cortex of the cerebrum—are conveyed to the gray substance of the spinal cord. Here—reaching the terminal arborizations of the reflex collaterals—they surround the motor anterior horn cells and cause (like all sensory irritations) a motor correlation; namely, a continuous slight discharge of motor energy. We can, with great probability, assume the muscle tonus to be the effect of this continuous output of power from the power station of the anterior horn cells. The motor power station in the spinal cord is like a fire which is kept aglow by the constant addition of fuel from the periphery, and only flames up brightly when it is caused to do so—as we shall soon see—by other agency.

The intimate relation of the muscular tonus to sensory stimulation is shown, among other ways, by the fact that a pathological increase of tonus is diminished by lessening the peripheral irritation (absolute rest, warm bath, and the like) and, inversely, it may be increased by intensifying these irritations (cold and pain).

The reason for assuming a muscular tonus to be the explanation of pathologically increased or decreased muscular tension will subsequently be apparent.

Sensory impressions of the periphery are not the only agents by means of which the anterior horn cells are caused to give off force. On the contrary, the most distant areas of the nervous system may produce an effect upon the motor power station and a consequent discharge of force: For instance, the cerebellum, the basal ganglia, the trunk of the brain, and, last but not least, the cortex of the cerebrum. The action of the cortex of the cerebrum, the central convolutions, upon the motor power station of the spinal cord is by far the most important; it represents what has been called the conscious movement of the will. This simple relation between the brain and spinal cord in regard to the motor function must be borne in mind for it forms the base of our later descriptions. Therefore, *in the anterior horn cells is located the true, active, and absolutely essential power station*; the central convolutions act upon this power station only in that, at a given moment, the source of power sends an impulse of contraction into certain muscles. This cerebral effect upon the anterior horn cells is chiefly observed in what is known as the pyramidal tract, perhaps to a slight extent also in other tracts.

The relations of the cerebrum to the motor power station are not confined alone to the function just described. On the contrary, physiologic and clinical facts make it obvious that the cerebrum (perhaps also other parts of the brain) has a second action, opposed to the prior one, which prevents the too great development of power in the anterior horn cells. This inhibitive regulation of the continuous discharge of power, which according to our view is developed and promoted by an afflux of sensory irritations, must be assumed for muscular tonus from the obvious fact that in pathologic cases in which the power station, in itself completely intact, is disconnected from the brain, there is a decided increase of muscular tonus. We know less of the nature of the negative "inhibitive" influence of the cerebrum upon the anterior horn cells than of the positive effect of the cerebrum upon the spinal cord, but it is not impossible that the apparent inhibitive action is caused by other powerful influences.

After this deviation we return to the examination of the patient, and, in observing the symptoms of motility, the previously sketched brief diagram must be kept in mind.

The first test of motility is the *position of the muscles of the face when at rest*. Here we examine the wrinkles of the forehead, the position of the eyebrows and upper eyelid, the width of the apertures, the frequency with which the lids close, the position of the bulbi, the angle of the mouth, the nasolabial fold, and the width of the nasal opening. In these tests we must always compare the right with the left side. The majority of deviations from the normal in the position of these structures depend upon disturbances in the innervation of the facial nerve. Besides this nerve we must also consider the nerves of various muscles: Particularly the oculomotor (for the position of the eyes [strabismus] and for the position of the upper lid in rest [ptosis]) as well as the sympathetic nerve. The latter controls the position of the eye in the frontal plane by means of the smooth muscle of Müller which extends through the orbit, and at the same time regulates the normal opening

of the lids. Protrusion of the eyeball is most prominent in Graves' disease, and is here attributed to a disturbance in the innervation of the sympathetic. Decrease of the palpebral fissure, a sign of paresis of certain branches of the sympathetic, is found in many spinal processes, particularly in syringomyelia, as well as in diseases of the brachial plexus, and is sometimes congenital. Mere gaping of the palpebral fissure without protrusion of the eyeball indicates paresis of the orbicularis oculi supplied by the facial nerve. An abnormal dilatation of the palpebral fissure (usually hysterical contracture) is in rare cases produced by contraction of the facial muscles or of the levator palpebræ. We must observe whether the pupils are circular and of equal size. All of their other relations will be described under the reflex symptoms.

We will now turn to the examination of the *extremities in rest.*

In the *upper extremities* we should observe whether the head of the humerus is sufficiently firm in the socket (supraspinatus), and whether the arms, relaxed and drooping, are in the correct position midway between pronation and supination. When the objective findings are scant, a slight deviation from this normal middle position is often the only distinct sign revealed to the practised eye, and warns us to test minutely the rotators of the arm. We should also note whether the basal phalanges of the fingers are normal during rest, i. e., slightly flexed, or whether there is a tendency to extension and hyperextension (weakness of the interossei). Of course a conspicuous change of this kind will no more be overlooked than a conspicuous flexion of the hand in radial paralysis. But a slight degree of flexion of the hand may readily escape the eye.

The examination of the *lower extremities* during rest must be made with the patient in the horizontal position; here also a slight outward or inward rotation of the leg may elude observation.

On the other hand, a slight drooping of the tip of the foot, such as occurs in paresis of the extensors of the foot and in hypotonia of the muscles of the lower leg, will be very noticeable. The heavy mass of the quadriceps upon the upper thigh will sometimes appear as if it had slid off externally, thus making the outline of the femur more prominent than normal, or we may note that the entire posterior surface of the leg—even the popliteal space—seems to be adherent to the underlying bone. These are simultaneously indications of flaccidity of the muscles, or hypotonia.

Another symptom the observation of which is of decided significance, is the elasticity of certain muscles, for instance, a permanent hyperextension of the great toe. This is found with especial frequency in some forms of ataxia and in spastic conditions.

The examination of the erect trunk during rest gives us opportunity to investigate further what was observed in the legs when in a horizontal position, particularly to estimate the condition of the gluteal muscles. The *position and course* of the so-called gluteal fold at either side forms a criterion for the examination of the gluteal muscles.

In *the trunk* we should observe any lateral deviation, any curvature of the vertebral column, any inclination of the pelvis and possible asymmetry of the iliac crests. In some cases of paresis with pain in one leg, the diseased side of the pelvis is always held higher, even during rest. An abnormal position

of the pelvis—too great an inclination in the horizontal plane—is most likely to be found in persons suffering from muscular dystrophy.

The position of the shoulder-blades, their distance from the median line, the normal, nearly vertical, lines of their internal borders, and their normal position upon the thorax, should be noted in every case.

Abnormal tension of the muscles which connect the pelvis with the vertebral column is usually of a reflex nature. This is most distinctly pointed out by the unilateral or bilateral prominence of the rigid, contracted erector trunci.

After observing the condition of the members and the trunk during rest, we must proceed a step further, and investigate the *passive motility,* i. e., the possibility of excursions of the different parts of the body toward each other—for this test the patient must entirely relax. Normally, passive movement of the extremities is not perfectly free. On the contrary, when an arm or a leg of a normal person whose attention has been diverted is flexed or extended, we note a certain resistance—I might say a smooth and gliding one—like that felt on moving an oiled piston to and fro in a syringe. This physiologic muscle resistance is due to the tonus of the muscles, the nature of which I have attempted to explain. If, therefore, the tonicity of the muscles is increased, passive movement is the more difficult. If the tonus is decreased, passive movement is facilitated. Of course, we presuppose that the joints are perfectly free. Increase of muscular tonus, according to its degree, is designated as ***muscular spasm, hypertonia,*** or a ***spastic condition*** of the musculature. Decrease of muscular tonus is called *hypotonia.*

A moderate ***spastic condition*** is present if, on attempting passive movements—sometimes in flexion, more often in extension—we meet with a resistance which must be overcome.

If the spasm is slight, we should not continue the passive movements slowly and cautiously, but, on the contrary, we should grasp the extremity, either the hand or the foot, hold it for a moment motionless, and then suddenly and completely flex or extend the entire extremity. I think it important that the member to be tested should be grasped distally from its most peripheral chief joint; for we thus test all the muscles simultaneously, and all possible muscular spasms are sure to be evoked. The suddenness of passive movements, or, what is also advisable, the sudden repetition of a movement interrupted in about one-half of its excursion, stimulates the sensory muscle and cutaneous nerves, increases the tonus of the anterior horn cells, hence increases the possible slight muscular spasm, for all influences and irritations which increase or diminish the normal tonus have the same conspicuous action upon spastic muscles.

Extreme degrees of muscular spasm are characterized by the flexion of extremities or portions of the body during the most severe muscular spasms.

These extreme degrees of spasticity of the muscles are true contractures, i. e., ***active, muscular contractures,*** either because the anterior horn cells in the spinal cord are in a state of pathologically increased irritability and activity, or because the sensory irritations conveyed to the anterior horn cells are abnormally intense and continuous (reflex trismus in neuralgia of the fifth nerve), or because there is no central inhibition from the cerebral cortex (contracture in cerebral hemiplegia), or because power is constantly sent

from the cerebrum to the power station (hysterical contracture), or because, for any other reason, there is an immoderate discharge of power from the power station in the spinal cord, and this is so strong that the muscle constantly contracted produces a distinctly visible motor effect, i. e., is actively contracted. The genesis of contracture and the accentuation of its activity are therefore important, because the same coarse mechanical effect produced by active contracture may also be attained by other processes.

For instance, a muscle may gradually be so shortened by cicatricial contraction, by inflammatory processes, by the merely secondary contraction and retraction of its connective tissue, that a coarse motor effect is produced. Such a shortening, i. e., from retraction and contraction of the connective tissue contained therein, occurs in every muscle when the points of insertion are brought closer together, for example, during enforced rest of a joint or in consequence of disease of the joint.

Such a contraction develops in all muscles which are permanently in a hypertonic spastic condition. Hence, in addition to the originally purely *active* contracture there is, after a certain time, also a *passive* contracture which gradually controls the former until at last the originally active contracture is no longer apparent clinically, and only the passive contracture can be demonstrated. Similar to the passive contracture produced in permanently spastic muscles, which has been described, there is another passive contracture. This develops in the antagonists of these paralyzed muscles when, in any area of the body, only one definite muscle group, or an important locomotor muscle, is paralyzed or arrested in function. As, owing to the absence of opposing muscles, a certain hypertonia apparently exists in these antagonists, this passive contracture which develops closely resembles the one previously mentioned.

I have attempted to define clearly the nature of the spastic condition and contracture. I have already stated how a muscular spasm is clinically revealed. Now we must discuss the important and often difficult *problem* of the *differentiation of active and passive contracture.*

The first and chief difference between these forms is that, on attempting passive movements, active contracture shows itself by an elastic, but not quite uniform, resistance, while passive contracture is revealed by a uniform, constant and rigid resistance which resembles that noted on attempting to bend a piece of wire or lead pipe.

That a sudden, energetic attempt at passive movement will increase a spasm and the resulting active contracture, I have already stated. Passive contracture, on the other hand, is the same whether we begin passive movement cautiously and gradually or suddenly and energetically. Spasm, active contracture, shows great variations: Early in the morning after a quiet sleep and when the patient is otherwise rested, during and after a warm bath, when there is a general corporeal and mental feeling of well-being and contentment, active contracture diminishes in intensity. In the evening, however, and if the patient is tired, is cold, if he must make any mental or bodily exertion, if there is pain, active contracture becomes more marked. Passive contracture is in no way affected by these circumstances.

A capricious condition, rapidly changing, is therefore significant of active, the absolutely unchangeable and rigid one, of passive contracture.

Besides passive movement, we have other aids to the recognition of the nature of a contracture; first, the condition of the *tendon reflexes.*

The importance of testing the reflexes we shall later more minutely discuss. According to the scheme which I proposed for the examination of the muscle tonus we may readily comprehend that when there is a pathologically increased muscle tonus, or spasm, the tendon reflexes are increased. If, in a subsequent examination, we systematically test the reflexes, and find a very active tendon reflex or even clonus in an extremity which has resisted passive movement, this unquestionably indicates active muscular contracture.

It is also to be noted that an increase of reflexes actually present may be difficult to demonstrate because of old, long-standing active contractures. The secondary passive contractures which gradually result in these conditions fix the joints and the affected muscles in such a manner that reflex mechanism —at least in its motor portion—is hindered.

Like the tendon reflexes, the *cutaneous reflexes* serve to indicate the nature of the spasm. The cutaneous and periosteal reflexes are usually increased in active contracture and, as a rule, the toe reflex is pathologically changed.

In the discussion of spasm and contracture we have so far omitted a point of vital importance; namely, the influence of the *attention* of the patient in producing passive movement by muscular tension. When undertaking passive movements we usually tell the patient "to relax entirely" or "to let the member hang loose."

We rarely succeed with this request. As a rule—although *unconsciously* —the patient does exactly the contrary; the muscles to which his attention is attracted are only made more tense the more he attempts to relax them, and this may make a physician not skilled in these maneuvers believe that muscular spasms are present.

Another source of error springs from the fact that as soon as the physician attempts passive movements in an extremity the patient—again unconsciously —*actively* performs the same movements, and in spite of all urging seems unable to refrain from conjoint action without, on the other hand, relapsing into a state of muscular tension.

In all such cases we can only succeed by an *actual* deflection or diversion of his attention into other channels. Hence it is wiser to say nothing to the patient about relaxing and the like, to say nothing of the extremity that is to be tested; the patient should be engaged in interesting conversation which requires no effort on his part, no allusion being made to his disease. In the meantime, uniform passive movements should be attempted and, in order to further distract the attention of the patient, should be practised in *all* of the extremities. It is advisable to avoid a definite rhythm since a change of this rhythm is likely to attract the patient's attention.

If no satisfactory conclusions can be arrived at, we should undertake very thorough and powerful active movements, so as to set all parts of the extremity simultaneously into *activity*, and should then note whether the patient's efforts meet with resistance, whether this motion becomes suddenly slower or is even arrested, or—and this is a finer reaction—whether he complains of stiffness and a feeling of tension in any one joint.

If, notwithstanding these maneuvers, we reach no definite opinion—and

this is by no means rare—nothing remains but to reexamine the patient in the manner previously described.

When we have positively demonstrated active contracture, we have an important aid for all subsequent diagnostic conclusions. In all those cases of paresis or paralysis of the extremity in which spasm or contracture is simultaneously present so that the condition is designated *spastic* paralysis, the pathologic process is located *above*, i. e., centrally from the motor power station, and in some area of the brain or spinal cord the pyramidal tract must be damaged or disturbed. As we have seen, it sometimes happens that the power station is no longer regulated by the brain, but responds to the irritations brought to it from the periphery with a constant production of power.

When, however, the pathologic process is situated in the power station itself and destroys this, spastic paralysis becomes flaccid paralysis, since all the sources of power are destroyed.

There is, however, as is evident from the scheme proposed, *one* possibility in which, in spite of disease of the power station in the anterior horns, *spastic* paresis develops. This condition arises, in the first place, if the motor anterior horn cells are *only in part* destroyed, and, secondly, if the pyramidal tract above this area is simultaneously damaged. In this condition—which we see in *amyotrophic lateral sclerosis*—the damage to the pyramidal tract as well as atrophy of the muscles in which a number of anterior horn cells are destroyed, produces paresis. The simultaneous spasm is due to the fact that, owing to the absence of the "inhibitive fibers" of the damaged pyramidal tract, the cells still existing in the power station continuously produce force which is transmitted to the muscles.

Spasms and active contractures of the extremities will be found in tumors, hemorrhages, sclerosis, etc., in which the processes directly or indirectly act upon the *motor zone of the brain* or the intracerebral course of the *pyramidal tract*. In these cases the monoplegic or hemiplegic type will predominate. In damage of the *pyramidal tract* within its *spinal* course, the paraplegic spasm will predominate on account of the small anatomical area involved. It is not surprising that actual spasms and contractures should occur in all functional diseases in which we assume a pathologic action of the cerebral cortex—therefore in hysteria and certain psychoses.

Those active spasms and contractures which appear as persistent, *abnormal*, peripheral *sensations* and *pains* in the implicated region or in the region of the paresthesia have been touched upon previously in discussing trismus, which sometimes accompanies neuralgia of the fifth nerve. In the same group of active contractures belong the spasm of the lids in affections of the conjunctivæ, the contracture of the erector trunci in certain cases of pain in the back, the contracture of certain muscles of the back in sciatica, probably also the so-called Ehret's contracture the distinguishing feature of which is that while walking there is constant pain in the foot, and this gradually produces a contracture of the tibialis anticus.

Trismus in neuralgia of the fifth nerve has been attributed to a constant discharge of force from the motor power station (in this case the motor nucleus of the fifth nerve) and is produced by the excessive irritation of the severe pain in the sensory branch of the fifth nerve. For some reasons, however, it seems likely—this is particularly true of the similar spasms just mentioned

—that it may be due to other influences on the part of the cerebral cortex which also produce force (unconscious voluntary innervation).

Spasms in *tetanus* must be regarded as active, and most likely due to direct toxic influences upon the anterior horn cells.

Contractures in the flexors of the leg, frequently seen in *meningitis* (Kernig's sign), are peculiar in that they appear only while the patient is in the sitting posture, and disappear when in the recumbent position. These contractions also are active, but we are still ignorant of their origin.

We note a *special* and peculiar form of active muscular contraction in *paralysis agitans*. In this disease the muscles of the nape of the neck, of the arms, and of the hands are always abnormally hard and tense.

Although the tendon reflexes—not the cutaneous—are frequently increased in paralysis agitans, the tension of the muscles is unquestionably of very different nature, being less elastic, and more moderate than in spastic hypertonia.

Hypotonia of the muscles, the opposite of hypertonia, is less difficult to differentiate.

Hypotonia, as the name indicates, consists in a decrease of the normal muscle tonus. A smaller current of power is discharged from the spinal power station than under normal circumstances. From a purely theoretic consideration, a decrease of normal tonus may be produced in as many various ways as increase of tonus. In the genesis of hypotonia, the conditions now to be named frequently come under consideration: Absence or defective nature of the sensory irritations transmitted by the posterior roots to the spinal cord (tabes, other processes in the posterior roots); diseases of the power station itself and defective supply to the motor nerves (certain spinal and neuritic muscular atrophies and "flaccid" paralysis), as well as shock of the spinal cord, and diseases of the cerebrum and cerebellum (some forms of porencephalia and other extensive cortical and cerebellar diseases). In passive movements hypotonia is revealed by the fact that the joints permit a great latitude of abnormal movements; for example, the forearm may be passively over-extended, the legs may be drawn apart almost to an angle of 180°, in flexion of the lower limb the heel may be placed against the tuberosity of the ischium, the knee may be over-extended, and the lower extremity extended at the knee may be easily and painlessly flexed to a position beyond the vertical. Before investigating the abnormal excursions of the joints, it is sometimes well to remember that on attempting passive movements an absence of resistance will be conspicuous, and while in the normal person we must suggest relaxation, in the patient with hypotonia the individual members—apparently only subject to weight and without organic connection with the whole—readily become flaccid.

In the legs hypotonia may be easily recognized, so that the condition of the tendon reflexes—these are absent or greatly diminished—is no longer necessary for the diagnosis.

Only after we have exactly informed ourselves as to the passive movability should *active movements* be tested. First, we should ascertain the gross power which the individual muscles and muscle groups are able to exert. For this purpose we should naturally be more or less familiar with the special physiology of the muscles, and here the classic work of Duchenne is still an authority.

The practical investigations of the chief muscles of the body is best made by beginning with the *cranial nerves* and the *head.*

The patient is asked to rotate the eyes upward and downward, to the left and to the right. The movements of the eyes must be synergistic. The abducens muscle should bring the border of the iris to the external angle of the eye. But if the bilateral movement of the abducens should appear to be insufficient, we must be cautious in assuming therefore a bilateral paresis of the abducens, since fretful and stupid patients, or sometimes those with a general neurosis, are from mental weakness incapable of rotating their eyes outwardly to the physiologic extreme.

Slight paresis of the muscles of the eye, especially of both oblique muscles, readily escapes detection by this method of examination, and is more readily recognized by examining for double vision.

We must, of course, strictly differentiate between a strabismus due to paralysis of the muscles of the eye and a non-paralytic one, so-called concomitant strabismus. Strabismus may arise, too, from active spasm of individual muscles of the eye (meningitic processes, hysteria).

It must be observed that an incomplete movement of the eye upward is sometimes only a seeming one, and is due to a slight drooping of the upper eyelid (ptosis).

The movements of the *lids* may be studied while examining the muscles of the eye. On raising and lowering the plane, the upper eyelid must simultaneously and uniformly follow the movement of the eye so that no white streak of sclera is ever visible between the iris and the border of the lid. If the upper lid does not follow uniformly but spasmodically, we have the so-called "Graefe's sign" which is due to a disturbance of the sympathetic innervation of the smooth muscles of the orbit, and occurs in Graves' disease without being absolutely pathognomonic of this condition. Drooping of the upper lid (ptosis) and inability to raise it sufficiently depends upon disturbance of the levator palpebræ which is supplied by the oculomotor nerve. Partial paralysis of the levator palpebræ is functionally corrected by the contraction of the frontal muscle. Hence, in doubtful cases in testing the muscle which raises the lid the action of the frontalis must be excluded by fixing the skin of the forehead with the finger.

The impossibility of closing the eyelids or of lessening their abnormal gaping is a sign of paralysis of the upper branches of the facial nerve. Spasmodic conditions in the muscles of the lids, as in the muscles of the eye, are rare and occur chiefly in hysteria. Tremor and convulsive movements of the lower eyelid on attempting to close the lids is usually seen in neurasthenia or other neuroses. In the normal person the eyelids may be so tightly closed by the orbicularis muscle which is supplied by the facial nerve that the closure can hardly be overcome by the investigator.

The other muscles supplied by the facial nerve should now be tested; the patient is asked to wrinkle the forehead (often this is only possible by a synergistic movement in looking upward), to move the nose, to open the mouth wide, to close it firmly, to pucker the lips, to move the angle of the mouth to the right and to the left.

To ascertain if there is weakness of the lower branches of the facial nerve, those supplying the mouth, we closely examine the branch supplying the

forehead, for the recognition of paresis of the lower facial branches often leads us to a diagnosis which would otherwise remain obscure. This depends chiefly upon the fact that in various cerebral and intracranial processes there is much more frequently a paresis of the lower branches of the facial nerve than a paresis of the upper branch. The latter is less likely to be functionally involved, because, unlike the branch supplying the mouth, it is innervated from both hemispheres of the brain.

Inability to retract the angle of the mouth, due to facial paralysis, often appears more distinctly in certain conjoined movements than in merely opening the mouth; for instance, when the patient makes firm pressure with the hand, or shows the tongue, etc. Care must be exercised to exclude asymmetry of the mouth—which is often congenital or may be due to defective teeth—so that this be not ascribed to a disturbance of innervation.

The angle of the mouth and both sides of the face should be observed during lively speech, and especially during *laughing*. The laughing must be spontaneous; if the patient is told—as is often unwisely done—to laugh, the movement is in most cases purely *voluntary* and a mere imitation of emotional movement. The observation of voluntary emotional (affective) movements in laughing and crying is therefore important because, in the first place, indistinct and scarcely noticeable facial paresis with varied localization becomes most distinct upon affective movements. Here the weakness of one facial nerve often shows itself not so much by the total absence or the quantitative diminution of its motor effect as by the fact that the contraction of the paretic side appears after a preliminary stage of rest, and after there has been a fully developed contraction in the normal state at the conclusion or at the height of the expression of emotion, that, therefore, when both sides of the face contract, the contraction of the diseased side occurs later (normally it should occur at the same time).

The testing of affective movements is also important because certain conclusions as to localization may be drawn from the irregular action of the mouth in voluntary and in emotional movements. The motor innervation of affective movement is most likely conveyed from the thalamus region or from the basal ganglia by a path independent of the tract for voluntary motion. This explains why in some cases voluntary innervation of the angle of the mouth may be preserved and the affective movement be absent or only slight (foci in the region of the thalamus), and, inversely, why there are cases in which voluntary movements are absent but the emotional are retained (certain cases of cerebral hemiplegia, infantile pseudo-bulbar paralysis).

In an examination of the movements dependent upon the facial nerve, that of the *tongue and palate* must be included.

The tongue must lie still in the mouth, and when at rest must not be elevated nor deviate to either side. When the tongue is protruded this should be done quickly and without tremor, and it should be extended so far that the tip will reach at least to the anterior border of the lips, while the raphé of the tongue corresponds to the median line of the chin. The middle of the chin must be marked with the finger while the tongue is protruded. Notwithstanding the absence of actual paresis, some patients, especially hysterics, do not protrude the tongue beyond the edge of the teeth, which condition is

due to psychical influences, and analogous to the imperfect lateral movements of the eyes.

The patient should be able to move the tongue rapidly to and fro in the mouth, to insert it in the spaces between the alveolar processes and the mucous membrane, to turn the tip of the tongue upward and downward, and to roll it laterally.

In unilateral *paresis* of the *hypoglossal nerve* the tongue during rest deviates to the side of the non-paralyzed muscle, as the weight of this muscle overbalances the weight of the paralyzed one. On protruding it, however, the unilaterally paralyzed tongue deviates to the side of the paralysis, because the normal muscle, which overbalances, mechanically turns to the side where there is least resistance, therefore toward the flaccid paretic half of the tongue—and thereby draws the entire anterior half of the tongue with it. The mechanical conditions in contracture of one-half of the tongue are are in the main the same. Here the normal half of the contracted organ is opposite the side mechanically least resistant, therefore the tongue when protruded deviates to the non-contracted side. With simple paresis of the hypoglossal nerve—no matter of what character—the deviation of the tongue may be differentiated from the deviation in the last mentioned hemispasm of the tongue: First, because the entire tongue never deviates in paralysis of the hypoglossal nerve but only the tip, and, moreover, it is never so pronounced and extreme as in hemispasm. In the latter condition we are reminded of the voluntary oblique protrusion of the tongue in grimaces; for in this exaggerated way the tongue is often protruded.

The tongue which protrudes and deviates in consequence of paresis may be readily returned to a normal median position; a spastic deviation, however, cannot be corrected, but shows great elastic resistance to the finger. Touching the protruded tongue, which we are often tempted to do, is apt to leave us in doubt; for it is sometimes very difficult to determine from the protruded tongue whether that half which has deviated has the normal tonus or is abnormally flaccid. The decision of this question is greatly facilitated if atrophic conditions are simultaneously present. If both halves of the tongue are *atrophic*, the entire organ appears shrunken, often as if dried up, the impression of the teeth is seen on the edges, also small but deep fissures and longitudinal grooves. If the atrophy is unilateral, only one-half of the tongue has this appearance. The fissures due to atrophy may readily be differentiated from the *cicatrices* found on the edges of the tongue in epileptics. These cicatrices should be carefully looked for in all patients with spasms. If found on only one-half of the tongue, they may indicate the unilateral nature of the spasms.

Unilateral paralysis of the tongue does not interfere with the patient's eating, and less with his speech than might be supposed—much less than unilateral peripheral facial paralysis. Of course, there is always a purely subjective limitation in unilateral paresis of the tongue. Bilateral paralysis of the tongue naturally causes great disturbance.

The *soft palate* should be inspected simultaneously with the tongue. Normally both arches of the palate are equally curved, are equally elevated during rest, and are uniform. The position of the uvula is of comparatively little importance. As a rule, the palate cannot be moved voluntarily. In an exami-

nation of patients, therefore, we must cause them to make a movement which synergistically raises the arches of the palate. Phonation is suitable for this purpose, for example, they may utter the vowel *a*. If in doing so one side of the palate moves less than the other or remains completely immotile, or if the palate moves obliquely to one side instead of medially and vertically, there is unilateral paresis of the palate—provided no cicatrices or adhesions can be found. Unilateral contractures of the palate are extremely rare. In bilateral paralysis the entire structure of the soft palate during phonation remains completely immotile, or does not extend to the posterior wall of the pharynx.

The movability of the soft palate is also tested by producing the so-called palate reflex. This mode of examination for various reasons is not adapted to motor testing, as I shall point out later when describing the reflexes. A voluntary act of deglutition on the part of the patient is better; normally, during this act, the pharyngeal cavity is completely closed by a close approximation of the soft palate to the posterior wall of the pharynx. Regurgitation of fluid through the nose on voluntary swallowing is proof of the inadequacy of this closure and of paresis of the soft palate.

As several nerves are concerned in the innervation of the soft palate, namely, the pneumogastric, the sympathetic, the glosso-pharyngeal and the facial (the last by a branch given off in the geniculate ganglion which passes into the spheno-palatine ganglion and terminates in the palatine nerve), the explanation of paresis of the palate is not always easy. The difficulty is enhanced when, owing to the implication of individual nerves which supply the velum of the palate, considerable individual variations appear.

Paralysis and paresis of the tongue and soft palate, like paralysis in the course of any other cerebral nerve, may be due to disturbances in any point of the motor tract between the cortex of the cerebrum and the muscle. From the many possibilities and clinical pictures which result, I should like to emphasize one especially typical in which the lips, the pharynx, and the tongue are simultaneously and bilaterally paralyzed or paretic. This is the picture of so-called *bulbar paralysis* or paralysis glosso-pharyngo-labialis. In this symptom-complex the pathologic focus is situated in the motor power station itself, i. e., in the nuclear structures of the bulbus, which are analogous to the anterior horn cells. The seat of the focus in this nuclear region therefore produces, what I have previously described in the anterior horn cells, a degenerative atrophy of the muscles of the lips and tongue, but this characteristic condition is even intensified in so-called bulbar paralysis.

After investigating the movability of the palate, we turn to the muscles of *mastication*. The muscles which here come into action, the temporal, masseter and both pterygoid muscles, are supplied by the motor branch of the fifth nerve, and are tested by telling the patient to press the lower jaw tightly against the upper while the physician simultaneously attempts to prevent this movement. Normally the jaws lock together extremely tight, and cannot be moved by the investigator either to the right or left. With a slighter development of power, while the jaws are firmly closed, we may determine by palpation of the masseter muscle between the external angle of the eye and angle of the jaw and also of the temporal muscle in the temporal groove whether one alone or both muscles are paretic. The chief function of the

pterygoid muscles is to move the lower jaw laterally and this function can be readily tested by a lateral movement against resistance. As a rule, the muscles of mastication play no great rôle in pathology.

In the examination of patients with organic nervous diseases the investigation of the *muscles of the larynx* is more important. Voluntary phonation and inspiration, the closure and opening of the glottis, are observed with the laryngoscope. If this is always done in suspected cases, a decrease or entire absence of motility in one vocal cord may be demonstrated in cases where such a condition has been unsuspected. We may find either a median position (as the expression of paralysis of the posterior cricoarytenoid muscle, i. e., the initial stages of a paralysis of the recurrent laryngeal either of central or peripheral origin) or a cadaveric position of the vocal cord (the expression of total paralysis of the recurrent laryngeal nerve). The foregoing enables us to understand why paralysis of the posterior cricoarytenoid muscle should occur relatively often at the onset of many organic nervous diseases.

We now come to the *act of speech.* Disturbances of speech are extraordinarily important, and occupy a prominent place in neurologic symptomatology. The most significant features of central speech disturbance have been described in a special article in this volume by the most eminent authority upon speech disturbances, Wernicke. His description indicates the signs by which we determine the existence of an internal central disturbance of speech, and also the particular form of the aphasia. I shall, therefore, here limit myself to a few suggestions regarding the differentiation of the most important "*speech disturbances*" of non-central nature. Speech disturbances due to anatomical lesions or to functional impairment of the muscles, nerves and nerve nuclei of the medulla oblongata, which are called into action by speech, are included under the designation "*dysarthria.*" Most of these disturbances which are now to be sketched will become obvious in an examination of the patient without an especial test.

If, because the pharyngeal space is imperfectly closed in phonation (paralysis of the soft palate), some of the expiratory air passes out through the nose, the speech has a peculiar nasal twang. If there is paralysis of the muscles of the tongue, the lips, and the cheeks as well as of the soft palate, the speech will be nasal and very indistinct (bulbar paralysis). In peripheral disturbances of the organs of speech the nasal factor may not be so prominent, but the speech is often thick, heavy, indistinct, and slow because the production of correct sounds has made the numerous fine distinctions in the contractions of the muscle difficult or impossible.

In all dysarthritic *speech disturbances*, especial attention must be given to faulty formation of the voice and a faulty mode of speaking. Often on attempting to speak the voice is uncertain, or on persisting it becomes irregular, vibrating and trembling. To determine the nature of dysarthritic speech disturbances, it is well to have the patient intonate clear vowel sounds like *a* and *e* and to prolong them for a short time.

If the disordered speech is not due to the difficulty of enunciating sounds (stammering), but because the union of syllables (which is normally quiet and uniform) is interrupted by spasms in the muscles and organs of speech, we are dealing with stuttering. *Stuttering* is merely a functional disturbance of speech, its cause probably originating in the cerebrum. In stuttering all

of the muscles of the face may be contracted simultaneously with the muscles of the tongue and the mouth, particularly when the patient makes an effort to speak. If he is alone or does not raise his voice but merely whispers, stuttering will often cease. A disorder of speech which resembles stuttering manifests itself by an interruption in the sequence of syllables, so that the patient in beginning a word or phrase always halts at a certain letter or syllable, and repeating this several times, holds on to it until he is able to pronounce the following syllable. This kind of speech often appears to be due to affectation or design, and is noted in *hysteria,* particularly that form of traumatic origin. Among the disturbances of speech common in hysteria the most important are *aphonia, egophony* and the *falsetto voice.* All three are disturbances in the production of voice. In aphonia, during voluntary speech the glottis cannot be closed; the patient can only whisper. During paroxysms of cough in these patients, the rima glottidis closes and an audible sound arises; in the same way hysterical patients with aphonia sometimes speak naturally when excited, or when they speak unconsciously and involuntarily. Egophony (the bleating of the goat) is the production of a peculiar hoarse, bleating and suppressed kind of voice; the peculiarities of the falsetto voice are generally known and need no description. (See article, "Disturbances of Speech" by H. Gutzmann, in this volume.)

Among the most common disturbances of speech is the so-called *scanning* speech which is observed in multiple sclerosis. In this form of dysarthria the patient divides his words into syllables, but dwells upon one syllable until he can produce another. As the power of modulation generally suffers simultaneously, and the patient speaks very slowly, speech becomes somewhat monotonous and labored.

Before leaving disorders of speech I must call attention to a very characteristic disturbance which, in the main, belongs to the disturbances of internal speech. As, however, in every case of disturbed speech, there are usually also dysarthritic affections, I must state that this speech disturbance, "*syllable stumbling,*" is characteristic of progressive paralysis. We test this by having the patient repeat a long word which is difficult to articulate, for example, "Konstantinopolitanisch." If this affection exists the patient will repeat it about as follows: Konstantino—Konstanto—poli—tanisch—konstanti—nopel—politsch—tanisch, or the like. If we listen closely, in the faulty repetitions of this dyslogia and dysphagia we will note dysarthritic difficulties also in the production and formation of individual sounds and letters. Among similar examples for practice are the following: "Around the rugged rock the ragged rascal ran"; "Peter Piper picked a peck of pickled peppers"; "A big black bug bit a big black bear." In paralysis the speech generally becomes more distinct the more frequently the word is repeated.

Another cranial nerve, the spinal accessory, and, to a small extent, the cervical nerves, innervate the trapezius and sternocleidomastoid muscles, the most important *movers of the head and the cervical vertebral column.* Lesser movements, particularly in the joint between the occipital bone and the atlas, are produced by the small muscles of the neck. Resistance is generally limited to the forward and backward movements of the head and cervical vertebral column and to lateral movements and turning of the head. Normally any of these movements may be performed so vigorously by an adult that the

physician cannot prevent them. The same is perhaps true to an even greater extent of raising the *shoulder*, which is also done by the action of the trapezius muscle.

In testing the function of the *muscles of the back* the most important movements are the following: Adduction of the shoulder-blades to the median line (action of the rhomboideus and of certain portions of the trapezius), retraction of the shoulder (latissimus dorsi), as well as flexion and extension of the back and of the vertebral column at various heights (abdominal muscles, ilio-psoas, erector trunci, quadratus lumborum and others), and turning of the vertebral column (semispinalis dorsi and the small muscles of the back).

After these functions have been tested, the general motility must be investigated, particularly the coarse power of the extremities. We begin with the *upper extremity.* First, with equal rapidity, the arms should be raised perpendicularly, and we should note whether both arms simultaneously reach the vertical plane, whether in this movement the slight flexure of the forearm toward the upper arm is equally marked on both sides, which is normal, and whether in raising the arms to the vertical position the extension of the hand and fingers is bilaterally uniform. For detecting slight debility in the musculature of the arm which is not revealed by coarse tests owing to the resistance which will be described later, we have a fine reagent in the previously mentioned test: The slightly paretic arm on rapid raising reaches the vertical plane later, the forearm is flexed less tensely upon the upper arm, and the hand and fingers are less strongly extended against the forearm, than is the case upon the normal side.

The chief resistant movements are the following: We should attempt forcibly to lower the extended arm of the patient. Next, when the arm is extended laterally, we should try to draw it toward the middle and, *vice versa,* when the arms are stretched posteriorly and downward we make an effort to move them from this position. Finally, the arms which are extended laterally from the thorax at an angle of about 45° are to be forcibly lowered while at the same time the physician attempts with his hands to resist this movement.

As already indicated, slight weakness, particularly on movements of the shoulder-joint, may escape this test by resistance; probably because the amount of power developed in the shoulder-joint is extraordinarily great.

The movements of the elbow-joint are examined to see with what strength the forearm can be flexed and extended, the physician trying to prevent the movement. In conditions of debility, in flexion of the forearm the supinator longus may be tested alone by having the pronated forearm flexed, and the biceps alone may be tested by having the supinated forearm flexed. Pronation and supination of the forearm may very easily be examined by producing the corresponding resistant movements.

The flexors and extensors of the hand and fingers and the muscles of the hand are tested by having the patient squeeze the hand of the examiner as tightly as possible. Squeezing the hand is at the same time a good test of the strength of the muscles of the upper arm and for some of those of the shoulder and back; on tightly closing the hand all of these muscles become tense when the arm is fixed.

Of the action of the extensors of the hand, we can easily convince ourselves

by making a fist. In making a fist each extension of the root of the hand results in stretching as far remote from one another as possible the points of insertion of all the muscles which flex the hand and the fingers. It is easy to see that this increases the dynamic action of the flexor muscles. In radial paralysis pathology furnishes a proof of the correctness of these statements, for, in this condition, the fist cannot be tightly closed.

A number of instruments have been invented (the best representative being the *spring dynamometer*) to test the grasp of the hand. As a rule, all of these instruments may be dispensed with.

Special examination of the muscles of the hand is only necessary when symptoms point to the hand, or when we suspect certain diseases of the spinal cord in which experience proves the muscles of the hand to be usually implicated.

The power to separate the fingers (the interossei, extensor digitorum communis) and then to restore them to the normal position (interossei) is tested by placing the volar surface of the patient's hand and entire forearm upon a flat table after which the investigator places his hand with slight pressure upon the back of the patient's hand. Normally, in spite of the pressure of the overlying hand, the separation and adduction of the fingers will be quite free and rapid, and will be done by a single movement. The adduction and opposition of the thumb will normally be so great that the investigator can scarcely overcome it. The completeness of opposition can be tested in another way, the patient being told to touch the tips of all the other fingers with the tip of the thumb extended in all of its joints. In this test the distal joints of the fingers that are to be touched must be extended, only the metacarpal phalangeal joints being flexed.

This movement can only be performed in normal breadth and with the normal power, which can hardly be overcome by the investigator, when the opposition muscles as well as the interossei functionate normally.

If we suspect slight debility, the function of the muscles of the forearm and hand may be tested by having the patient rapidly and alternately flex and extend the fingers as, for instance, in piano playing. Even slight differences between the normal and diseased extremity become noticeable. Notwithstanding the fact that the right arm is normally somewhat stronger than the left, these movements as in piano playing are normally made with the same power by both the right and left hands.

An irregular tremor of the forearm which appears upon powerfully grasping with the hand suggests a slight degree of debility which is not detected by resisting movements.

Sometimes it is advisable to extend both arms either perpendicularly or horizontally or at the side, and to observe whether the arms simultaneously become weak, or whether this may vary.

In some diseases it is necessary to note whether *the strength gives out.* Thus in many functional states of debility, so-called *adynamia*, the first putting forth of a strength in movement may be very good, but it soon gives out, and the terminal mechanical effect is very slight. A similar condition will occasionally be observed in so-called *myasthenic* diseases. Here the original and marked muscular power, which is sufficient for the performance of the first movements, becomes slighter on each repetition.

If, in these cases, the arms of the patient are vertically raised, several times in rapid succession without permitting a pause, the arm can the first time be raised to the vertical, the second to a somewhat lower position, and after a few repetitions—perhaps the tenth—can be moved only very slightly from the wall of the thorax.

This phenomenon, however, can only be designated "myasthenic" provided there are other signs (electrical) of the myasthenic pathologic picture, for similar but less conspicuous diminution of strength is occasionally seen in neurasthenia and hysteria.

The last stage in testing gross muscular power is the *examination of the legs.* Here it is best to have the patient flat upon his back. At first the leg, extended at the knee, is to be raised with all the might, while the physician makes strong pressure downward at about the middle of the thigh. The leg is then powerfully abducted and adducted, the physician each time making powerful resistance. The extension of the thigh against the pelvis can best be tested in the lateral position. The power of the chief flexor and extensor of the pelvis upon the thigh (the ilio-psoas and the gluteus magnus) may also be tested inversely with the leg fixed; for example, while the patient is standing erect or in the recumbent posture with the leg fixed, the pelvis is bent forward and extended, or the patient is asked to rise from the dorsal decubitus in spite of resistance and then to lie down.

Flexion and extension of the knee-joint may be tested in the recumbent posture or, still better, while the patient is lying upon his abdomen. The power of flexion and extension of the foot is tested by telling the patient to move his feet dorsally or in the plantar plane with all his might. It is advisable for the physician to illustrate these movements with his own foot or to perform them passively with the patient's foot. When the foot is flexed dorsally, the physician uses his whole strength and attempts to draw down the patient's foot. If the flexors of the foot have normal power this can always be done. The plantar muscles of the foot (as well as all the other muscles of a normally vigorous lower extremity) are remarkably strong. The only movement in the lower extremity of the normal adult which the physician can counteract is the flexion of the knee.

As a rule, such an examination as sketched above is sufficient for the muscles of the lower extremity. For special examination, the necessary points are indicated by the physiology of the muscles.

It is well to bear in mind that when the ilio-psoas flexes the thigh against the pelvis it at the same time rotates the leg somewhat outwardly while the tensor fasciæ latæ simultaneously turns it a little inward; that the tibialis anticus bends the foot dorsally, and at the same time elevates the internal border of the foot, while the extensor digitorum communis simultaneously with the dorsal flexion of the foot raises the external border of the foot. In a minute examination of the muscles of the leg we must always remember that the muscles upon the posterior surface of the thigh pass over two joints and, like some of the muscles running from the pelvis upon the extensor surface of the limb—*passing through* the thigh—extend to the leg.

The *movements of the toes,* as a rule, can only be tested by a simultaneous and voluntary innervation of all the toes. In hysterics I have occasionally found the curious faculty of moving the toes singly and separately from each

other, like the fingers. There is normally no difference between the power of the right and left legs.

In the legs, just as in the arms, we can sometimes determine a slight degree of weakness with resistant movements from the facts that when certain motions, such as flexion and extension of the knee-joint, are rapidly repeated, or both legs are alternately extended, tremor may appear, or dropping of the leg is observed prematurely; when the patient is directed to put first one and then the other leg upon a chair without aiding himself with his hands, if one leg is slightly paretic the patient is obliged to support it in putting it upon the chair, or perhaps some other uncertainty or a difference between the legs may become apparent. The gait often reveals a slight weakness in one or both legs.

Examination by means of resistant movements has the disadvantage that it is dependent upon the coöperation or, rather, upon the *psychical condition of the patient.* If, in the examination, a motor condition of debility appears to depend upon the psychical element, this circumstance, in all probability, contraindicates the organic nature of the paralysis. If a voluntary weakness of function is brought about by the patient, i. e., by simulation, the debility is of hysterical, hypochondriacal, or neurasthenic nature. As the most common variety of this paralysis I must mention hysterical *astasia-abasia* and *general,* hypochondriacal, neurasthenic *adynamia.* The psychical nature of these affections is especially revealed by the fact that the muscles intended for certain movements which the patient deems important (for instance, walking, pressure of the hand, etc.), are paralyzed, or at least paretic, yet are able to perform their functions as soon as the final resulting mechanical or locomotor effect is different from that which the patient has inseparably associated with the actual function of the muscles affected. This will likewise explain the fact that the shoulder and elbow movements are often quite strong in neurasthenics yet the pressure of the hands is very feeble, and, *vice versa,* that in the lower extremities the knee and hip movements may be weak while flexion and extension of the foot are very energetic: Therefore, the movement which the patient believes to be the most important is always the weakest.

Pareses and paralyses of *organic* nature are much less dependent upon the influence of the psychical condition, although not entirely independent of this. Debility due to organic defects follows interruptions to and damage of the motor tract from the cerebral cortex to the muscle. A detailed description of the many peculiarities of organic paresis is not within the scope of this article. I shall only mention that hemiplegic organic paresis or paralysis signifies a pathologic seat in the brain. Paraplegia (paralysis of both arms or of both legs or of both arms and legs) contraindicates that the seat of the disease is in the brain, and favors its situation in the spinal cord or in the peripheral nerves. A few of the fundamental laws for the topical differential diagnosis of organic paralysis were mentioned in the description of spastic muscular conditions. Here I must reiterate that organic as well as functional paralysis may be of spastic or flaccid type.

THE EXAMINATION OF THE COÖRDINATION OF MUSCULAR MOVEMENTS

(Coördination of muscular movements, ataxia, static ataxia, test of ataxia in the arms and legs, the erect posture, various degrees of swaying with closed eyes and with feet in juxtaposition, swaying in organic ataxia and in functional affections, swaying in amaurosis with closed eyes, swaying of patients with genu varum, observation of the gait, inclination of the pelvis, pelvic movements, paretic gait, equine (steppage) gait, spastic gait, spastic-paretic gait, ataxic varieties of gait, determination of these, waddling gait, the gait in multiple sclerosis, cerebellar gait, dragging of a leg, intermittent claudication, arrhythmic and "sweeping" gait in hysteria, influence on the gait of using a cane.)

Even though we have determined by resistant movements that the individual muscles are of normal power, nevertheless voluntary motions may be markedly impeded, because in all voluntary movements the action of several muscles is necessary. It is evident that in all muscles which perform a definite movement by their simultaneous contraction the various degrees of contraction must bear a certain relation to each other. If one of the muscles whose action is necessary for a certain movement contracts at the wrong time, or too much or too little, i. e., too early or too late, too violently or too feebly, the simultaneous contraction of all the other muscles will not produce the desired result, for the general activity depends upon the normal function of all muscles of the group. A lack of harmony in a common functioning muscle group may be due to the fact that one muscle of the group is paretic or paralytic, or that spastic conditions exist in one or several muscles, etc. This purely mechanical disturbance of coördination we shall not now discuss further. On the contrary, we turn to a form of incoördination—called ataxia—due to a faulty or insufficient activity and disposition of each muscle of the group which has a function to perform, as well as to a faulty control and regulation of the necessary degree of contraction in the muscle group. Here the existing disturbance—diagrammatically presented—arises because the relations and processes in the periphery (ataxia in many affections of the spinal cord, particularly in tabes dorsalis) are imperfectly transmitted to the subordinate, so-called coördination, centers in the brain, the trunk of the brain, and the medulla; or it may arise because the coördination centers themselves are diseased (diseases of the cerebellum, certain affections of the motor cortical region); or, finally, because the motor nerves are not intact (certain forms of neuritis [so-called ataxic neuritis without demonstrable sensory disturbance] in diphtheria, etc.).

The limits of our theme do not permit me to give a diagrammatic sketch of ataxia, particularly as the most important questions concerning it have not yet been cleared up. But one particular form of ataxia, *static ataxia* (in contrast to movement ataxia), I must mention. By static ataxia I mean a disturbance in the coördination of those contracted muscle groups, chiefly tonic, which, for instance, are particularly active in standing erect, in sitting, in maintaining the equilibrium of the body, in raising or extending a member, etc.

From the foregoing it is evident that in the examination of our patients —although ataxia may occur in almost any muscle groups—much depends on

whether the arms or legs are ataxic, and whether the equilibrium of the body can be preserved in all positions.

In testing ataxia of the extremities the most suitable movements are those which require the conjoint action of the greatest number of individual muscles in order to perform a minimal movement. The finer and more complicated the movement, and the greater the number of muscles called into action, the more sensitive is the reaction.

Therefore the patient should be directed to touch with the tip of his index finger certain parts of his face (the tip of the nose, the lobe of the ear at either side, the eyes, certain teeth, etc.); or he is asked to point rapidly with a lead-pencil to certain spots upon a paper, to thread a needle, and the like; or food and drink are to be carried to the mouth, or he is asked to button and unbutton a garment, to write, to play the piano, to pick up with his fingertips a needle which lies on a smooth surface—the ataxic will use the volar surface of the end of the finger or even the middle joint—etc. A method less fine which is in common use, is to bring together the tips of the two index fingers; sometimes it is advisable to flex the individual joints of the arms and fingers slowly and uniformly, and then to extend them. If these movements are not performed evenly and harmoniously, if they are clumsy, paroxysmal and ununiform, or are waddling, ataxia is present—provided there is no paresis or spasm. Sometimes ataxia is most obvious when the patient performs the desired movement rapidly, sometimes when it is very slowly done. If we are in doubt concerning the existence of ataxia—as will often be the case—or if we have special cause to suspect it, the patient should not be allowed to control the movements by the use of his eyes.

Ataxia in the upper extremity is markedly increased on closing the eyes. The slight ataxia at first present may show a diminution on subsequent attempts. This is especially true in slightly complicated movements, for example, in bringing together both the index fingers and the legs, and still more so in the tests for ataxia of the legs which will later be described.

Better than any of these methods, at all events less complicated and tedious, is the observation of the trivial actions and movements of the patient. The way in which he takes off his coat, puts his fingertips on the inside and his thumb on the outside of the front of his coat, loosens the collar and buttons it, how he unfastens his waistcoat, whether on donning the coat he tries to adjust his cuffs or the sleeves of his shirt, etc. These observations, if accurate, will tell us, even before beginning the actual investigation, whether or not the patient has ataxia of the hands.

In the lower extremity it is best to test for ataxia with the patient in the horizontal position. One of the best known tests is to have the patient place one heel upon the knee of the other leg or upon the great toe of the other foot; he should then be asked to touch with the tip of his foot anything that is held out toward him, to describe in the air a certain figure, an 8 or the like, or to raise slowly and uniformly the leg extended at the knee-joint, and in like manner to let it fall. If there is an ataxic disturbance of the leg, in the horizontal position this becomes evident as soon as the patient closes his eyes.

Simple as these tests appear to be, they will rarely leave us in doubt as to whether or not actual ataxia is present. This is partly due to the fact that

stupid and clumsy patients are incapable of understanding or accurately performing complicated movements. On the other hand, in slight ataxia—provided the patient is intelligent—the repetition of a simple tactile test which has been practised only a few times will often cause ataxia of the legs, for the moment at least, seemingly to disappear, particularly that produced by one special movement.

As a rule, we need not be particular to test the legs, but may rest content with the test of the heels and toes provided this has been correctly carried out two or three times. Naturally, other and easier movements will be correct. One of the most difficult tests is the simple, slow, but uniform elevation and lowering of the leg when extended at the knee-joint.

A test of the coördination when *standing* and *walking* leads us to the description of both of these functions. Not only must the coördinative faculty be investigated, but passive motion and the development of coarse power is complemented by observation while *standing erect*, particularly on walking. Hence we now follow the same method of examination with complicated movements while walking and standing, which can only be normal when passive motion, power and coördination are also normal.

The investigations of physiologists have taught us that on standing erect the position of the legs is not fixed by gravity but by muscular tension. If, therefore, the muscles are decidedly debilitated or incoördination exists, normal standing is impossible during their uniform contraction—static ataxia.

A good test of the patient's ability to preserve the equilibrium of the body while motionless and standing erect is to have him place both feet close together at the heels and toes, and then shut his eyes. This lessens to a minimum the base of support for the feet, while by the simultaneous closing of the eyes any slight disturbance of the sensory conduction, indispensable for normal coördination, becomes increasingly noticeable. Therefore by this or a similar test (for example, having the patient stand on one leg) minimal disturbances of coördination are sometimes revealed which would not be apparent in the horizontal position.

While the normal person—perhaps after a slight uncertainty at the start—is able to stand firmly in the position just mentioned and only occasionally bends the trunk slightly, in the ataxic great uncertainty at once appears. The upper part of the body sways, the feet which were at first in close juxtaposition separate and move to and fro, the extensor tendons on the dorsum of the foot begin to twitch in the effort to restore the disturbed equilibrium, the patient attempts to balance himself with the arms, and finally opens his eyes in order to support himself. This is designated "Romberg's symptom," and in the form described is only found in true ataxia of the trunk or legs. We usually succeed in determining the ataxia on thorough examination in the recumbent posture. There is, however, another kind of swaying on closing the eyes and with the feet in juxtaposition in which ataxia will not be revealed by examination in the horizontal position. Here the swaying itself is different, and only when most extreme can it be confounded with the true Romberg sign. The patients do not attempt to overcome their uncertainty by movements of the trunk or the feet, or by correction of the extensors of the foot. On the contrary, these are impossible because of the coarse and violent movements of the body which at once appear upon closure of the eyes.

A true swaying appears when the patient grasps at objects for support, makes a few steps forward or backward, or seems about to fall. On opening his eyes, all uncertainty quickly disappears, much more rapidly than in true ataxia.

To differentiate the kind of swaying which follows closure of the eyes, we must bear in mind that a true ataxic upon closing his eyes will sway most severely, and is unsteady on standing still even with open eyes, while in the non-ataxic, in spite of marked swaying with closed eyes, this is not the case. The latter form of swaying is observed in certain cases of neurasthenia, hypochondriasis, hysteria, and other neuroses. It is due to a psychical cause,[1] and may be decreased or caused to disappear by various measures. It is advisable for the physician to examine the reaction of the pupils after the patient has just placed his feet into juxtaposition. As the physician converses with the patient, he tells him to cover one eye with his hand, and the physician for an instant covers the other eye with his own hand, thus encouraging the patient and distracting his attention. In such cases we may of course attain the same end and cause the swaying to disappear by other means if only we can distract the patient's attention from the test about to be undertaken; this will prevent restlessness, anxiety, or any other similar hypochondriacal effect. Patients of this kind often spontaneously complain of vertigo.

Scarcely to be distinguished from the behavior of this group of neurasthenics are the actions of another class of patients who also complain of vertigo when this test is made. These are patients with cerebral arteriosclerosis and with certain focal diseases of the brain. In these cases the swaying upon closure of the eyes cannot be stopped by suggestion, like the swaying due to psychical conditions, or by distracting their attention. On the contrary swaying often begins when the eyes are open, like the swaying in severe ataxia.

The enumeration of the various forms of "Romberg's symptom" by no means exhausts the varieties of swaying. Another characteristic form of swaying is found in disease of the cerebellum, and is designated *cerebellar ataxia*. In the horizontal position, as a rule, no ataxia can be perceived. But on standing, and standing with the eyes open, unsteadiness and swaying similar to that of a person under the influence of alcohol becomes noticeable. This swaying is peculiar from the fact that closing the eyes increases the uncertainty much less than might be supposed, and as is the case in other forms of swaying.

I must call attention to a point always to be borne in mind in testing for Romberg's sign. It sometimes happens that those muscles which unite the trunk with the pelvis are either partially paretic, and therefore make standing impossible, or, like the muscles of the extremity, they are in a state of incoördination, i. e., are actually ataxic.

In a case of this kind, as may be readily understood, examination of the muscles of the leg in the horizontal position will reveal perfectly normal coördination. Here the impossibility of standing quiet with the feet in juxtaposition has nothing to do with the lower extremity, but is due to inability to fix and balance the upper part of the body and the vertebral column.

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[1] In this connection the fact is interesting that many cases of amaurosis sway when their feet are in juxtaposition as soon as the eyes are closed.

Before turning to the consideration of the gait I must point to a source of error, frequently overlooked, which sometimes produces Romberg's symptom when no ataxia is present. If the patient is bow-legged or has any other deformity of the legs it is often difficult for him to balance his body with the feet in juxtaposition and the eyes closed, hence the seeming "Romberg's sign."

The observation and judgment of *the gait* forms one of the most important yet difficult processes in the examination of our patients. A normal gait implies the putting forth of sufficient coarse power, and that there is normal (neither increased nor diminished) muscle tonus, intact coördination, undisturbed cerebral innervation, and free movability of the joints. Moreover, in disturbances of the gait there are abnormal sensations and pain in the leg, therefore centripetal processes are active. All of these points may be considered when we examine the gait.

In order that his gait may be observed, the patient should remove his lower clothing and his shoes and stockings. Slight disorders of gait, especially if ataxic, are more readily perceptible in the naked feet than otherwise. If possible, the pelvis and the lower part of the trunk up to the navel, as well as the legs, should be inspected during walking. While the patient walks forward and backward in a straight line on the smooth floor of a large room, the physician should note whether there is a normal tendency of the pelvis to a horizontal plane (forward inclination of the pelvis and lumbar lordosis are seen in muscular dystrophy), and whether the crests of the ilium are equally high and remain in this position during the act of walking. In certain pathologic conditions, particularly in paralysis of the flexor muscles of the thigh, when the patient raises his leg on attempting to walk the entire side of the pelvis is immediately elevated, and the trunk is slightly inclined backward and to the opposite side. In other cases (with paralysis of the gluteus medius and gluteus minimus, muscular dystrophy), just as in congenital dislocations of the hip-joint, the moment the feet are placed upon the floor and one leg is freely moved the crest of the ilium on the moving side is distinctly seen to descend, while on the motionless side an upward movement is noticed. This "waddling" gait is due to the fact that the two gluteal muscles lack the power to keep the pelvis firmly attached to the trochanter. Hence, on walking, the trochanter at the side of the leg is removed from the crest of the ilium, i. e., it rises or, more correctly, it is drawn upward so that by gravity the unsupported surface of the pelvis sinks downward.

More points are to be noted in the legs than in the pelvis: First of all, whether the insertion of the leg into the hip-joint is normal, i. e., whether the tip of the foot appears to turn out or inward more than normal, and whether all the necessary movements of the joint in walking are performed in their normal excursus. If the leg is not sufficiently flexed or extended at the hip or at the knee-joint, or if the foot is not sufficiently flexed dorsally and the tip is therefore not raised from the floor, there is—provided no passive hindrance exists—simply a *paretic* disturbance of the gait. Such a disturbance of gait is observed in all flaccid paralyses of the legs, for instance, in anterior poliomyelitis, in neuritis, etc. The characteristic *steppage gait* belongs to the simple paretic forms of gait. In this gait, which depends upon paral-

ysis of the muscle group supplied by the peroneal nerve, the tip of the foot hangs flaccidly upon the floor. The leg must be hyperflexed at the hip and knee in order to lift the foot from the floor and to make any forward movement. This causes a gait which resembles the walk of a horse (equine gait).

If the movement of the leg from the floor and toward the floor is springy (similar to the spring of the blade of a pen-knife in opening and closing), and instead of the normal and gradual removal of the foot from the floor the sole of the foot pressed against the floor makes a prolonged, lingering, sliding or shuffling sound, we are dealing with a *spastic gait*. When the ear has become somewhat accustomed to it, this disturbance in gait will be recognized by the peculiar scraping sound due to the sliding of the sole of the spastic foot over the floor. If the knees at each step rub against each other, and thus force the feet to describe a long drawn out S-shaped figure, we know that the adductors of the thigh are spastically contracted.

A patient with this spastic gait well-marked reminds one of a soldier practising a "slow step"; the extended trunk appears to hurry ahead while the entire lower part of the body seems to be held back.

As a rule, spastic gait is not purely spastic but is combined with paretic conditions. This is because spastic symptoms primarily follow an interruption of the voluntary innervation of the pyramidal tract.

Spastic-paretic gait is found in myelitis and in traumatic diseases of the cervical and thoracic portions of the spinal cord, in amyotrophic lateral sclerosis, and sometimes in multiple sclerosis, in syringomyelia, and in various spastic cerebral paralyses. Another spastic-paretic gait is the so-called *circumduction gait,* in which the leg firmly extended at the knee-joint and the tip of the foot pressed against the floor are moved forward in an arc by elevating the same side of the pelvis and flexing the upper part of the body backward to the opposite side. In passing, it may be said that a spastic gait may often be diagnosticated by examining the soles of the patient's shoes: We will find a worn appearance at the tip of the sole where the foot scrapes the floor.

A third disturbance of gait, different from the two previously mentioned but no more rare, is the *ataxic*. I must emphasize that this is not a uniform phenomenon, as is, for instance, the spastic gait, but it appears in different conditions. All forms of ataxic gait have in common that on placing the feet upon the floor they do not reach the point aimed at, and which the patient himself expected to touch.

If an ataxic patient is told to walk in a straight line at each step putting one foot just in front of the other, this is either impossible or just as difficult as if he were required in walking to touch at each step one or another definite spot upon the floor with the tip of his foot or his heel.

On attempting to follow instructions the uncertainty increases, and the physician notices that only with the greatest difficulty and by strenuous and compensatory efforts to balance the body, sometimes with the swaying of the entire body, do the feet reach the spot indicated on the floor or one alongside it.

When the disturbance is slight, the previously mentioned tests of ataxia may be made more difficult by having the patient close his eyes. Under these

circumstances a normal person, after a preliminary slight swaying, walks with steadiness.

Among other tests to determine the ataxic gait, I advise the so-called "turn about face" in which the patient, as in the military, turns upon one heel on his axis, without changing the position of the feet which are at a right angle with each other.

It is often well to interpose this rapid turning between the simple tests in walking, so that the command of "right about face" will be sudden and unexpected. On being confronted with this immediate necessity to perform a prescribed movement, slight degrees of ataxia visibly increase.

I have stated that the ataxic gait is not a strict entity. One disorder of the gait which belongs to the ataxic group appears, at the first glance, to have nothing in common with those previously described.

The disturbance to which I allude is conspicuous because the patient throws his legs outward at random, he stamps, or lifts his foot high up from the floor, the gait resembling that of a fowl. The knees appear to be over-extended. On close observation we notice that the patient, in spite of apparent certainty, never quite hits his mark, but either fails to reach it or throws his foot too far, and is finally happy that he can bring it back to earth at all. This gait is seen in severe ataxia associated with conspicuous hypotonia of the muscles of the leg; therefore in some (but by no means in all) cases of tabes.

In other ataxic patients, in the first moment of examination the ataxic gait is less conspicuous than something which reminds us of the spastic gait, a detached appearance of the leg and a stilt-like walk. Simultaneously we observe a peculiar trembling of the legs and body, and at the first glance it is hard to say whether the uncertainty, the zigzagging, is due to pure ataxia or to the tremor. This gait is found in some cases of multiple sclerosis, an affection which, as we have seen, often presents the simple spastic gait. From the foregoing it is evident that the ataxic gait as well as the paretic and spastic elements of other gaits may be admixed.

In speaking of static coördination we have already mentioned *cerebellar ataxia*. This reveals itself in walking just as it does in standing. The patient sways like an alcoholic, i. e., while the individual step and the action of the joints are quite normal the patient reels and sways to and fro from the fact that with each step he must regain his lost equilibrium, hence the constantly swaying, deviating direction of his gait.

These three varieties of gait, the paretic, the spastic, and the ataxic, with their combinations, by no means exhaust all the difficulties in walking; one of the most frequent and most common disturbances of gait we have not yet considered, namely, mere *dragging* of one leg and *avoidance* of its use.

If we observe that a patient drags one leg and refrains from standing upon it as long as upon the other, while no other spastic or ataxic symptom can be discovered, this usually means nothing but that the patient spares the leg.

This assumption seems all the more plausible if the patient puts only the tip of his foot or its external border on the floor. As a rule, in these cases there is pain or other abnormal sensation in the leg; if this disappears the gait at once returns to the normal.

The best example of this latter disturbance in gait is intermittent claudica-

tion, which always continues as long as the pain lasts, and is due to an insufficient supply of blood to the muscles of the calf.

The simple limping gait is conspicuous, in the first place, by its irregular rhythm which deviates from the normal, because only one leg is pathologically moved. Under similar circumstances, i. e., by dissimilar but marked motor impediment to the use of both legs, the paretic, spastic and ataxic gait may show arrhythmia, besides other symptoms which have been described. This also should receive due attention.

In describing different varieties of gait I have only discussed those noted in organic diseases. The question arises, Do none of these varieties of gait occur in hysteria? In answer, it must be stated that all varieties of gait occur in hysteria except the ataxic waddling gait and the true circumduction gait of hemiplegics.

A gait strictly characteristic of hysteria does not exist, unless we consider as such the "sweeping" gait which occurs in hysterical hemiplegia; here the paralyzed leg is scarcely moved at all at the hip-joint, but is dragged stiffly like a broom.

The spastic gait of hysteria closely resembles that of organic disease, and dysbasia and abasia, which are so common, differ but little from ataxic or cerebellar swaying. An aid to the differential diagnosis of the hysterical forms is the great changeability of mind under psychical influence.

Before discussing other symptoms in the motor sphere I must call attention to a valuable point in the investigation of the gait: The patient's walk should always be tested without permitting him the support of a cane. The aid of a cane greatly changes the mode of walking, particularly in the ataxic, and makes a differentiation extremely difficult.

INVOLUNTARY MOTOR SYMPTOMS

(Associated movements, tremor, tremor during rest, test of intention tremor, the tremor of paralysis agitans, coarse and fine tremor, nystagmus, shaking tremor, fibrillary tremor, myokymia, idiomuscular contraction, athetosis, choreic movements, their occurrence, tic, its nature.)

In the examination of the motor sphere we have so far concerned ourselves only with voluntary movements. I will now consider a number of *involuntary* movements which we observe in examination. That form of involuntary motion which is perhaps most closely allied to the voluntary is the so-called *coördinated movement*. Coördinated movements, i. e., the contractions developed by involuntary movements of other muscles than those which obey the impulses of the will, are invariably the accompanying phenomena of motor activity in an individual whose motor sense is dormant. The more highly developed the individual's motor sense, the more control he exercises over the impulses of his will, and he limits to these muscles the contractions necessary for the desired effect. Thus the child gradually learns to suppress a number of coördinate movements; but in most persons many of these movements (for example, the innervation of the facial nerve when making firm pressure with the hand, etc.) persist.

We occasionally meet persons in whom, without other symptoms of disease, these well-developed coördinated movements appear; for instance, whenever

they move the fingers of one hand they also move those of the other hand, or they simultaneously move the toes. In such cases, usually neurotically predisposed persons, these coördinated movements are often observed in other members of the family, and this can be regarded only as a curious anomaly.

Under pathologic conditions, however, coördinated movements occur in individuals not formerly thus affected. During the course of hemiplegia, coördinated movements often appear upon the paretic side; if, for example, the patient makes forcible pressure with his hand upon the healthy side, the fingers of the paralyzed side react slowly and sluggishly. As this symptom appears almost simultaneously with contracture of the paralyzed extremity, it must be regarded as unfavorable. In hemiplegics we sometimes see coördinated movements in the paretic foot when the patient tries with all his might to innervate the paralyzed upper extremity.

Another form of involuntary motor activity is *tremor*. By the term tremor we mean the rhythmic sequence of a number of similar involuntary movements of but slight excursion. Such a tremor will usually at once become noticeable upon examination: for example, the tremor in paralysis agitans, the tremor of the alcoholic, senile tremor, the physiologic tremor due to cold, etc. Other forms of tremor, however, are distinctly revealed only when an extremity is tonically innervated; for instance, when the hands are extended, and among these we find the physiologic tremor which follows excitement and excessive corporeal exertion, the closely related tremor of neurasthenics, hysterical tremor, that of Graves' disease, etc. The tremor present during rest increases on extending the hands. A peculiar form of tremor which occurs during rest or on tonic innervation, can never, or exceedingly rarely be observed, and only becomes distinct when the patient attempts certain movements (*intention tremor*). Even if no tremor is observed in the patient during rest, or upon extending the hands, and no trembling appears when a piece of paper is placed over his hand, nevertheless there may be intention tremor. We test for intention tremor by telling the patient to grasp the finger of the physician which is held in front of him, or we direct him to drink water from a glass filled to the brim, or to carry out some similar movements. Intention tremor, which chiefly occurs in multiple sclerosis, reveals itself by this sign; trembling movements appear which extend perpendicularly from the direction of the intended movement, and these constantly become more rapid, distinct, and violent the more nearly the hand of the patient reaches its goal. These trembling movements are never of great extent, nor do they appear so irregularly as to obliterate the aim of the intended voluntary movement. By this fact we distinguish intention tremor from ataxia, a differentiation which occasionally is extremely difficult. Since not only intention tremor but most other forms of tremor increase with complicated voluntary movements, in the diagnosis of true intention tremor we must adhere very strictly to the previously mentioned characteristics.

The tremor of paralysis agitans forms a decided contrast to intention tremor, being most marked during rest; with intended movements it usually (but not always!) ceases or decidedly lessens. The last mentioned tremor may be readily recognized being the most complicated variety. Usually the tremor of shaking palsy presents the so-called coin-counting characteristic, i. e., there are separate and distinct tremors of the bent fingers and thumb. Occa-

sionally we observe in the forearm the movements of pronation and supination. We now come to the description of the different movements in tremor, and must differentiate between the coarse, medium, and very fine vibratory tremors. The finer the tremor, the more rapid does it become. To the first mentioned group belong the tremor of multiple sclerosis, senile tremor, and that of paralysis agitans. The rapid form is seen in neurasthenic tremor, in chronic poisoning (tobacco), and especially in Graves' disease. Alcoholic tremor may be rapid or slow. We can inform ourselves of the nature of the tremor by watching the vibration of a sheet of paper placed upon the dorsum of the trembling hand or by a test of the writing. If we desire an accurate count of the individual movements, this is readily obtained in the slow forms, and is 4—5—6 per minute. Nothing now remains but to determine which muscles or which joints are moved.

This is usually quite easy. But it is more difficult—sometimes impossible—to determine which parts are primarily active and which are passive and only secondarily and mechanically implicated. This difficulty is enhanced by the fact that while, in the beginning, there is merely a passive movement of some part of the body, for example, the head, under the eye of the observer this may turn into active tremor.

Tremor is observed in the head as well as in the extremities, but rarely in the trunk or eyes. Tremor of the eyes, *nystagmus*, cannot be pathogenetically grouped with most of the other forms of tremor. In some diseases (multiple sclerosis or certain congenital cerebral defects) it may be evoked by having the patient roll his eyes to one side or upward. If laterally a spasmodic horizontal, if upward a rapid vertical or rotary, tremor of the eyeball appears. Horizontal nystagmus may be produced in healthy persons by rapidly turning the eye several times in its longitudinal axis. In multiple sclerosis or other affections this reaction is naturally more prompt and more intense.

A form of nystagmus is sometimes seen in hysteria; in fact, all kinds of tremor have been described in this affection. But one variety of tremor is frequently observed in the hysterical cases (especially in the traumatic), and very rarely in patients with organic disease; namely, a peculiar, coarse, "shaking tremor" with severe oscillations. Sometimes this reminds us of the atypical tremor of paralysis agitans; occasionally it resembles the coarse intention tremor.

Fibrillary muscular contractions are not to be confounded with tremor. They resemble the latter in that slight tremblings succeed each other in a certain rhythm. These movements, however, do not simultaneously affect the entire muscle, but merely parts of the muscles, the fibrillæ; hence, they never produce a locomotor effect. On the contrary, these fibrillary contractions are revealed only by the fact that the small area of skin over the contracting portion of muscle suddenly rises and falls. This phenomenon is usually repeated several times in rapid succession in the same or neighboring areas. If the fibrillary muscular contractions are very active and extensively distributed, we speak of "*muscle waves*" (*myokymia*). Fibrillary muscular contractions may occur in any muscles; in the tongue they are often erroneously regarded as tremor.

Fibrillary muscular contractions and myokymia are frequently observed in

organic and also in functional diseases—particularly in neurasthenia. This symptom appears in almost all diseases of the peripheral nerves and the anterior horns; therefore in all those organic nervous affections which run their course with degenerative muscular atrophy. Hence, fibrillary contractions form a valuable diagnostic aid in the differentiation of degenerative from simple muscular atrophy.

A single fibrillary muscular contraction may be evoked if we stimulate a superficial muscular area by a sharp tap with the percussion hammer. If, however, instead of a rapidly disappearing contraction in the parallel muscle bundles we perceive a transverse quiver at the point where the blow was given and this lasts several seconds, we have before us a pathologic phenomenon, the so-called "*idio-muscular contraction.*" This is most often noted in general cachexia. Instead of the single and fleeting "fibrillary contraction" after stimulation by the blow of a hammer, we sometimes note, analogous to and accompanying the sluggish galvanic contraction, a sluggish fibrillary contraction. This sluggish mechanical contraction, however, is not so valuable a sign as a sluggish electrical contraction, for in certain muscles, even under normal circumstances, a contraction by no means lightning-like appears upon tapping.

We now turn to the more complicated forms of involuntary motion which appear almost solely in the extremities, namely, *athetotic* and *choreic movements. Athetotic* movements are usually confined to the fingers and toes. In athetosis, we note a continuous, uniform, and slow flexion, extension, abduction and adduction of the fingers, these movements not being produced simultaneously in all of the fingers, but each finger performs a movement independently. These motions are very grotesque; they are never observed in voluntary and necessary movements, but are occasionally seen when a person yawns or stretches himself. The tempo of the finger movements often made in yawning reminds us of those of athetosis. Athetotic movements occur bilaterally—usually as a congenital condition—in neurotics and in families. Hemiathetosis is not infrequently a sequel of hemiplegia, either of children or of adults. At all events, athetosis always indicates a disease of the brain. In athetosis, naturally, the power of voluntary motion is decidedly impaired, and still more so when combined with the second of the previously mentioned disturbances, namely, *chorea.* Choreic movements chiefly differ from those of athetosis by their lightning-like tempo. The contractions of the muscles of the face, of the extremities, and of the trunk are very brief. The forehead is wrinkled, the mouth is drawn, the tongue rolls, the eyes are turned, and, simultaneously, the shoulder is raised and lowered, the thorax is expanded, the arm is pronated or supinated, the forearm is flexed, the fingers are briefly flexed or extended or otherwise moved. Analogous contractions are observed in the feet. The most unlike portions of the body may be implicated by the choreic movements, either simultaneously or a few minutes afterward, this and that part of the body being alternately attacked.

If these choreic movements appear singly and substantively, the neurosis, *chorea,* is present. In its milder forms this affection is not serious; an extreme degree is by no means rare, and makes the unfortunate person attacked the helpless prey of a distressing and continuous muscular action which nothing will check, and the resulting exhaustion and impossibility of taking

food occasionally even cause death. Choreic movements are closely related to the athetotic; they appear unilaterally in cerebral foci and in hemiplegia, and resemble pathologic processes which have their seat near the optic thalamus. In this form they are often continuous and persist for years, and extreme muscular atrophy appears in the affected extremity. Unlike athetotic movements, choreic movements are not rare in hysteria.

If the choreic movements are infrequent and merely implicate the face or the fingers, the diagnosis may be difficult because there are also other disturbances of motion which—in their abortive forms—closely resemble chorea. The genesis and pathology of these disturbances of motion, the "*tics*," differ from those of chorea. True tic (not the tic-like clonic contractions in the course of certain cranial nerves which are caused by irritation of the peripheral nerves) invariably produces a complexity of movements which in themselves, as individual motor phenomena, reveal nothing pathologic, and perhaps become conspicuous only by the rapidity and severity of their course. For example, we observe that the patient spasmodically throws his head backward or to the side as we do, for instance, when a collar annoys us, or we notice that a child suddenly grasps the region of the knee, as if to draw up a stocking which has slipped down, or we see that the patient's advancing foot shuffles over the floor as is the case when a small object adheres to the sole of the shoe, etc. If any of these movements occur but once, they seem of little consequence. It is only when we note that the patient frequently moves his head, shuffles his foot upon the floor, or grasps the region of the knee in the manner described, that we perceive these movements to be abnormal. It is true that other tic movements, which under similar circumstances may possibly be regarded as accidental, may make their appearance during the examination and at once characteristically stamp the disease; for instance, a patient suddenly claps his hands, in walking he may suddenly incline to the side or turn around, he may give vent to inarticulate sounds or obscene words (coprolalia), etc. The exact study of tic movements and similar phenomena has made it absolutely certain that tic is caused by a disturbance of the psychical components of the motor act which represent uncontrollable voluntary movements. Hence tic is never to be regarded as the sign of an organic affection.

EPILEPTIC AND HYSTERIC SPASMODIC ATTACKS

(Course of an epileptic attack, nature of the muscle contractions, absolute loss of consciousness; course of a hysterical spasmodic attack, nature of muscle twitchings, clouding of consciousness; pupillary reaction in the epileptic attack and in the hysterical spasmodic attack; difficulty of determining the pupillary reaction; the effect of external irritation upon the course of the spasmodic attack; the manner of falling in epileptic and in hysterical attacks; biting the tongue and other differentio-diagnostic factors.)

Following what has been stated of the examination of the motor sphere, I must call attention to a phenomenon which neither physiologically nor pathologically can be regarded as an entity, and which reveals itself clinically in many somatic systems, most obviously in the motor sphere. This phenomenon consists of *spasmodic attacks*. Our discussion will here be limited to general spasms, since localized spasms will be described in another part of this work.

I shall first describe the symptoms which may be observed in epileptic and hysterical attacks by merely looking on, and will later discuss the points which are important in a differentio-diagnostic respect. Epileptic spasms appear suddenly like flashes of lightning from a blue sky. Only rarely is there a well developed, so-called *aura* (peculiar sensations in the cardiac region, vertigo, ascending heat, etc., sometimes hallucinations). The patient gives vent to a cry or utters a gurgling sound and then falls without noting his surrounding local conditions, and thereby not infrequently injures himself.

A brief tonic stage at the onset with a lateral rotation of the head and eyes or with general extension of the extremities may readily be overshadowed by the succeeding clonic contractions which form the chief feature of the spasmodic attack. These clonic contractions usually occur at once, with great severity and great power. Arms and legs of both sides are symmetrically and rapidly flexed and extended, or they reveal a semi-tonic flexion or extension with gross vibratory movements consisting of alternating minimal hyperflexions and hyperextensions. On inspiration the thorax becomes spasmodic and greatly expanded, remaining thus for some seconds, or it is elevated and lowered with gasps. The obstruction to respiration makes the face bluish red, the production of saliva is stimulated by simultaneous spasmodic attacks in the mouth, the saliva exudes from the lips with a hissing, rattling sound, and is changed into foam consisting of large bubbles. The lips are usually parted. The teeth of the lower jaw are firmly pressed against the teeth of the upper jaw. In this locking of the jaws the tongue is frequently caught between the teeth, is dented or lacerated, and bleeds.

The eyelids are either open or half open; not infrequently the muscles of the eye are implicated in the spasm. The muscles of the trunk are not so markedly involved in epileptic spasms as are the muscles of the extremity. The spasm lasts, as a rule, but a few minutes; after a few isolated muscular spasms have succeeded one another at somewhat longer intervals it ceases just as suddenly as it began. The face rapidly regains its normal color, the respiration becomes more quiet. While the patient is absolutely unconscious during the attack, and reacts to nothing, he now reacts to a touch, to a call, etc., for he opens his eyes, moves his head, murmurs a few words, perhaps drinks a glass of water which is handed to him. Usually, however, he is still so confused and debilitated that he takes but little notice of his surroundings; on the contrary, he turns upon his side and falls into a deep sleep. From this the patient awakens feeling very weak, and from familiar sensations he knows he has had an attack. Of the attack itself he generally has not the faintest recollection.

Although variations in true spasmodic movements are not at all rare—I need only refer to epilepsia cursoria and epilepsia rotatoria (in which latter condition the patient during the spasm turns on his own axis)—as a rule all epileptic spasms conform to the type which has just been described, so that we may speak of a "typical" epileptic attack.

On the contrary, in hysteric spasm it is impossible to speak of a "typical" attack. Just as the spasm of one epileptic attack in the main resembles that of another, just so surely do hysterical spasmodic attacks show manifold variations.

A hysterical spasmodic attack never sets in with such severity and suddenness as an epileptic attack. As a rule, there is some pain, some peculiar sensation, "nausea," "palpitation of the heart," "anxiety" and the like, to show that an attack is beginning, and this causes the patient to seek a place of safety for the possible fall. Frequently the hysterical attack follows some unusual mental condition or emotion. In this case, the original and at first physiologic expression of this emotion, crying or laughing, becomes more immoderate and by its duration constitutes a pathologic symptom, for without a sharp line of demarcation it passes into an actual attack. It is remarkable that in nearly all hysterical spasmodic attacks the muscles of respiration which physiologically are so closely related to the expression of emotion are predominantly implicated. While, as a rule, the epileptic attack begins with a brief tonic spasm followed by a long clonic stage, in the hysterical spasmodic attack tonic and clonic spasms may alternate. Tonic spasms in hysteria are often of long duration, even lasting several minutes, which is not the case in epilepsy.

What follows the initial stage of the hysterical attack, which is often marked by laughing, crying, or moaning which causes a marked increase in respiration, varies so extraordinarily in individual cases, and often even in the same case, that no uniform symptoms can be described. Sometimes the patient merely flexes or extends the limbs as in epileptic contractions, at other times he stamps his feet or drums with the extremities, or upon the floor or in bed he turns upon his own axis, rotates his head to the right and to the left or forward and backward, flexes and extends the trunk, or raises and drops the pelvis as in vomiting; in the severest attacks we observe the well known "*arc en cercle,*" a tonic spasm involving the entire musculature of the back and the legs, so that only the rigid head extended backward and the heels touch the floor or bed, and the rest of the body is arched upward (opisthotonos). These movements which have been described may each occur singly and constitute the sole feature of the attack, or they may alternate. In some cases, during the course of the spasm very complicated movements appear which can hardly be designated as *spasms.* The patient bites, scratches, or strikes about him, he rises in bed, tears his hair, etc. These "*muscle deliria*" form a transition stage, sometimes are even accompanying phenomena of actual delirium which not infrequently appears in the course of a hysterical spasmodic attack, and begins or terminates the spasm. Hence, it is evident that the mimical muscles are predominantly involved in the spasms of hysteria. The eyes, as a rule, are firmly closed and the branches of the facial nerve supplying the mouth are often innervated. Frightful grimaces are not rare in this condition.

During the hysterical spasmodic attack consciousness is never wholly lost, but is more or less impaired. It frequently shows a marked change in that the patient, during the attack, is completely lost to his surroundings, and believes himself to be in a strange place or position, and even loses the consciousness of his own identity.

Recovery from a hysterical attack may be quite sudden with an immediate and full return to consciousness, or it may be gradual. The patient looks anxiously about, half astonished, helpless, not knowing what to do, rises, and then, as a rule, recalls, as a dream, what has happened. In the unconscious

interval of the psychical condition, the patient has apparently been aware of the attack. Thus, the underlying cause of the emotion which brought about the attack is frequently remembered at the moment of waking. The entire duration of a hysterical spasmodic attack may vary from minutes to hours.

All that I have stated of the two most important forms of spasm may readily be observed without special investigation and without even touching the patient. After a number of spasmodic attacks have been witnessed, and considering what has here been stated, the diagnosis of the nature of the attack will be very easy. If, however, this experience is lacking, if the physician himself does not see the attack or only a part of it, or—what is most often the case—the attack is not typical, other factors must be considered before we can make a differential diagnosis between epilepsy and hysteria. The most important differentio-diagnostic factor is the *reaction of the pupils to light.* It should be remembered that, as a rule, there is no reaction to light in the epileptic attack, but this reaction is present in the hysteric attack, although there are exceptions to this rule; for, as rare occurrences, we note hysteric spasms in which the pupil, owing to a spasm of the dilator muscle, is continuously dilated, it does not follow the stimulation of light reflexly, and it cannot contract.

While hysterical pupillary rigidity as a feature in the differential diagnosis is of very rare occurrence, difficulty in determining the reaction of the pupils is much more common. In the first place, rigidity of the pupils *in the epileptic attack* by no means continues during the entire spasm, but often only *for a few seconds.* Toward the end of the epileptic attack, which may be the only part of the attack that the physician sees, the pupils again react. Inversely, the pupil is just as often dilated in the hysteric attack as in the epileptic attack, and not only resembles the pupil of the epileptic, but reaction to light, although not absent, is extremely slow. In such cases the physician should permit the light to fall two or three times into the eye before he trusts his own observation, and even then he is usually in doubt as to whether the reaction was absent shortly before the examination and is now only slowly returning, whether the attack is epileptic, or whether he is dealing with a mydriatic and slowly reacting hysterical pupil. But these internal difficulties are not the only ones; there are also external ones. Manifestly, it is very difficult to determine the reaction of the pupil during a spasm. I shall only mention the difficulty of securing a good light just in front of the eye of a patient who is in spasm, and the possible danger to the patient's eye by carelessly holding a light too near it, or by permitting a particle of the light, for instance, a part of a match, to fall into the eye. I will merely state that it is often very difficult to get at the eyes or to see the pupils at all while the head of the patient is moving in spasm, or when the eyes are spasmodically rigid, are turned upward, or are moving to and fro. Fortunately, however, from the last mentioned condition we may often draw diagnostic conclusions as to the nature of the attack. If, when we attempt to open the lids and examine the eyes, the patient closes them more tightly, or if he violently beats his head about from right to left while we attempt to fix it, and thus tries to prevent an examination, this may be regarded as a positive proof of the non-epileptic character of the attack. And here we come to another point which may be of use in the differential diagnosis. From the fact that during the

spasm the patient tightly closes his eyes or tries to draw his head away from the hands which attempt to hold it—even when calling to him, sprinkling his face, etc., are absolutely without result—we may be sure that loss of consciousness is not complete, but that the patient reacts to sensory impressions. In hysterical spasms this consciousness is often obvious on testing with Briquet's grasp (deep pressure in both hypogastric regions): the patient either feels the pain, or in some other way becomes conscious of the pressure. By this grasp—as well as by many other violent or irritative maneuvers—we often succeed in aborting a hysterical attack or changing its course. Another fact is that by firmly holding the spasmodic limbs during a hysterical attack the intensity of the spasm is increased.

As soon as we note *that the spasm*—generally speaking—*is decidedly influenced in any way by external measures or conditions we may assume with great probability the non-epileptic nature of the attack.* Two important differential points are that in hysterical attacks the patients, as a rule, never injure themselves by falling nor do they bite their tongues, while epileptics may severely injure themselves during a spasm, sometimes even suffocating or drowning. According to the degree of consciousness in these opposing forms of spasm, we note that during a hysterical attack the patient sometimes moans and cries, he speaks a few words, or moves his lips as if speaking. This does not occur in an epileptic *attack* (I do not now refer to the so-called psychical equivalents and dazed conditions, such as precede stupor in epilepsy).

The muscular twitchings in hysteria are usually of altogether different character from those of epilepsy, and this may be attributed to the apparently profound loss of consciousness during the hysterical spasm. In epilepsy there is merely primitive flexion and extension, or rotary and similar movements; in the attacks of hysteria we usually see highly coördinate and complicated movements which, on account of their peculiarity, are in sharp contrast to epileptic contractions which are not characteristic of voluntary and intended movements.

In concluding our differentio-diagnostic considerations, I must mention the significant circumstance that involuntary evacuation of urine or feces during a spasmodic attack makes its epileptic nature almost certain; I have now touched upon the most important points by the aid of which most cases can be diagnosticated.

EXAMINATION OF THE SENSORY FUNCTIONS AND OF SENSATION

In the examination of sensation and of the sensory functions of the body we must depend more upon the reports of the patient than upon the investigation of motion. Only the statements of the patient will enable us to decide whether sensory irritation is normally perceptible. The observation of the reflexes, the movements of tendons or of the skin after stimulation are not positive tests; cutaneous reflexes are by no means rare, yet in spite of this the patient's sensation may not be normal; or, inversely, cutaneous reflexes may be absent while touch is correctly appreciated. Therefore, in a restricted sense of the word, the objective determination of sensation or of

the sensory functions is impossible. With these preliminaries, we shall proceed to investigate the organs of special sense and their functions in so far as this is practicable in the office of the physician.

EXAMINATION OF THE OPTIC NERVE

(Eye-ground examination, medullary sheath of the optic nerve fibers, optic neuritis, resemblance to this, choked disc, occurrence of optic neuritis and choked disc, variation in the intensity of the latter, atrophy, optic nerve atrophy after optic neuritis, genuine optic nerve atrophy, the function of sight, the field of vision, testing the field of vision with and without the perimeter, scotoma, limitation of the field of vision, hysterical amaurosis, hemianopsia.)

After ascertaining the reaction of the pupil, we should in every case next endeavor to examine the eye-ground with the ophthalmoscope. Although we may be quite positive of the diagnosis of neurasthenia or hysteria, we should never omit an opportunity of directly examining by means of the ophthalmoscope the only nerve which it is possible for us to examine in every patient.

In examining the eye-ground we first give our attention to the optic nerve papilla. Changes in the retina—above all retinitis albuminurica—are by no means rare, but the changes in the optic nerve are more important. A peculiarity of the origin of the optic nerve, which is not pathologic but which, it appears to me, occurs often in neurotics, occasionally causes errors. I refer to the contents of the *medullary sheath* of the optic nerve, which is by no means rarely found. The entrance of the optic nerve in such cases resembles a sun-burst with protuberances upon its border: Medially from the periphery we see the light yellow, shining disc of the optic nerve from which bundles of the same light color radiate in various directions. This radiating appearance is due to the fact that at their ends these bundles in the retina proliferate somewhat, and diverge to a slight degree.

This abnormal but not pathologic feature of the optic nerve disc must not be confounded with the pathologic cloudiness of the papilla which denotes optic neuritis. In this affection, the normally sharp outline of the papilla is no longer clearly defined; the usually distinct line of demarcation is obliterated, the color of the optic nerve is no longer a glowing golden yellow of various tints, but, as a rule, is uniformly a yellowish red. Moreover, the vessels, particularly the veins, are not slender and slightly tortuous, but are filled, dilated, and markedly tortuous.

With some practice the recognition of optic neuritis is generally easy. Confusion is occasionally brought about from the fact that anomalies of refraction make the optic nerve disc appear somewhat dull, as, for example, in hypermetrophic children. But I must issue a warning not to lay too much stress upon the fulness of the vessels.

If, besides the signs of optic neuritis, we note that the papilla is very prominent, if the vessels, which in this case should extend from the retina above the higher level of the optic nerve disc, are "kinked," if there are hemorrhages in the surroundings of the optic nerve disc, or if the entire eye-ground appears swollen or infiltrated, we are dealing with *choked disc* (stasis papillæ). In practice, a sharp differentiation of choked disc from simple optic neuritis cannot always be made.

Optic neuritis—as well as choked disc—usually occurs bilaterally, primarily in pathologic conditions in the anterior areas of the basal cerebral membranes (cerebrospinal syphilis and similar processes) with inflammatory symptoms. Optic neuritis, however, occurs also in certain diseases of the spinal cord (myelitis, tabes, multiple sclerosis) without essential inflammatory processes, as well as in rare cases of multiple neuritis. Unilateral optic neuritis indicates a circumscribed pathologic process implicating only *one* optic nerve.

When choked disc is recognized, we may feel assured of an intracranial increase of pressure.

This increase of pressure is usually caused by a brain tumor. It may, however, also be due to serous meningitis, and in rare cases to an abscess of the brain or to hemorrhage. Primary increase of pressure in the posterior cranial fossa especially favors the development of choked disc because the latter is separated by the tentorium from the remaining parts of the intracranial space. Here I must call attention to the fact that optic neuritis and choked disc may vary greatly in intensity during the course of the disease. In a case on which I subsequently held an autopsy I determined the periodic and absolute disappearance of choked disc which was due to a glioma of the cerebellum, a finding which was confirmed by a prominent ophthalmologist.

Another change, much more frequently found in neurologic practice than optic neuritis and choked disc, is *atrophy of the optic nerve.* This is manifest in the ophthalmoscopic picture in that the papilla has either entirely or in certain areas lost its beautiful golden yellow color, and is paler than normal. The pallor varies between an abnormally light yellow and an almost porcelain-like color. If the pallor in atrophy is due to an optic neuritis which has run its course, the pallor usually implicates the entire optic nerve disc.

If, on the other hand, we note that only a portion of the disc is pale, usually the nasal half of the inverted picture, genuine primary atrophy is indicated, and this in the course of time may lead to atrophy of the entire transverse section.

Non-neuritic atrophy of the optic nerve will be found in tabes, in progressive paralysis, in multiple sclerosis, and in certain general intoxications (chronic alcoholism, lead poisoning, nicotin poisoning, etc.).

The investigation of the function of sight is only of importance to the neurologist inasmuch as it is unnecessary for him to determine errors of refraction and to test the refractive media.

The test of the central *acuity of vision* is in most of our cases of subordinate importance; it may be made by the aid of the familiar Snellen's tables.

The examination of the *field of vision* is much more important in the diagnosis. If no perimeter is available, the investigation of the field of vision is very inaccurate, but with much practice results may be obtained without a perimeter. The patient is placed with his back against the light, one eye being covered with his hand. The physician seats himself opposite his patient, who is told to rivet his eyes upon the physician's face. Now a light tissue, for instance, a folded handkerchief, is waved to and fro, each time at a different angle from the periphery to the center of the field of vision. Whenever the patient, looking into the face of the physician, sees the handkerchief approaching, he must say "now." In this way hemianopsia and even con-

centric limitations of the field of vision not of too slight degree may be determined.

In examination with the perimeter, a piece of paper about 1 c.c. in size is fastened upon a black staff as a testing object. With this we perform movements similar to the coarser test with the handkerchief; but, instead of moving the test object with the free hand, it is moved upon the firm or rotary meridian of the perimeter. In every investigation of the field of vision we must be careful to see that the head is kept perfectly motionless, and, if necessary, the upper eyelid must be drawn slightly upward with the fingers. Moreover, it must be borne in mind that the test object is longer visible if it be moved from the center of the field of vision to the periphery than when moved in the opposite direction. As a rule, the limits of the field of vision are attained by moving the test object from without toward the center. If, instead of a white test object, a colored one is used, the field of vision for colors may be determined, a process which is indicated when we find pallor of the optic nerve.

While the external limits of the field of vision may, under some circumstances, be determined without a perimeter, this is absolutely necessary for the recognition of a dark spot (scotoma) in the center or other region of the field. The test object should not be larger than 0.25 c.c. The examination of the field of vision necessitates considerable practice on the part of the physician as well as no small degree of intelligence and the coöperation of the patient.

Limitations of the field of vision and scotomata are observed in all diseases combined with optic atrophy; in doubtful cases the limitation of the field of vision makes it certain that a doubtful pallor of the optic nerve is the expression of beginning atrophy.

Concentric limitation of the field of vision is also observed in hysteria and other neuroses. Functional limitation of the field of vision, unlike visual defects in diseases of the optic nerve, are not absolutely constant. Sometimes the limits of two fields of vision observed at the same sitting do not coincide. The most extreme degree of concentric limitation of the field of vision which is seen in nervous diseases is furnished by hysteria, and not by organic affections.

Hysterical *amaurosis* sometimes develops gradually from an extreme visual limitation of this kind.

A peculiar, but also rare, defect in the field of vision is *hemianopsia*, in which one-half of the field of vision is absent. According to the absent half of the field of vision in both eyes affecting symmetrical or non-symmetrical halves of the retina we speak of "heteronymous" or "homonymous" hemianopsia.

Hemianopsia may readily be diagnosticated without a perimeter. We may suspect its existence if the patient complains of defective vision "in one eye," also if his sight is poor upon the street, and he frequently "stumbles over things." Hemianopsia is a transitory symptom in migraine; usually it indicates an organic disease of the brain.

EXAMINATION OF THE SENSE OF SMELL

(Nasal respiration, manner of testing the sense of smell, physiologic hyposmia, hyperosmia, central disturbances of smell, the sense of smell in paralysis of the fifth and seventh nerves.)

Examination of the senses of smell and taste is not necessary in all cases; as a rule, our attention is directed to these senses by the symptoms.

Before testing the smell we should ascertain whether both sides of the nose are alike permeable to the inspired air. If the nasal respiration is restricted to one nostril, the sense of smell on the other side will of course be diminished. We must choose as reagents substances which will not simultaneously stimulate the fifth nerve and thereby produce abnormal sensations in the nose; oil of peppermint, asafetida, watery tincture of valerian, cologne, etc., are suitable. The mode of examination is simple: The patient should close one of the nostrils with the finger while the substance to be smelled, in a small bottle, is held in front of the other. The report of the patient will indicate whether or not there is any perception of smell. Some substances of disagreeable odor cause a rapid reflex backward movement of the head which can scarcely be suppressed.

In the absence of other local changes (catarrh) a constant and decided difference in the sense of smell of the two nostrils on free nasal respiration must be regarded as pathologic. Moreover, the differences within physiologic limits in the sense of smell in the same individual—especially the certainty and rapidity of perception—are exceedingly great. In some persons the sense of smell attains merely a very rudimentary development.

General *hypersensitiveness* of smell as well as a general *decrease* of the same, is significant of functional nervous diseases. In hysteria we not infrequently see unilateral anosmia as a partial phenomenon of hemianesthesia. Disturbances of the sense of smell which are certainly not functional, especially the unilateral, are found occasionally in cases of increased cerebral pressure, particularly in pathologic processes in the anterior cranial fossa (tumors, meningitis, fractures of the base of the skull, and the like).

Besides those primarily quantitative disturbances of the sense of smell which have been mentioned, there is also a derangement of the sense of smell which is usually associated with simple hyposmia. While these patients have an appreciation of smell, they are unable to combine with it their memory pictures of smell. This phenomenon is partly due to the fact that certain, perhaps especially characteristic, components of the sensation of smell are decreased or lost, and the others can no longer be recognized. It is also conceivable that in these central disturbances there is a phenomenon analogous to soul-blindness. Paralysis of the fifth nerve sometimes disturbs the sense of smell by dryness of the nasal mucous membrane; facial paralysis makes inhalation difficult, and thus sometimes prevents the patient from smelling.

Abnormal sensations of smell are noted in neurasthenics, hallucinations and delusions of smell in hysteria, in paralytics, and in epileptics as an aura of an attack.

EXAMINATION OF THE SENSE OF TASTE

(The chorda tympani and the glossopharyngeal nerves, the test of taste, individual variations in taste, disturbances of the sense of taste.)

The sensation of taste depends upon the function of several nerves. In the anterior portion of the tongue the nerve of taste is the fifth nerve, or its branch the lingual, which by a circuitous path—chorda tympani, trunk of the facial—brings the sensory fibers from the third to the second branch of the fifth nerve. The glossopharyngeal nerve transmits the function of taste to the posterior portion of the tongue and the palate. A perfectly normal sense of taste is only possible when, besides the proper function of the two nerves mentioned, the sense of smell is not appreciably disturbed pathologically.

Taste is best investigated in the following manner: The patient is told to protrude the tongue, and we observe whether it is free from coating. During the whole examination the tongue is to remain protruded.

The patient informs us by writing upon a piece of paper what sensations of taste he has experienced. Upon this paper the four qualities of taste are written: Sweet, acid, salt, and bitter. If the patient is conscious of no taste he makes a negative motion with his head. He is not permitted to speak during the examination for, by retraction of the tongue within the mouth, the testing substance may be distributed over the entire tongue. I must call attention to the fact that the sense of taste in different individuals varies greatly in degree as well as the relative implication of the tongue and the palate.

With a glass rod or a small dropper or pipette one drop of a strong sugar solution is distributed over the edge of one side of the tongue. The patient must indicate the sensation of taste by making signs with his finger. The same process is repeated upon the symmetrical other half of the tongue. By nodding his head, the patient may report whether the taste on both sides of the tongue is uniform or not. The mouth should be cleansed with a little water, and an examination be made with other reagents. The best substances for this purpose are the following: Table salt, vinegar, and tincture of quinin. The solutions should not be so strong as to produce a burning sensation in the tongue or in the nose. On account of the persistence of the taste, solutions of quinin should be employed last, and after using vinegar it is well to pause a short time before continuing the investigation.

Apparently there are some individuals who, even under normal circumstances, have no perception of taste while the tongue is protruded. In these cases, after distributing the fluid on one side of the tongue this organ is dried and drawn back into the mouth. The distribution of the substance over the entire tongue is thus prevented, but, on the other hand, the palate becomes implicated in the function of taste.

A bitter taste is said to be chiefly transmitted by the posterior portion of the tongue, and this area should be tested in the same way as the anterior part. In hysteria there is unilateral and bilateral absence or diminution of taste. Among the organic conditions in which disorders of taste occur, we must mention affections of the facial nerve in the Fallopian canal, diseases

of the fifth nerve, and paralysis in the course of the peripheral glossopharyngeal nerve or its nuclear region.

Perverted taste is frequent in paralysis; but many neurasthenics and hypochondriacs also complain of abnormal, and usually unpleasant, sensations of taste.

A disturbance of taste can be strictly differentiated only after it has been several times demonstrated by examination.

EXAMINATION OF THE SENSE OF HEARING

(Test of air and bone conduction, Rinne's test, disturbances of hearing and equilibrium combined, diminution of the power of hearing, atrophy of the auditory nerve, hysterical deafness, commotio labyrinthi, subjective auditory sensations.)

In the following I shall briefly outline the examination of the organ of hearing, and shall mention those points most important to the neurologist. In all doubtful cases, and whenever the symptoms referable to the ear and the function of hearing are decidedly important in a neurologic sense, the ear should be examined by an otologist.

This is particularly true when there is a possible affection of the labyrinth or, in a restricted sense, a nervous defect in hearing.

The examination of the ear should begin with an inspection by the speculum and the reflector, then we test the function by the conduction of air. The patient closes one ear with the finger tip, and, standing at his other side, we whisper into his open ear with great distinctness and at varying distances clear vowel sounds, such as "papa," etc. Then we determine in meters the maximal distance at which whispered words are still heard. The other ear is then examined in the same way. The employment of a watch for this test instead of the whispering voice is not advisable.

Normal conduction of air is only possible when there is no noteworthy change in the conduction apparatus of sound (auditory canal, tympanum, tympanic cavity, and Eustachian tube). For the neurologist, the next step in the examination of the ear is the determination of the *bone conduction* (craniotympanal conduction). This conduction through the bone is tested by striking a deep sound with a tuning fork, which is then placed upon the middle line of the skull (for a comparative test of the ears), or upon the mastoid process (to test one ear). The patient must state whether he hears the sound, and upon which side he hears it. It must, however, be remarked that in determining bone conduction the coaction of air conduction cannot be fully excluded.

Normally the tone of the tuning-fork is heard longer through air conduction than through bone conduction. Therefore, as soon as the tone produced by bone conduction has died away the tuning-fork is to be held in front of the ear, and the patient should report whether the tone which disappeared through the bone is heard anew through the air.

The so-called Rinne's test depends upon the simultaneous action of air and bone conduction as described. Its normal coincidence, i. e., longer perception by air conduction, is called a positive result, while the prolonged duration of bone conduction is termed a negative result.

A positive, i. e., a normal, result of the test favors a nervous or labyrinthine defect in hearing, while a negative result, on the contrary, indicates that the seat of the disease is in the apparatus of sound conduction.

A combination of defective hearing with disturbance of equilibrium always favors an affection of the middle ear, provided there is no intracranial disease.

In all cases in which disease of the middle ear is diagnosticated, the decision as to whether the labyrinth or the auditory nerve itself is diseased depends upon other and usually quite fruitless observations.

It is very difficult to utilize the simple data obtained in a case of defective hearing. In the present state of diagnosis of the diseases of the ear, the results of examination, particularly Rinne's test, must be used with great caution. We must remember that in advanced age many persons—according to Tröltsch about one-third—have defective hearing, at least in one ear.

Organic pathologic processes, aside from hemorrhages and fractures of the base of the skull, consist chiefly of neoplasms and inflammatory processes in the posterior cranial fossa and at the base of the brain which are mostly unilateral and produce difficulty in hearing. An actual degeneration of the auditory nerve takes place in tabes dorsalis; multiple sclerosis, by one of its foci, may destroy the auditory nerve.

Hysteria may produce defective hearing or deafness, especially if it be of extreme degree. The diagnosis of *hysterical difficulty* in hearing and deafness may be very perplexing if there are no other hysterical stigmata and there is no anesthesia in the region of the deaf ear. The prognosis of hysterical deafness is doubtful. In some cases of hysteria hyperacusia is said to have been observed.

Of other non-organic affections of the hearing I must mention *commotio labyrinthi* which plays an important rôle in all injuries of the head. The diagnosis of this disorder, frequently met with in practice, is often difficult and uncertain.

Subjective perceptions of hearing and rushing sounds in the ear may be produced by focal diseases near the trunk of the auditory nerve, more frequently, however, and usually in a most distressing form, by arteriosclerosis of the brain. Hysterical subjective perceptions of hearing are, as a rule, neither invariable nor permanent.

If tinnitus aurium occurs paroxysmally and is accompanied by attacks of vertigo we are dealing with Ménière's disease.

SENSORY CONDITIONS OF THE SKIN

(The impossibility of determining sensation "objectively," test of tactile sensation of the skin, of the pain sense, of slight sensation of pain under physiologic conditions, of pain sense in the deeper and superficial cutaneous areas in tabes dorsalis and similar affections; investigation of sensation and of skin and muscle pain with the faradic current; awakening of sensation during the test of sensation; temperature sensation and the nature of the test; locality sense of the skin; occurrence of sensory disturbances, sensory disturbances in functional and organic diseases; compensatory property of the sensory functions; unequal diminution of various cutaneous sensations; dissociated sensory paralysis, its occurrence; retarded sensation of pain.)

The conception and the nature of sensation as a conscious faculty excludes the recognition of the nature of sensation otherwise than from the reports of

the patient. Even those cutaneous reflexes which certainly pass through the cerebral cortex are not an absolute and reliable index of sensation, for such a cutaneous reflex may sometimes be produced without conscious sensation. We see, therefore, the diagnostic value of the patient's reports in this extremely important part of the examination, and consequently, in investigating sensation, we should refrain from influencing the expected replies of the patient. This is all the more necessary as the patient is most susceptible to suggestion when describing sensations.

A complete test of sensation must include the skin, the various qualities and the sensibility of the deeper portions of the mucous membranes, and especially the joints and the muscles.

In practice, the employment of complicated instruments is rarely necessary; rarely is more required than a small brush, a pin, or similar object.

The cutaneous sensation is best tested by lightly touching symmetrical areas at either side of the body—first the head and face, then the trunk and the extremities—with the point and then with the head of a pin. It is always advisable to stimulate two or three times in rapid succession one or two of the small cutaneous areas. By this means we arrive at a general conclusion, and protect ourselves from the error of at once deciding upon a disturbance in sensation in any area if one incorrect report be given. The normal person can always differentiate contact with the point of a pin from that of the head of a pin except in those areas of the skin which, like the soles of the feet or portions of the palms of the hands, are covered with callus. After each individual touch the patient is to answer—of course without looking—by saying "point" or "head." In comparing the perception of touch in the two sides of the body we should only ask: Was the sensation upon the left side the same as upon the right? We should never ask whether the sensation was stronger upon one side than upon the other. If a difference in sensation between the entire right and left sides of the body is reported, we should subsequently compare the regions and members of that half in which sensation is not normal, and limit this difference. If a change in sensation is reported in individual members or areas of the trunk, we must ascertain by further testing whether this disturbance of sensation is along the course of the peripheral nerves, whether it is segmentary, or, finally, whether it is confined to limits apparently produced psychically.

Tests with the pin must sometimes be complemented by the brush; while examination merely with a pin is sufficient to demonstrate hemi-anesthetic or hemi-hyperesthetic regions over the entire body it does not suffice for the exact determination of limited sensory disturbances; a gentle touch with a fine hair brush, a piece of cotton, or the like must here complete the investigation of tactile sensation. On the other hand, hyperesthesia cannot be differentiated with the brush; the point of a pin is much more effective. How far the sense of pain is to be taken into consideration in testing with a pin cannot here be stated: A test of the sense of pain should never be omitted, for a disturbance of this sense usually escapes the notice of the patient and even the physician if he does not particularly investigate it. The degree of pain sense, as I shall later explain, is extremely valuable in the diagnosis.

The pain sense is tested either by pinching the skin, or by placing the

point of a pin not too gently upon the skin and observing whether the pain is sufficiently intense, and is experienced at once. A delay in the perception of pain is easily recognized. In utilizing these tests we must remember that the sensation of pain in different individuals varies greatly within physiologic limits. I have observed it to be especially slight in fat persons poor in pigment.

Moreover, it must be borne in mind that the sense of pain is not the same in different portions of the body.

After demonstrating that the superficial cutaneous areas react painfully to the prick of a pin, we still lack proof of the perfect intactness of the pain sense. For it is a remarkable fact that the pain sense of the deeper cutaneous areas and layers of the skin may be greatly decreased, yet the patient will experience pain from the mere prick of a pin.

For instance, after we have stuck the point of a pin through the skin we can often move it to and fro without the patient experiencing any pain, while the slight prick in the upper cutaneous areas has caused the usual degree of pain. This condition is occasionally seen in hysteria, but in my experience much more frequently, and in the majority of cases as one of the first symptòms, in tabes dorsalis and paralysis. If, in these cases, we prick with a pin only the superficial cutaneous layers we often find nothing pathologic.

In all of these tests it must be borne in mind that the intensity of the pain produced is less if the pin be rapidly introduced.

The previously described methods of testing sensation without an instrument may be supplemented by the aid of an electric induction apparatus and thus completed. The results of different examinations may then be registered and readily compared with each other. A large, moistened, flat electrode is placed upon whatever point in the median line or at the side of the body is to be examined. The other pole, armed with a brush or a flat metal electrode, is placed upon the area to be examined. The current should be closed and then slowly increased, and the patient instructed to report when he feels a slight quivering sensation (the equivalent of contact sensation), after which the current is still further increased until he experiences pain. With this method it must remembered that the pain produced by a strong faradic current does not resemble that produced by the prick of a pin or by pinching; for while the latter is felt only in the skin, a strong faradic current, besides producing pain in the skin, also makes the muscles painful by causing strong contractions therein. This is the key to the apparent contradiction noted in the results occasionally obtained when the pain sense is tested with a pin and with the electric current. When the muscles do not react to faradism, even strong currents often produce no pain; but testing with a pin may prove that the cutaneous sensibility and the pain sense are intact.

The variation of a few milliampères of current in different tests is not at all remarkable, for the apparatus may have changed, or the degree of moisture in the skin may vary; but the results of even a single examination may also vary. Here a highly important law, operative in all sensory investigations, is manifested: That, as the resistance of the skin to the galvanic current is gradually decreased to a certain extent during the application of

the current, so is the sensibility of the skin increased during, and by, every sensory examination.

Of the other important sensory qualities of the skin only the *temperature sense* remains to be examined. This is tested by touching the skin alternately with a warm and a cold substance, with the wood or the metal of the percussion hammer, with cold water (about 5° C.), or with hot water (about 20° to 30° C.) contained in test-tubes. The water should be neither very cold nor very hot; otherwise, pain may be produced. The patient, whose eyes should be closed, must state with which tube he has been touched.

Here it will be noted that the stimulation from the touch of one and the same hot or cold application in different portions of the normal body is perceptible in varying degrees (according to Goldscheider's examinations).

The sense of locality in the skin, i. e., the consciousness of the region in which definite cutaneous stimulation occurs, immaterial whether by tactile pain or a temperature effect, need not, as a rule, be investigated. If, with these methods, the patient states what point has been stimulated, this is usually sufficient. If it is necessary to investigate the locality sense somewhat more minutely, simple figures, circles, lines, etc., should be drawn with the finger upon the skin, and the size, outline, and nature of the figures should be described by the patient.

Disturbances, usually diminution, of the various qualities of cutaneous sensation may occur when the sensory conduction from the periphery to the cerebrum is at any point organically interrupted or damaged. There are also purely *functional* disturbances, which, as a rule, are more conspicuous than the organic ones. They appear either as general or unilateral anesthesia of hyperesthesia, limited to extremities or portions of extremities functionally belonging together. Anesthesia in areas of functional nature is rare, but hyperesthesia of certain areas is common. Organic disturbances of the sensibility of the skin follow the peripheral nerves or the distribution of the spinal roots (radicular type) according to their seat and area of distribution. Hemianesthesia or hemi-hyperesthesia of organic nature is rare, and points to a disturbance of the central sensory tracts, the lemniscus, or the posterior parts of the capsular region. As already indicated, in the sensory sphere there is an extraordinary functional faculty of compensation. From this originates the important diagnostic law never to conclude because sensation is unimpaired that the sensory tract under consideration is intact. When the function of the peripheral sensory fibers is lost the sensory disturbance is never distributed in its full intensity over the entire area innervated by the diseased nerve.

The question now arises: Are the various qualities of cutaneous sensation invariably the same because always implicated in a uniform manner, or, as a rule, is only one sense of the skin, perhaps the tactile, implicated, while the other functions are normal or implicated to a much less extent? This question cannot be briefly and generally answered either affirmatively or negatively; for the one as well as the other condition may be observed. In nervous diseases of peripheral origin all sensory functions of the skin are uniformly diminished; in tabes dorsalis, in paralysis, and in syphilitic spinal disease the pain sense is usually more markedly implicated than the tactile sense, but

here also there may be a uniform diminution of all the qualities of cutaneous sensation.

Hysterical disturbances of sensation in the skin, when distributed over the entire body, are more frequently manifested by the implication of the pain sense alone than of both the pain and tactile sense. *Hemi-anesthetic* and monoplegic hysterical disturbances generally involve the pain sense and all other cutaneous sensations; in fact, they form one of the most marked sensory disturbances with which we are acquainted.

From what has been stated it is evident that patients may show the greatest variety of disturbed cutaneous sensations either in combination or dissociated. Usually, however, we understand by dissociated sensory disturbance ("*dissociated sensory paralysis*") a definite disturbance; i. e., the tactile sense of the skin may be retained while the pain and temperature senses may be greatly diminished or entirely absent. In such cases the point of a pin introduced far under the skin is distinctly felt, but is not painful. "Dissociated sensory paralysis" is found most invariably and conspicuously in syringomyelia. It is also found, less marked but much more frequently than is usually assumed, in neuritic processes, in tabes, and in paralysis, as well as in alcoholic and hysterical persons. In the aged, the leg covered with varices often shows certain dissociated sensory paralyses, and a similar condition is occasionally observed in normal persons who are obese.

Before discussing sensation in the muscles, the bones, and the joints, we must mention another rare phenomenon of cutaneous sensation: *Delayed pain sensation.* This phenomenon appears during the test if the patient is told to say "now" as soon as he is aware of contact, and to say "oh" as soon as he experiences pain. Tabetics who exhibit retarded pain sensation during this test immediately respond to a pin prick in the skin with "now," and after some time, often one or two seconds, they say "oh," showing that they have experienced pain.

SENSIBILITY OF THE DEEPER TISSUES

(The importance of sensation for motility, examination of sensation in passive movements, test of the stereognostic sense, of the sensation of tugging, vibratory sensation, disturbances of sensation in the deeper areas.)

The examination of the sensation of the deeper parts, the muscles, ligaments, tendons and joints, is important because only by the combined action of the intact sensory conditions are the previously mentioned portions of the motor apparatus rendered capable of exercising their complete activity, particularly their faculty of locomotion. This shows that in disturbances of the *finer* movements the sensibility of the deeper parts must always be examined. Unfortunately we are unable to test the sensation of the individual parts separately, since we only possess methods for testing certain sensory complexities, in which normally the individual sensory components of the deeper parts are usually intact. These sensory complexities which may be easily tested are the following: The sensation of the position of the limbs, the exact sensation of active or passive movements, sensation for the amount of pulling exerted upon a limb, and, finally, the stereognostic sense, i. e., the faculty of perceiving by the touch, without the aid of any other sense,

the shape and size of an object (for instance, an object placed in the hand). All of the previously mentioned sensory qualities are derived from the sensation of the deeper areas. Only in the stereognostic sense does the sensibility of the skin as well as the sensation of deeper areas become of marked importance. It is true that in the other previously mentioned symptom-complexes it is impossible entirely to exclude cutaneous sensation.

The investigation had best be conducted in the following manner: After the patient closes his eyes, an arm or leg (the latter if he is in the recumbent posture) is slowly and steadily moved from its normal position of rest into another position, for instance, the limbs are crossed, or they are flexed at a certain angle, etc.; we then ascertain whether the patient is distinctly conscious of the nature and direction of the movements which have brought his limbs into this final position, or position of rest. A normal person will distinctly recognize this. Passive movements in the various joints of the extremities must naturally be modified according to the anatomical relations of these joints. The effect of gravity must be excluded by a proper position. If the elbow-joint, the wrist-joint, the knee-joint, or the ankle-joint is to be moved, this area should be supported above and below the joint by the hands, and a mild but uniform rotary pressure should be exerted while passive movement of the joint is slowly performed. It usually suffices if this test is performed with the fingers or toes, as this enables us to detect any disturbance that is present. It is a mistake to bring about the movement by supporting merely the tip of the toe or the finger; on the contrary, in testing the fingers the joint to be moved should be so encircled by two or three of the physician's fingers above and below the joint as to produce uniform pressure and cutaneous irritation on all sides—in the toes this is sometimes very difficult. In this position we attempt slight flexion, extension and lateral movement of the fingers or toes. In examination of the foot the toe which is to be moved must not come into contact with the other toes. Unintelligent persons must be instructed not to make any active movement of the joints to be tested. If, in spite of this, the patient constantly produces muscular contractions, this almost certainly proves that the sensory sphere is affected; for the patient who has not fully understood the passive movement will instinctively try to correct his incomplete perception by the aid of *active* muscular contractions. From the sum of the resistance experienced in these active movements (produced by the hand of the investigator) he endeavors to estimate the change in position passively produced.

A normal person recognizes the slightest passive motion in the fingers. But in the toes, even a healthy person may occasionally, on the first attempt, confound the third with the fourth toe. In no instance should we base our conclusions upon a single answer which may be incorrect.

Testing the "*stereognostic sense*," the combination of cutaneous sensation and sensation of the deeper parts is also quite simple. The patient, whose eyes are closed, is handed some familiar object, a lead-pencil, a knife, a coin, or the like, and is asked if he recognizes it. The normal person will do so immediately. If the patient under examination feels the object, or takes it first in one hand and then in the other, it proves that his power of recognition is defective. If the stereognostic sense is greatly impaired, the object may be taken from the hand of the patient without his knowledge. As a

rule, the patients themselves are conscious of this disturbance of the stereognostic sense, but refer it to the motor sphere. Like ataxic patients, they usually complain of " weakness " in the hands.

These methods of examination usually suffice in practice. A number of instruments and apparatus for more exact tests are described in physiologic text-books. For clinical purposes there is one instrument which enables us to test the patient's ability to estimate correctly the force exerted when we pull his extremities. This apparatus for testing the lower extremities consists of two stockings, in the heel of which there is a pocket in which a ball about the size of an apple is placed. The stockings are pulled on like ordinary stockings and, while the patient's eyes are closed, we put into the pockets a few of the balls which are of the same size, but of different weight. The patient must now estimate the relative weight of the different balls.

There is another method for investigating deep sensation which, on account of the uncertainty of its results, has not been generally adopted for clinical purposes.

If a large, deep-toned tuning-fork is placed on a part of the body, especially where the bone lies close to the skin, the person experiences a peculiar buzzing sensation which, according to Egger, arises from the periosteum, the ligaments, and the capsules of the joints, and which Seiffer and Rydel have designated "*vibratory sensation.*" Most likely the skin is also involved in the perception of vibrations. This vibratory change is noted in diseases of the sensory tract, and is sometimes found when all other sensory qualities are implicated. Sometimes, however, the disturbances of the vibratory sense are of elective character, inasmuch as they accompany derangements of the sense of location, also those of passive movements, and the pain and temperature senses.

Disturbances of deep sensation, which may be investigated by passive movements with closed eyes as well as by testing the stereognostic sense, may appear in combination or singly, like the disturbances of the vibratory sense and disturbances of cutaneous sensation. With disturbances of cutaneous sensation, they form a common occurrence in tabes dorsalis.

In peripheral diseases, as a rule, deep sensation is deranged only in the most severe cases when several nerves of an extremity are simultaneously implicated.

Among the cerebral affections, disturbances of the sense of location and of the stereognostic sense are most frequently met with in those of the cerebral cortex; here they occur isolatedly, without a disturbance of cutaneous sensation. Positive disease of the brain stem with the same symptoms has, however, been described.

Sensation of the deep parts is often disturbed in hysteria; usually there are also cutaneous disturbances of sensation; in rare cases these are absent.

EXAMINATION OF THE REFLEXES

THE TENDON REFLEXES

(Classification of the reflexes, jaw reflex, triceps reflex, forearm periosteal reflex, patella reflex; manner of testing the patella reflex, difficulties of its production, its exhaustion and strength; patella clonus; Achillo-tendon reflex, manner of testing it;

spontaneous production of foot clonus; mechanism of tendon reflexes and their disturbance, disturbance of tendon reflexes by disease within the reflex arcs themselves, increase of tendon reflexes by disturbances external to the reflex arcs, absence of tendon reflexes in disturbances above the reflex arcs, in shock to the spinal cord, from purely mechanical causes; hysterical clonus and "pseudo-clonus.")

According to whether reflex movements are produced by stimulating a tendon, or the skin and mucous membranes, or by the irritation of certain internal parts, we differentiate three varieties of reflexes: The tendon reflexes, the cutaneous reflexes, and the "internal reflexes."

The simplest, and at the same time the most important of these, are the tendon reflexes. From the extraordinarily large number of tendon reflexes which may be normally produced in man only the following need, as a rule, be tested:

The Jaw Reflex.—This is produced by placing upon the lower incisor teeth of the slightly opened mouth a spatula or something similar which extends from 1 to 3 cm. into the oral cavity, the greater portion of it protruding from the mouth. We hold the projecting end, and tap with the percussion hammer upon the upper surface of this portion. By this movement the tendons of the muscles which elevate the lower jaw are suddenly stimulated, the muscles react by a brief contraction, and the lower jaw is slightly raised.

Triceps Reflex.—Physiologically considered the triceps reflex is homologous to the most important of all reflexes, the patella tendon reflex. The latter is produced by the action of the extensor tendon of the leg, the former by the action of the extensor tendon of the forearm. The clinical importance of the triceps reflex is by no means equal to that of the patella tendon reflex. To test the triceps reflex the forearm is loosely flexed, the internal condyle being lightly supported, and we tap with the hammer against the tendon of the triceps. As the tendon of the muscle is very short, it is best to ascertain by palpation just where the muscle terminates and the tendon begins. If we neglect this, we are liable to strike with the hammer the muscle instead of the tendon, and thus produce a direct and mechanical muscle stimulation instead of the reflex. The movement which follows stimulation of the tendon is a quick but very distinct extension of the forearm.

By *tapping* in the region of the *styloid process of the radius* we may produce a reflex—probably conducted from the periosteum—which consists in a slight flexion of the *forearm.* This reflex is of no clinical significance.

Patella Tendon Reflex.—The most important tendon reflex is the *patella tendon reflex.* It may be evoked with the patient in either the recumbent or sitting posture; whether the test had best be performed in one or the other position, must be decided in the individual case. Where the test is doubtful we should try both positions. In the sitting posture, the legs must either be flexed at an obtuse angle or one must be loosely crossed over the other, the muscles being flaccid. We must always be sure that the muscles, particularly those on the flexor side of the thigh, are actually relaxed.

Now the physician gives a quick and not too forcible tap at about the middle of the quadriceps tendon below the patella. The normal response to this tap is a slight extensor movement of the leg, and this at once ceases.

Simple as the production of this reflex is in most cases, in others it may

cause great difficulty. The most important part of the process we have already indicated: All muscle tension in the legs must be prevented. This is best done by diverting the attention, by talking to the patient, by giving him arithmetical problems, etc. The diversion of the motor impulse into other tracts may also aid us. For this purpose the patient may grasp the hand of the physician, or he may lock his fingers together and pull with all his might as if to tear them apart but without detaching them (Jendrássik's grasp). If, notwithstanding this, the patient does not relax—and in hypochondriacs this is not rare—we sometimes best attain our purpose by having the patient, instead of sitting on a chair or in bed, sit upon a table in such a way that his legs swing like a pendulum. Or, while the patient is sitting quiet and the physician is apparently investigating another part of the body, the examiner quite unexpectedly taps the quadriceps tendon.

If this is not successful, we may sometimes produce the reflex while palpating the tendon. One hand is placed over the patella in such a way that the tips of the fingers touch the ligament, and then we tap lightly with the hammer the finger which rests on the tendon. If there is the slightest contraction of this muscle, it is at once felt by the finger over the tendon. Sometimes it is advisable, instead of tapping the middle of the tendon, to tap a lateral portion. We should never pronounce the patella reflex to be absent until all of these maneuvers have been tried, if necessary repeatedly. Remarkable as it may appear, it is nevertheless a fact that even experienced and skilful investigators have erred in assuming the absence of the patella tendon reflex, while others, more patient, have succeeded in eliciting it. It is sometimes difficult to form an opinion as to the presence or absence of the patella tendon reflex because the feeble reflex produced upon the first attempt does not subsequently occur. In such cases (multiple neuritis, myelitis, incipient tabes) the reflex is abnormally exhausted.

The normal strength of the tendon reflex is very difficult to determine. Generally, the tip of the foot moves about 10 cm. on extension of the lower leg. In children the normal reflex is more active.

The patella reflex may be increased in varying degrees: The leg may show a single but greater excursus than normal; instead of responding by a single movement, there may be several contractions steadily decreasing in strength. With an extreme increase in the reflex, a tap even in the vicinity of the patella tendon produces a reflex-like contraction. We must differentiate from this the half voluntary, half reflex movements (flexion of the leg, adduction of the thighs, trunk movements) which appear in very irritable patients when we suddenly tap for the patella tendon reflex. Hysterical patients, particularly neurasthenics, on the production of the reflex often experience a very unpleasant feeling of irritation throughout the entire body.

Whenever we find the patella tendon reflex increased, we must try to elicit the so-called *patella clonus.* For this purpose the patient should lie upon his back with the legs extended; there must not be the slightest tension of the quadriceps.

The patella clonus is produced by grasping the patella firmly with the thumb and index finger while the root of the hand and the forearm are placed upon the thigh. Suddenly, with an energetic jerk, the patella is brought straight downward without, however, permitting it to slip from between the

index finger and thumb. If there is clonus of the patella, this will be repeatedly drawn up by the action of the quadriceps muscle while the investigator tries to hold it down. An almost continuous, vibratory movement of the patella is produced, this being quite uniform and persisting so long as the physician draws the patella downward. This physio-pathologic process corresponds exactly to that in the production of the simple patella reflex: By pulling the patella downward—just as when we tap with the hammer upon the tendon—the tendon itself is somewhat elongated, and there is simultaneously an irritation of the sensory fibers of the tendon. This reflex stimulation leads to a contraction of the quadriceps; the patella rapidly moves upward, but is again immediately drawn down by the hand of the examiner. The sensory irritation from this downward tug produces simultaneously a reflex in the tendon, and the reflex contraction of the quadriceps which at once appears and again draws the patella upward. This action is repeated as long as the hand of the physician draws the patella downward.

In producing the patella clonus we must be careful to press the patella down with the *tips* of the index finger and thumb and not with their basal phalanges, for if we encircle the patella with the angle formed by the basal phalanges of the thumb and index finger, in grasping the patella we involuntarily touch the leg of the patient with the free distal phalanges of the thumb and other fingers and by a too firm grasp prevent the tugging of the patella.

A phenomenon similar to patella clonus occurs when the quadriceps is voluntarily, although unconsciously, made tense (as by hysterics and neurasthenics). In this case also a to and fro movement of the patella may appear, but it is never the true patella clonus (pseudo-clonus).

Among the important tendon reflexes is the *tendo Achillis reflex*. This may be tested by grasping the tip of the patient's foot when he is in the recumbent posture, forcing the tip and the foot slightly upward, and at the same time flexing the leg at the hip and knee-joint, and then delivering a blow with the plexor upon the posterior of the passive and tense tendo Achillis. The tendon responds with a brief contraction of the muscles appertaining to it, and the tip of the foot moves downward. This may also be tested while the patient is lying upon his abdomen; he must so relax the muscles that the physician can flex the leg slightly toward the thigh, and then tap the tendon; or the patient may kneel upon a chair in such a way that his feet extend beyond the edge of the chair, and, while in this position the physician taps the tendon.

In producing this reflex the main thing is for the patient to relax his muscles completely, and with ignorant or very irritable patients this result is more difficult to attain than the production of the patella tendon reflex. When the Achilles tendon reflex is greatly exaggerated, it becomes a clonus, and the mechanism of this clonus and its analogy to the mechanism of the patella clonus may be readily understood when I state that the foot clonus is produced by relaxing the muscles of the leg—best with the patient in the recumbent posture—and briskly moving the tip of the foot dorsally and upward, but not holding it too firmly in this position. As long as the tip is forced upward, the foot makes flexure and extensor movements in rapid succession. Sometimes the mere placing of the tip of the foot upon the floor at a right angle, or strong flexure of the knee is sufficient to produce the foot

clonus. In such cases the patients complain that the foot "trembles" when it is placed upon the floor.

Before reviewing those cases in which a change in the tendon reflexes is to be expected, I must briefly recall the mechanism of tendon reflexes. The stimulation produced by tapping the tendon is conducted centripetally through the sensory nerves to the posterior roots of the spinal cord, which it enters. Corresponding to the anatomical division of the posterior roots into long tracts which pass upward to the nuclei of the posterior columns and short fibers which penetrate the gray substance of the cord, that is, the collaterals, a part of the sensory irritation which is brought to them becomes the substratum for the conscious sensory perception transported upward, and in part it forms the foundation of reflex phenomena in the motor cells of the anterior horn. Those deviations of the posterior root fibers which perform the last mentioned function are therefore called reflex collaterals. In the transverse section of the spinal cord they extend in arches from the port of entrance of the root into the anterior horn. These, as we have previously seen, belong to the elements which are capable of producing power in the anterior horn cell. If the stimulation applied to the tendon of the muscle extends to the anterior horn in the manner described, the motor cell sends a current of power downward through the anterior spinal cord root into the peripheral motor nerve. By means of its terminal plate the latter transmits the stimulation to the muscle, and this responds with a single rapid contraction. This reflex process may be regarded as a typical example of tendon and periosteal reflexes.

The reflex processes may be quantitatively disturbed when there is a disturbance at any point of the reflex tract. In diseases of the sensory endorgans or of the sensory nerves, the reflex is diminished or absent, for the stimulation cannot be transmitted to the anterior horn cells which form the reflex center (marked cutaneous changes, especially thickening, neuritis, or the severance of nerves). In diseases of the posterior roots and in destruction of the reflex collaterals (tabes dorsalis or hemorrhages into the posterior portion of the transverse section) this causes the reflex to be diminished or absent. The same is true of all pathologic processes which destroy the cells of the reflex center in the anterior horn (spinal muscular atrophy, cavity formation, hemorrhages, inflammatory processes in the anterior horn, etc.). In slight affections, particularly in functional disturbances, of the anterior horn cells, the tendon reflexes are increased rather than decreased: For instance, in intoxications (strychnin), particularly when there is general hyperirritability of the gray substance in the central nervous system, in hysteria, in neurasthenia, and in nearly all of the so-called functional nervous affections, also in anemic and other general debility, in the stage of convalescence from internal diseases, and in many similar conditions.

If the descending portion of the reflex arc—anterior spinal root, motor nerve, muscle—is diseased, corresponding to the severity of the affection, there is generally a decrease or even absence of the tendon reflex. At the onset of some diseases, as in the so-called "irritable stage" of neuritis, the reflex is said to be increased. It is difficult to decide whether this increased reflex in certain stages of neuritis is to be attributed to an increased irritability of the motor nerve or to concomitant secondary phenomena. In severe

cases of motor neuritis or other grave affection of the motor nerve, as well as in the degeneration of muscular tissue, and when there is loss of contractile substance (dystrophy), the tendon reflex is absent. It follows from what has been stated that in *organic diseases within the reflex arc* the reflex is generally diminished or absent.

We have seen that the source of motor power for the tendon reflex, the anterior horn cell of the spinal cord, is not an absolutely substantive organ; on the contrary—aside from other considerations—it is in a high degree dependent upon the brain, and is constantly influenced by this organ in its production of power. We have also seen that with a disturbance of the relation between the anterior horn cell and the brain, the continuous output of power from the anterior horn cell, which normally is strictly limited to the production of so-called muscle tonus, is increased, and leads to hypertonia and spastic conditions, and thus markedly changes not only the rest condition of the muscles but also the voluntary movements of the body.

An analogous change occurs in the tendon reflexes as soon as the connection between the anterior horn cells and the brain is interfered with. Here also there is a tendency on the part of the uncontrolled anterior horn cell to a waste of power: The power sent to the descending reflex arc is abnormally great, the tendon reflex is increased, not rarely there is clonus. This increase of tendon reflex in disease of the pyramidal tracts which connect the brain and anterior horn of course occurs only when the diseased focus is situated centrally from the spinal reflex center. It is immaterial in this whether disease of the pyramidal tract originates in the spinal cord itself (myelitis, spastic spinal paralysis, syringomyelia, etc.), in the medulla (bulbar hemorrhages, tumors, etc.), or in the brain (cortical and sub-cortical pathologic foci of various kinds, capsular processes, apoplexy, pontine diseases, or those situated in the posterior cranial fossa, etc.). This increase of tendon reflexes by implicating the pyramidal tracts in any part of their course might well be designated classical increase of tendon reflex, since it represents one of the most important and most common clinical phenomena. When it appears bilaterally, it may sometimes be confounded with a functional and general increase in the reflexes; on the other hand, when unilateral it is invariably uniform, and by its peculiarity may be recognized as an organically produced reflex disturbance.

I cannot deny that an increase of reflexes in disturbances in the course of the pyramidal tracts is not the absolute rule. Under some circumstances, the tendon reflex, instead of being increased, wholly disappears. This is the case in some congenital affections, or those acquired in early youth, usually those in which there are cerebral (porencephalic) foci; but this phenomenon is most common when there is complete destruction of the transverse section of the spinal cord, even when this takes place far above the reflex arc. At first sight this paradoxical condition of the tendon reflexes appears to nullify our whole diagram, previously given. Subsequent investigations, however, have furnished conclusions which bridge over or at least qualify this contradiction, either from the fact that with complete and high severance by transverse section the tendon reflex is absent, because certain hypothetical "reflex tonic" fibers pass from the cerebellum to the spinal cord—therefore fibers which stimulate the reflex are destroyed—or because even with high-seated,

long existing, transverse lesions the gray substance of transverse sections of the spinal cord much deeper situated are nevertheless damaged—at least functionally—so that the stimulation travelling from the reflex collateral to the anterior horn no longer meets a cell which is absolutely capable of transmitting reflexes, although possibly it is otherwise still capable of function. This latter view, which assumes a disease descending from the transverse lesion to the gray anterior horn substance (its effect upon the tendon reflexes being perhaps analogous to that of poliomyelitis), has been confirmed by autopsies, but still fails to satisfy us. A tenable theory should assume that when the tendon reflexes below the transverse lesion are absent the tendon reflexes above this lesion should also be absent. For if the tendon reflex is absent because the gray anterior horn substance situated caudally from the transverse lesion is diseased, we cannot understand why this change in the gray substance should not also extend centrally from the transverse lesion.

The absence of the tendon reflexes immediately after a severe cranial hemorrhage as well as during profound loss of consciousness is attributed to "*shock*" of the gray anterior horn substance. These absent reflexes are to be strictly differentiated from those conditions in which rigid active or passive contractures make the production of a reflex or any other movement mechanically impossible.

When there is a mere increase of tendon reflexes, clonus is noted if the increase of reflex reaches a certain degree. Here I must reiterate that *true clonus is met with in hysterical conditions.* A more common functional affection is the previously mentioned "pseudo-clonus" which is produced by unconscious voluntary muscular contractions, and differs from true clonus in that the contractions soon lessen and cease.

The most common form of clonus is that involving the patella and the foot. Clonus of the hand is extremely rare.

CUTANEOUS REFLEXES

(Toe reflex, difficulty of its production; Babinski's phenomenon, its occurrence, cremaster reflex, abdominal wall reflex and its test, eyelid reflex, sneezing reflex, retching reflex, palate reflex, absence of pharyngeal reflex in hysteria, in epilepsy, and in smokers; nature of the cutaneous reflex, analogies between the toe reflex and other cutaneous reflexes, action of the cutaneous reflexes in cerebral and spinal diseases. Antagonism between tendon and cutaneous reflexes, relation of the cutaneous reflexes to the pyramidal tracts, action of cutaneous reflexes in diseases of the peripheral nerves and of the anterior horns, increase of cutaneous reflexes in tabes dorsalis and in neurosis, slight importance of cutaneous reflexes for the condition of conscious sensation.)

In examining the reflexes of the skin and mucous membranes, I shall mention only those which are clinically most important. First among these is the *toe reflex.* The investigations of Babinski have taught us that the normal toe reflex is this: Slight irritation of the sole of the foot, for instance, as when we stroke it with the handle of the hammer, will produce a slight plantar flexion of the four external toes and simultaneously a slight adduction of the same. This toe movement is the effect of a contraction of the interossei muscles. With this movement of the toes, even on very slight irritation of the sole of the foot, other reflex muscular contractions often occur in the tensor fasciæ latæ, in the sartorius, and in the adductors. But, for the

clinical examination of the reflexes, the signs on the part of the toes are the most important. If, instead of a plantar flexion of the toes on the application of a mild irritant to the sole of the foot, there is a single dorsal movement of the great toe, usually slow, or a dorsal movement of all the toes simultaneously, we are dealing with a *pathologic toe reflex.* This pathologic form also occurs when simultaneously the great toe is moved upward and the other toes are moved downward.

Whether the latter reflex phenomenon can actually be regarded as a "pathologic form" of the normal toe reflex, or whether we are dealing with a reflex phenomenon quite independent of the normal reflex, is not yet certain.

The correct production of the toe reflex is more difficult than that of any other reflex. In spite of the greatest care, contradictory and imperfect results will often be obtained in the different examinations.

The result of the examination depends largely on whether or not the attention of the patient is called to the processes in his foot. Hence I advise that we produce the reflex as soon as the patient is in the recumbent posture with his legs bared, i. e., before we test the coarse power or make any other examination of the legs which may attract the attention of the patient to his feet and legs. For the same reason it is never advisable to produce the reflex several times in immediate succession. Another precaution in the first examination is to apply the irritation immediately to the desired point, i. e., about the middle of the internal border of the foot; and irritation should not be too rapidly nor too briskly produced, nor be too intense. If the stimulation is too strong a so-called flight movement appears with marked flexion of the knee-joint and dorsal flexion of the entire foot and toes which may completely suppress the normal sole reflex.

If we note that the tendon of the extensor hallucis longus is prominent even during rest, or that the great toe is extended, we are dealing with an almost tonic toe reflex, and may feel assured that irritation of the sole of the foot will produce distinct extension of the great toe.

The pathologic form of the toe reflex and an increase of the patella tendon reflex are found, although not invariably, in disturbances and interruptions of the pyramidal tract. Absence of the pathologic toe reflex, however, does not indicate that the pyramidal tract is intact. Babinski reported the pathologic form after epileptic attacks.

The fact that in children up to the age of about nine months—therefore so long as the pyramidal tracts are still undeveloped—only dorsal movement follows irritation of the sole of the foot is no less interesting scientifically than it is practically important. In the adult dorsal movements represent the pathologic form of the reflex, and whether these are ever actually observed in hysterical conditions has not yet been determined, since observations to the contrary are rare. The quantitative change in the reflex phenomenon plays no rôle in the toe reflex.

Besides the toe reflex we must consider clinically the *cremasteric reflex* and the various *abdominal reflexes.* While the patient stands or is in the recumbent position the *cremasteric reflex* is tested by stroking the handle of the percussion hammer upon the internal side of the thigh from the level of the scrotum downward. The reflex consists in this; the contraction of the cremaster muscle causes the testicle upon the stimulated side to rise

for a few seconds. This reflex is of extraordinary constancy; in a recent hemiplegia its absence has a certain practical importance because, in spite of unconsciousness being still present, it enables us to determine which side is paralyzed.

The *abdominal reflex*, or reflex of the abdominal walls, consists of rapid contractions produced in the muscles forming the wall of the belly by irritation of the lateral regions of the abdomen. We differentiate several reflexes of the abdominal wall according to the height of the stimulated cutaneous area. According to Oppenheim, it is sufficient for practical purposes if we test the supra- and infra-umbilical reflexes. The stimulation is best produced by a single, rapid, and gentle stroking of the lateral abdominal wall with a not too sharply pointed lead-pencil or the like. Normally there is a rapid contraction of the abdominal muscles upon the stimulated side. When the reflex cannot be obtained with the patient in the recumbent posture, it is often evoked if he stands. This is particularly true of very fat patients, and of those with very flaccid abdominal walls. If the abdominal walls are tense, as is often the case in nervous patients and in those with abdominal pain, or pain in the lower extremities, the production of the reflex is difficult. Notwithstanding all precautions, and even when there are no pathologic conditions, it is sometimes impossible to evoke the abdominal reflexes.

Clinically the most important mucous membrane reflexes are the *eyelid reflex*, the *retching reflex*, the *palate reflex*, and, perhaps, also the *sneezing reflex*. The *eyelid reflex* is the closing of the palpebral fissure on touching the conjunctiva. The *sneezing reflex* is an attack of sneezing as soon as the mucous membrane of the nose is slightly tickled. The *retching reflex* is relatively the most important of the mucous membrane reflexes; this is tested by depressing the tongue of the patient and touching the posterior pharyngeal wall with a brush, a piece of paper or the like; this induces retching which in rare cases (in alcoholics) may even be followed by vomiting.

The reflex produced in the soft palate by lightly touching the uvula or the soft portions is manifested by the contraction and elevation of the palate.

The behavior of the last two reflexes in the pharyngeal mucous membrane should always be tested in the various bulbar affections, since they often furnish data for localization. In a large proportion of hysterical and epileptic individuals, these reflexes of the pharyngeal mucous membrane are either diminished or abolished. As the same condition is also found in normal persons (particularly in smokers), and as many epileptics continue for years under the influence of the bromids which decrease reflexes, it appears to me doubtful whether the absence of these reflexes in hysteria and epilepsy should always be attributed to a pathologic condition.

Since the question now arises whether there is such a simultaneous action of the cutaneous reflexes as has been described in the various nervous diseases, we must first briefly consider the physiologic nature of cutaneous reflexes.

It is clear that the cutaneous reflexes, like the tendon reflexes, have an afferent sensory and an efferent motor branch in their reflex arcs. It is also evident that the output of power, the motor innervation of the descending branch, takes placcs in the anterior horn cells of the spinal cord. Concerning most of the reflexes it is still a mooted question whether the stimulation from the afferent branch of the reflex arc to the anterior horn cells is directly

and quickly transmitted from the spinal cord—as in the tendon reflexes—or whether the transmission of the afferent sensory irritation (which in the tendon reflexes rises to the cerebrum) is first to the brain itself, thence along the tracts of motor innervation, being finally transported to the area of power discharge in the spinal cord. The latter possibility does not practically differ greatly from a third one, according to which the reflex center of the cutaneous reflexes is situated in the spinal cord, but depends largely upon the condition of the brain, and is constantly influenced by this organ.

As stated, the foregoing questions are by no means solved. It is not likely that all cutaneous reflexes permit the same conclusion as to their reflex centers. For the normal toe reflex which has been much studied in the last few years it is very likely true that the reflex transmission, i. e., the transmission of the afferent stimulation, is a descending one from the cerebrum. For the other cutaneous reflexes this does not seem so certain, although clinical analogies favor it to some extent.

The clinical analogies referred to between the other cutaneous reflexes and the toe reflex are the facts that in cerebral foci, especially when the pyramidal tract is interrupted, there is generally a decrease or absence of the cutaneous reflexes upon the side opposite to the pathologic focus. My reference to the absence of the toe reflex in cerebral diseases must not be regarded as contradictory, for I previously explained that the so-called inversion of the normal toe reflex into its pathologic form (Babinski's sign) is probably to be accounted for by the actual disappearance of the toe reflex, instead of which there is an entirely new, but different, reflex condition. Here the other cutaneous reflexes correspond to the normal toe reflex, in that they are sometimes present notwithstanding the cerebral interruption of the pyramidal tract. Moreover, in high-seated and marked transverse changes in the spinal cord, especially with complete cross section, all of the normal cutaneous reflexes disappear. In cases of high-seated transverse disturbance of the spinal cord, those reflex movements which appear on dorsal flexion as a quick uplift and retraction of the leg after stimulating the skin of the foot are perhaps also to be regarded as a pathologic form of the toe reflex, and present new pathologic reflexes which are normally absent in this form. Here the reflex transmission may be certainly referred to the spinal cord, since the path to the brain is destroyed. From the action of the cutaneous reflexes in central diseases just outlined, the antagonism in these diseases between the tendon and cutaneous reflexes will become conspicuous. This antagonism is often shown not only by the diminution or absence of the cutaneous reflexes when the tendon reflexes are increased, but also because the tendon reflexes are invariably and uniformly either absent or increased; the cutaneous reflexes, however, are very inconstant and have diagnostically a varying importance. Their manifold nature furnishes a hint as to their complicated relations to the cerebrum. That the pyramidal tract, in particular, is intimately related to the cutaneous reflexes may be concluded from the fact that (according to Strümpell) there are normally fewer cutaneous reflexes in the upper than in the lower extremity. Accordingly, with the increasing functional development of an extremity, i. e., with the increasing number of distinct movements which one extremity is capable of performing, the cutaneous reflexes decrease in number or are abolished.

In diseases and interruptions of the afferent and efferent branches of the reflex arc, as well as in highly destructive processes in the region of the anterior horn cells which must furnish the energy for the motor reflex contractions, the action of the cutaneous reflexes is generally analogous to that of the tendon reflexes; i. e., under the previously mentioned conditions the cutaneous reflexes are either decreased or abolished: For instance, in all forms of anesthesia due to peripheral cause, in peripheral paralysis, in neuritic processes, and the like. That the cutaneous reflexes are often actively increased in tabes dorsalis—a disease in which there is usually a great diminution of sensation—instead of being decreased, may perhaps be accounted for by the irritative conditions in the diseased afferent branch of the reflex arc. In favor of this view is the circumstance that, in such cases, besides the anesthesias, or, better, the analgesias of the deeper cutaneous areas, there are often extreme tactile hyperesthesias of the superficial cutaneous areas. Perhaps this increase of the cutaneous reflex in tabes is only functional, and may be attributed to the neurasthenic condition which frequently accompanies tabes, for in many cases of neurasthenia and in most other neuroses the cutaneous reflexes as well as the tendon reflexes are increased.

Before concluding our description of the cutaneous reflexes, I must again issue a warning not to attach too great weight in diagnosis to the action of the cutaneous reflexes, above all not to draw too hasty conclusions as to conscious sensory conditions from their behavior. In functional diseases, but particularly in hysteria, we meet with conspicuous abnormalities in the action of the cutaneous reflexes and in conscious sensation. It would be a great error if, for example, we should diagnosticate anesthesia of the palate from the absence of the palate reflex.

THE INTERNAL REFLEXES

(Pupillary reflex, width of the pupils, occurrence of myosis and mydriasis in organic diseases, in epilepsy, and in other neuroses; anisocoria, its occurrence; sympathetic pupil reaction, light reaction of the pupil, testing the light reflex, its difficulties; duration of the light reaction, abnormally rapid and sluggish light reaction; sluggish pupils in the aged, rigidity to light of the pupils, hemiopic pupillary reaction, accommodation reaction of the pupils, Argyll-Robertson sign, its occurrence; ophthalmoplegia interna; relative preponderance of the reaction of accommodation over the reaction to light, Piltz's sign, consensual pupillary reaction, reaction of the pupils due to psychical irritation.—Reflexes of the discharge of feces and urine, questions regarding the action of the fecal and urinary reflex, diagrammatic presentation of the fecal and urine reflexes, action of the bladder and of the intestine in diseases of the brain and the upper portions of the spinal cord, and in diseases of the sacral cord; the importance of mechanical factors in the evacuation of feces and urine, fecal and urinary reflexes in neuritic processes, in neuroses, in hysterical bladder disturbances.—Sexual reflexes in man, in woman; sexual frigidity in woman; disturbance of the sexual reflex in the male, in organic diseases and in neuroses; priapism, pollutions, menstrual disturbances.)

Under the designation internal reflexes, we shall group for purely practical purposes the pupillary reflex, the reflex of the discharge of urine and feces, and the sexual reflex, notwithstanding the fact that an intimate relationship exists only between the reflexes for the discharge of feces and urine, and possibly for the sexual reflex.

The first and most important of these reflexes we may objectively test;

in the other internal reflexes we are, as a rule, dependent upon the reports of the patient.

The *pupillary reflex* should always be tested at the beginning of the examination. At this time we should observe whether the pupils are equal in size and whether their width corresponds to physiologic limits. In emmetropic persons the pupils, viewed in a clear, diffused daylight, have a diameter of 2 to 3 mm. Marked increase or decrease of this limit is to be regarded as pathologic. Abnormal narrowness of the pupil, *myosis*, is found in the aged, in tabes dorsalis, in syringomyelia, and occasionally in progressive paralysis, in cerebral syphilis, in morphinism, in nicotinism, in the early stages of increased pressure on the brain, and in paralysis of the cervical sympathetic. Abnormal dilatation of the pupil, *mydriasis*, is found in organic diseases of the brain, particularly in brain tumor, also in paralysis, in cerebral syphilis, in high-graded increase of cerebral pressure, in amaurosis, in paralysis of the oculomotor nerve, after the administration of atropin, etc. Abnormally distended pupils are found in some hysterical patients, particularly in epileptics. The pupils of apprehensive neurasthenics and hypochondriacs are often conspicuously dilated.

In deciding as to the uniformity or inequality of the pupils we must not make too subtle distinctions. Very slight differences in the pupils are observed even in health. Conspicuous inequality of the pupils (*anisocoria*) is found in all of the organic diseases in which myosis or mydriasis occurs, provided the anatomical change is not uniformly distributed to both sides, or if the condition is unilateral. Although inequality of the pupils is occasionally seen in functional diseases, yet decided anisocoria indicates the existence of an organic disease. After we have acquainted ourselves with the condition of the pupil during rest, we may proceed to test the pupillary reflex.

Before describing the most important pupillary reflex, the light reflex, I must briefly mention the so-called "*sympathetic pupillary reaction.*" This consists in a dilatation of the pupil on pinching or faradization of the skin of the nape of the neck. Only rarely are we in a position to make this investigation.

The *light reflex* is a contraction of the pupil upon the reflection of light into the eye, and its dilatation with a decrease of light. The reaction of each eye to light must be tested separately. While one eye is examined the other should be lightly covered. The light must be bright, whether it be daylight or artificial illumination. During the examination the patient should look into the distance, and not at a near object.

Testing the reaction to light by means of a lighted match or a candle instead of daylight is occasionally a source of error, for the patient unconsciously accommodates his vision to the object held before him, and thus contracts his pupils. It is better to make the test with the patient seated immediately before a window where the light is good, and he should cover one eye with his hand. While the patient looks off into the distance the physician covers the other eye, the one to be examined, with his own hand. After a few seconds he withdraws his hand, and notices whether the pupil is narrower. If the reaction to light is very slight or doubtful, it is well to keep the patient in darkness for some time, if possible for a few hours. We then examine to see whether the reaction to light has improved.

Even after following these directions it is not always easy to come to a conclusion. The light reflex of the cornea often interferes, particularly if the pupils are narrow. Of course, myosis makes the judgment of the individual case difficult because, if the pupil is contracted, the variations in the size of the pupil are naturally less than with a normally wide pupil.

Under these circumstances, as well as when examining during a spasmodic attack, in coma, etc., a small electric pocket-light is very valuable. This can be brought very close to the eye, cannot injure it, and gives a very intense light.

The reflex contraction and dilatation of the pupils is normally very rapid, and more so in young persons and children than in older persons. Abnormally rapid reaction to light is occasionally seen in excitable neurasthenics or hysterical persons. Sluggish and imperfect reaction to light as a prodromal stage to complete rigidity to light, as a rule favors the organic nature of the accompanying symptoms. Only in the aged do we observe a sluggish reaction or even the absence of reaction to light without pathologic significance. Hence, I advise great care in using pupillary sluggishness as a diagnostic sign if the patient has reached the sixtieth year of age. True *rigidity to light* is observed only in organic diseases. Besides diseases of the eye we must consider the following: Total atrophy of the optic nerve, diseases of the nuclear region of the oculomotor nerves, tabes dorsalis, progressive paralysis, multiple sclerosis, brain tumor, hydrocephalus.

A distinctive reaction to light is the so-called hemiopic reaction of the pupil. This is characteristic because in the hemiopic the pupillary reflex appears only when the retinal half which still sees is influenced by the light, while there is no illumination of the blind retinal half. The demonstration of the hemiopic reaction is exceedingly difficult, for the stimulation of light in the manner described is not readily produced.

So far we have been considering *rigidity to light*, that is, the reaction of the pupil to *light*. There is another reaction of the pupil, of great clinical importance, which like the reaction to light should be investigated in *every* case. This reaction does not strictly belong with those we are investigating, for it is not a reflex but a coördinated movement. However, it is best described when considering the reaction to light. The dilatation and contraction of the pupils is not only reflex, indicating the effect of light or greater stimulation by light, but with a number of movements of the external muscles of the eye and of the lids is also synergistic.

Upon *near accommodation* and consequent *convergence* of the eyes, as, for example, when looking at the finger, the pupils contract, and on looking into distance they dilate. This is the most important synergistic reaction of the pupil, the *reaction of accommodation*. As a rule, the eyes may be simultaneously tested for accommodation.

From the physiologic variations in the reaction to light and accommodation it is evident, *a priori*, that in their pathologic relations these reactions are independent of each other. Clinical experience confirms this. For instance, the most important difference between these reactions, the reaction to light, often completely disappears while the reaction to accommodation is undisturbed. This sign, the Argyll-Roberston phenomenon, is commonly observed in tabes dorsalis and in progressive paralysis, occasionally in focal dis-

eases of the brain. It is never the indication of a functional nervous disease. The inverse, rigidity of the pupil to accommodation with a normal reaction to light, is practically never observed—certainly not when the external muscles of the eye function normally. [This statement is perhaps too sweeping in certain cases of neuritis, as for instance in a number of cases that occurred in the Manchester epidemic, response of the eye for accommodation disappeared while the response to light persisted without involvement of the extrinsic muscles. However, the occurrence is so rare as to make the rule as stated by the author practically universal.—Ed.] Absence of reaction to light as well as to accommodation, so-called *ophthalmoplegia interna* or *complete pupillary rigidity*, is common. Complete pupillary rigidity, except when the eye itself is diseased, is always a sign of serious organic intracranial affection. Combined with other objective nervous symptoms it is found in paralysis, in tabes, in multiple sclerosis, in brain tumor, in hydrocephalus, and in similar diseases. Internal ophthalmoplegia appearing alone usually points to a syphilitic cerebral disease.

A decided decrease of the reaction to light and accommodation, or even its almost complete absence, is found in chronic alcoholism and in the aged. In all of these cases, however, the reaction to accommodation is greater than the reaction to light. In fact, under quite normal conditions, the impression is given that the contraction of accommodation, in comparison with the true reflex contraction, is more powerful and intense.

One of the synergistic pupillary reactions has been much studied during the last few years, particularly by Piltz: In many paralytics and tabetics, but also in patients with other affections, and even in a certain proportion of normal persons, a contraction of the pupil follows the firm contraction of the eyelids by the orbicularis. The eye should be tightly closed, when the physician, opening the lids and holding them apart, prevents their closure, at the same time observing whether the pupils contract. As contractions at once cease if the eyes are not tightly closed, it is evident that contraction soon passes into dilatation. Hence, in testing for Piltz's sign, after opening the eye, and following the preceding brief contraction, the pupil dilates and we obtain an *apparently* paradoxical reaction to light. Some authors who have observed this believe it to be an actual paradoxical reaction to light.

The appearance of Piltz's phenomenon is independent of the previously described reaction of the pupil, and is especially marked if there is rigidity to light or a combination of rigidity to light and to accommodation.

Among the synergistic pupillary reactions we must also mention *consensual dilatation* or contraction of both pupils. Normally, a change in the diameter of the pupils is simultaneous and uniform, immaterial whether this be due to reflex irritation or to coördinated movement. As is evident, the consensual reaction is easily tested.

Finally, to prevent any possibility of error, I must call attention to the remarkable fact that, under some circumstances, the mere presence of light or dark objects may cause a contraction or dilatation of the pupil. This is a somatic reflex due to psychical irritation—the presence of an optical memory picture.

As previously stated, the internal reflexes, the discharge of feces and urine and the sexual reflex, can be determined only by the reports of the patient.

We should inquire whether the *discharge of urine and feces* is normal as to quantity and frequency, whether voluntary evacuation is regular and without effort or delayed and difficult, whether the discharge may be interrupted at will, whether there are involuntary evacuations, finally whether the passage of urine and feces is accompanied by normal sensations.

The mechanism of the reflexes for the discharge of urine and feces is much more complicated and less familiar to us than the mechanism of the cutaneous and tendon reflexes. The disturbances of the internal reflexes may best be considered by employing the diagrammatic method, and comparing the reflexes themselves with the diagram of tendon reflexes or, even better, with that of voluntary muscle innervation.

Therefore the evacuation of urine and feces, while considered as analogous to the innervation of any muscle of the body, is a purely voluntary movement.

This "voluntary movement," which promotes discharge from the bladder and bowel, may normally, like any other voluntary movement, be so innervated at any time as to cause an action, and it may at any time be interrupted. It differs from true voluntary movement only because the innervation, as a rule, follows an irritation from the periphery (the bladder or the bowel). If the central portion of the voluntary tract leading from the brain to the anterior horns of the sacral cord, in which is located the power station for the muscles of the bladder and of the rectum be damaged, analogous conditions are to be looked for in the peripheral portion of the nervous apparatus like those which occur in interruption of the pyramidal tract in spastic paralysis of the extremities; that is, the peripheral reflex arc attains a certain independence by interruption of the pyramidal tract, and it acts alone or to irritation constantly coming from the posterior roots to the anterior horn cells sufficiently to keep the muscles in a permanent state of tonic contracture (as, for example, in spastic intestinal torpidity); or periodically there are brief tonic discharges of power in the anterior horn cells whenever the sensory irritation from the internal organs reaches a certain height (as intermittent automatic evacuation of the bladder). Besides—and this is the most conspicuous disturbance—the power of voluntary discharge as well as of voluntary interruption ceases.

Therefore, any pathological process which interrupts the central reflex tract in the brain or in the upper portions of the spinal cord may cause constipation and retention of urine. Under some circumstances, there may be involuntary evacuations of the bladder as well as retention.

Proceeding from this standpoint, and assuming that the power station in the anterior horns for the muscles of discharge is destroyed, a condition results which resembles flaccid paralysis, and want of tone in the expelling as well as retaining muscles causes a constant discharge of the contents of the intestine and bladder (dribbling of urine). The latter clinical condition is observed in hemorrhage and other destructive processes in the lower portion of the spinal cord.

Analogously as in a complete and high interruption of the spinal cord, and in severe shock to the cord, instead of an expected spastic paresis of the extremity we observe a complete flaccid paralysis and absence of the tendon reflexes (a condition similar to that in disease of the anterior horns), and this

change in the reflex for the bladder and rectum which has just been described (corresponding to a destruction of the motor spinal power station) is frequently seen in severe shock, or after complete transverse interruption of the spinal cord.

The diagram I have just given, which by analogy enables us to understand disturbances of the internal reflexes, shows deviations other than those due to internal neuro-physiologic peculiarities in the nature of these reflexes—just as we never have a rule without exceptions but are constantly forced to use expressions like "occasionally" or "sometimes"—in that many purely mechanical factors are involved in the evacuation of the bladder and rectum. Of course, these mechanical factors are influenced by the appearance of nervous disturbances which lead to a further change in the function of the pathologically altered powers of expulsion.

If in the preceding we have considered only spinal and cerebral diseases, this was because these diseases represent the majority of the disturbances of the internal reflexes; but neuritic processes, particularly polyneuritis, more frequently than is assumed produce disturbances in the discharge of urine. If we believe either the motor or sensory nerve, or both, to be diseased we have the explanation of the existing bladder disturbance.

Functional diseases frequently produce quantitative irregularities in the function of the internal reflexes. Among these is constipation in hysteria and neurasthenia, the so-called pollakiuria (abnormally frequent micturition), polyuria (excessive quantity of urine), the impossibility in neuropathics of voluntary urination in the presence of others, hysterical anuria, the occasional impossibility of passing feces during psychical preoccupation, enuresis nocturna, etc. Unquestionably, although rarely, there may be an actual disturbance of the mechanical function of the bladder which, in the absence of any organic change, must be regarded as *hysterical*. For instance, I some time ago treated a person suffering from paralysis of both legs and occasional incontinence of urine, in whose case, on account of the urinary difficulty, a diagnosis of organic disease of the spinal cord had previously been made by a very prominent physician. The sudden cure of the paralysis and the disappearance of the other symptoms proved the hysterical nature of the affection.

It is self-evident, but must be reiterated, that when the evacuation of urine and feces is disturbed, we must exclude the possibility of an internal or surgical affection of the intestine, of the bladder, of the prostate, etc., before we assume a nervous affection.

The last of the internal reflexes, the *sexual reflex*, belongs to the vasomotor *cutaneous reflexes*. In the male its motor components are comprised in the muscular exertion necessitated by the filling of the corpora cavernosa (erection), in the discharge of semen, and in its expulsion through the urethra. In the female it probably consists chiefly in the filling of the corpora cavernosa, and in the discharge of a physiologically unimportant glandular secretion.

Corresponding to their physiologic functions, disturbances of the male sexual reflexes are more important clinically than those of the female.

In regard to the sexual reflex, women occasionally report that during cohabitaton ejaculation occurs too late or not at all, or that a certain psychical impediment disturbs the reflex action, that libido is almost entirely lacking, and that they have noted these conditions. In these cases, usually occurring

in hysterical or otherwise neuropathic individuals, there is *sexual frigidity.* We have little knowledge of disturbances of the sexual reflex of the female from organic diseases.

If the male complains of disturbance of the sexual reflex, we must ascertain by questioning whether *erection* and ejaculation are normal, and whether the former continues a normal length of time. Absence or incomplete erection, as well as its abnormal duration, is an early indication of spinal disease, particularly of tabes. In practice, however, these symptoms are decidedly more common as the expression of functional weakness and inhibition, as in hypochondriasis and neurasthenia, than in organic diseases.

Among disturbances of the sexual reflex of an opposite nature, *priapism* must first be mentioned; it may be regarded as analogous to the spastic state of the extremity which occurs in high-seated interruption of the spinal cord, as a tonic spasm or spastic condition of the muscles which produces a regurgitation of blood in the corpora cavernosa, and thus causes erection. Priapism is a rare symptom. Corresponding to the occurrence of spasm in the extremities, it is found in high-seated interruption of the conduction in the spinal cord.

Priapism is said to occur also in functional diseases.

Another increase of pathologic activity in the realm of the sexual reflexes is formed by *pollutions.* These are found in organic and in functional diseases. They usually occur at night, rarely during the day. In the latter case they are produced by most heterogeneous psychical stimuli, fear, fright, anger, etc. Nearly all of the patients in whom this unfortunate condition is noted are extremely neurotic.

As a supplement to the so-called internal reflexes, we may briefly consider *menstruation,* for in menstruation also there is probably a reflex process. Disturbances of menstruation are very common in women with nervous affections; the menses may occur with abnormal frequency or be rare; the flow may be abnormally profuse or very scant. Therefore we must always inquire into these conditions. In patients who are severely predisposed to neuropathic conditions, menstruation often ceases because of general hypoplasia or aplasia of the genitalia, but amenorrhea occurs even without tangible change in the genital apparatus or in the general constitution, apparently as a purely nervous affection, in depression, hysteria or similar conditions. Of amenorrhea in organic nervous diseases, particularly spinal affections, we know very little.

In the preceding I have endeavored to give a synopsis of the most important points to be borne in mind during the examination of patients with nervous disease. To avoid prolixity, some important points have been briefly discussed; other significant diagnostic and symptomatic realms, such as electro-diagnosis, central speech disturbances, etc., have not been considered at all because they form the subject of special articles in this book.

I desire to call special attention to one point not yet discussed, for experience shows that this point is unfortunately somewhat overlooked. We should always bear in mind that neurology is a daughter of internal medicine, therefore no neurologic investigation can be regarded as complete unless the condition of the *internal organs,* especially of the heart and kidneys, has been thoroughly investigated, for only when we are in a position to decide as to the condition of the internal organs will our diagnosis of the pathologic nervous conditions be based upon a firm foundation.

MODERN AIDS IN THE DIAGNOSIS OF DISEASES OF THE BRAIN

By E. REDLICH, Vienna

If we date the beginning of the recent advance in the diagnosis of diseases of the brain which we are about to discuss at 1879, the year when Nothnagel's "Topical Diagnosis of Cerebral Diseases" appeared, we may state that prior to that period the diagnosis of cerebral diseases had in a relatively short time made rapid advances. Minute investigation and symptomatology, combined with the utilization of important and newly acquired physiologic factors, beginning with the electrical cortical irritation of Fritsch, Hitzig, and others, our insight into the normal anatomy of the central nervous system, the exact pathological anatomy and histology of cerebral disease—which so far as their importance goes were almost natural experiments in man—have led to the so-called *topical diagnosis* of cerebral diseases, i. e., to the localization of the parts of the brain affected, to a precision and minuteness which, compared with the former and often unsatisfactory methods of diagnosis, have awakened among physicians the liveliest interest in, and admiration for, neurology. The second and no less important question as to the nature of the process present of course permitted even a less positive decision, on account of the limited methods of examination then known; in the majority of cases there was little or nothing to aid us in arriving at *direct* conclusions. The history, the age of the patient, the pathologic changes going on in his body, the frequency of certain diseases, permitted at most merely a guess as to the nature of the process, and then we had to reckon with the factor of its probability.

Since the period mentioned, the diagnosis of diseases of the brain has advanced in all directions, although perhaps not so rapidly as it formerly did. Many of the opinions then formed were modified by subsequent investigations, others became more firmly rooted, many new ones have been added. If I may be permitted to use the expression, the territory of the diagnosis of diseases of the brain has been greatly extended.

It is hardly possible to review the present state of diagnosis of diseases of the brain within the scope of a short article. This, in its most concise form, would require many pages. I prefer, therefore, to describe a number of aids which we may invoke in the diagnosis of diseases of the brain as well as its neurologic symptoms. Much of what I shall state is not new, although most utilized in recent times, and having lately gained greater publicity than formerly; other things are new, perhaps too new, being still in many respects subject to discussion, so that it is to-day impossible to give an absolute opinion concerning the value of individual methods. Neurology has adopted many

of the modern methods of medicine, and has attained such practical success as to lend certainty to its conclusions. In some cases it has become possible by this means to lay bare the nature of underlying processes, and, under some circumstances, for instance, by demonstrating specific microörganisms, even the etiology of diseases of the brain has been made clear. We may readily understand how important this is in the operative treatment of diseases of the brain. Of course, in its endeavor to attain favorable results, surgery is often a step in advance of diagnosis. But in the brain, as elsewhere, exploratory operation has often been resorted to for the purpose of diagnosis.

Every examination should begin with **inspection**, not of the diseased organ but of its covering, the skull. Certain abnormalities of the skull, a premature closure of the sutures, *microcephalia* or *macrocephalia*, its general size or diminutive size, are naturally more valuable points to the psychiatrist, since they reveal, above all, the general damage to the brain. Even more significant are circumscribed flattenings of the skull, since, under some circumstances, these indicate circumscribed lesions of the brain, such as porencephalitic processes dating from childhood. For instance, we sometimes find a unilateral decrease in the size of the skull in cases of cerebral infantile paralysis, and on the side opposite the paralysis. In a case of epilepsy recently demonstrated by Infeld this unilateral decrease in the size of the skull was the only visible residuum of such a cerebral process. Of course, in such cases mere inspection of the skull is supplemented by its mensuration, for which we possess a number of methods the details of which I shall not discuss.

It is well known that Gall was inclined to base many of his conclusions on the mere inspection and examination of the skull; he believed that certain mental qualities, individual traits and faculties, of course as philosophico-psychologic abstractions rather than physiologico-psychologic functions, were developed in various individuals, and by definite factors corresponded to different formations. Gall's teaching was long regarded as a curious survival of a far distant period, but Möbius recently discovered some grains of wheat among this chaff. For example, he believes with Gall that mathematical endowment is indicated by a marked development of the upper external angle of the orbit. He investigated Gall's theory that the occiput developed according to the strength of the sexual function, etc.; in short he attempted to "sustain" Gall's opinions. This met with bitter opposition from neurologists, especially Rieger; I believe that we need not dwell on this subject, since it furnished no absolute facts nor valuable conclusions.

We will now discuss what is of greater practical importance. Inspection of the skull affords certain indications in the diagnosis of acromegalia, that peculiar disturbance of growth which, we must to-day assume, is the result of disease of the hypophysis. The general enlargement of the skull, the characteristic enlargement of the lower jaw, the increased size of the soft parts, of the lobes of the ear, also of the nose and the tongue, are so conspicuous to the expert that they directly indicate the diagnosis, although the enlargement of the hands and feet, the other symptoms, the history, etc., form absolute proof. The diagnosis of hydrocephalus can usually be made at sight. Among its typical symptoms are the semiglobular enlargement of the whole skull, the characteristic prominence of the frontal and occipital portions, the protrusion of the eyes from the forcing downward of the orbital plate, some-

times erroneously regarded as exophthalmos, and more correctly designated as flattening of the eye, the prominence of the temporal region, etc. In the still open fontanelles of very young children, the rigid tension of the skull contents and the increased cerebral pressure may be directly demonstrated. Under some circumstances, even in adults, tumors of the skull and marked cerebral pressure may lead to a loosening of the sutures, and to openings in the skull. Tumors at the top of the skull or even those of the brain, by eroding the bone may proliferate outwardly, and often lead to palpable and visible protuberances of the skull. This permits the direct inspection of a cerebral tumor. As a curiosity I must mention a case of Westphal's in which cerebral echinococci produced in this way an external opening which could be felt under the skin. In the diagnosis of traumatic epilepsy, so-called reflex epilepsy, as well as in essential epilepsy, and in traumatic cerebral abscess, it is obviously important to find cicatrices upon the skull, even though only in consequence of an injury during the attack. Under some circumstances these will not only confirm the diagnosis but will indicate the necessity of operative interference. In such cases it is advisable to shave the skull prior to examination.

In the veins of the skull, just as in other parts of the body, local stasis may occur in consequence of an obstruction to the circulation, and circumscribed, visible, venous dilatation or edema appear. This occurs most frequently in sinus thrombosis; thus, thrombosis of the sinus cavernosus leads to dilatation of the frontal veins, to cyanosis of the frontal region, and to swelling of the eyelids, this condition being proven by the demonstration of dilated veins in the retina by ophthalmoscopic investigation. Thrombosis of the transverse sinus leads to edematous swelling of the soft parts behind the ear, and to dilatation of the veins in this region, which are also prominent symptoms of suppuration of the mastoid process; thrombosis of the longitudinal sinus causes dilatation of the veins and edema along the median line of the skull. These local venous dilatations are also occasionally found in brain tumors, in superficial tumors, even in those directly above the brain tumor. I must mention that upon inspection we sometimes find small ecchymoses upon the skin of the forehead, in the conjunctivæ, or in the face, etc., after severe epileptic attacks, and this is of great significance since, as a rule, the diagnosis of epilepsy cannot be directly made if the physician is not an eye-witness of the attack, but must rely solely on the history.

It is self-evident that the *X-rays*, which have become so important in medicine, should have been resorted to in the diagnosis of diseases of the brain, but so far with but little success. The X-ray examination is of decisive importance in searching for projectiles in the skull. Exostoses and openings in the skull can also thus be diagnosticated with certainty. An attempt has been made to find many other things with the X-ray photograph—osteoporosis of the bones of the skull, hemorrhages in the meninges, thickening of these structures, etc. It was hoped that brain tumors could in this way be directly demonstrated, and thus give to the diagnosis of traumatic neurosis, this true cross of the neurologists, a firm basis. But here the greatest care is necessary to prevent gross errors. I shall quote a case of Slavyk which presented clinically the symptoms of brain tumor. In the X-ray picture there was a marked shadow in the region of the sella turcica and extending to the parietal

region. The autopsy, however, revealed the presence of a tumor of the pineal body, the shadow having nothing in common with this tumor. All experts in X-ray examinations can report similar errors. Generally, the bones of the skull form an impenetrable wall for the structures contained within, so that no shadow can be cast. Under very favorable circumstances, X-ray examination may reveal facts which are important in the diagnosis of endocranial processes, especially of tumors; for example, when marked calcification has taken place in the tumor, which may then be manifested by a distinct shadow. In three cases of tumor of the hypophysis—reported by Oppenheim-Cassirer, Fuchs, and Embden—the skiagraph revealed a dilatation of the sella turcica, therefore a result of the tumor which best supported the diagnosis.

In physical diagnosis, **percussion** follows inspection and palpation of the skull; and is also of some importance. We first test the sensitiveness of the skull to percussion; this may be either diffuse or circumscribed. Diffuse sensitiveness to percussion is often associated with sensitiveness to pressure which is distributed over the whole skull, or at least to great areas, and is a symptom which can only be utilized with extreme care. This is frequently observed in functional neuroses, especially in neurasthenia, hysteria, traumatic neuroses, etc. There is a form of headache designated as indurative headache; in this condition, according to general opinion, small infiltrations and palpable nodules appear in the galea aponeurotica; conspicuous sensitiveness in extensive areas to pressure and to percussion being here characteristic. In organic lesions of the skull and of the brain, there may be a diffuse sensitiveness of the skull to percussion, just as in disease of the bones of the skull; Oppenheim reports that this is occasionally a sign of osteoporosis and thinning of the bones of the skull. The condition is observed in disease of the meninges, in pachymeningitis, in tuberculous meningitis, in disease of the brain proper, such as tumors, etc. Much more important is a local sensitiveness to percussion sometimes combined with local sensitiveness to pressure. This may also be noted in the form of the well known clavus in neuroses, in neurasthenia, and in hysteria, and frequently in traumatic neuroses at or near the point of injury. In other cases, such a circumscribed sensitiveness points to organic disease. Diseases of the cranial bones, tumors and inflammatory affections of the skull, often run their course with excessive painfulness in the corresponding area. Brain abscess, and especially brain tumor when it presses against the bones of the skull and irritates them in circumscribed areas, may produce circumscribed sensitiveness to percussion, and thus prove a valuable, local, diagnostic sign. Sometimes, at least, the fossa of the skull in which the tumor has developed may be determined by the circumscribed sensitiveness to percussion. Of course, this may prove a source of error; percussion over a tumor of the frontal region will sometimes produce pain in the occiput, and *vice versa.* Many errors in diagnosis have arisen in this way. Sometimes we must be content with deciding by means of this local sensitiveness in which hemisphere the affection is situated, for when there are no local symptoms such as paralysis of the cranial nerves, paralysis of the extremity, or a difference in the reflexes, etc., this may be doubtful.

In conclusion, we may state the following: In every case of cerebral affection the sensitiveness of the skull to percussion should be tested, and this test

is valuable, but we do not base our diagnosis wholly upon it; on the contrary, we come to a definite conclusion only when the results thus obtained are in agreement with the other symptoms.

Of late, regular percussion of the skull, i. e., differentiating the note obtained by its percussion, has attracted much attention. We place one finger upon the skull and percuss with another finger, the patient keeping his mouth closed. Gilles de la Tourette and Chipault, for example, report that on percussion of the skull the note is shorter the thinner the skull, and *vice versa*; therefore, the note obtained by percussion of the occiput is shorter than over the frontal bone. In the child it is clear, in the adult dull, and it again becomes clear in the aged. Sometimes there is a circumscribed dull note over a brain tumor; but this is an isolated occurrence. A tympanitic note, particularly when the bones are very thin, sometimes indicates that a brain tumor is present; however, as was shown by Oppenheim and Bruns, in the nursling a tympanitic note is physiologic. A significant finding is the cracked pot sound. This is always evoked when the continuity of the skull is interrupted, and it is therefore physiologic in the child as long as the sutures remain open. Under pathologic conditions it may be found in hydrocephalus or in brain tumor, provided these have led to an opening of the sutures, also, as Gilles de la Tourette and Chipault report, in fractures of the skull. This cracked pot sound may also be circumscribed, sometimes in the vicinity of the tumor, but also at a distance from it as in the case of Wollenberg: With a tumor over the right occipital lobe, the cracked pot sound was elicited over the left portion of the frontal bone. We therefore see that in the diagnosis of cerebral processes the results of actual percussion are quite scanty; and it is only under especially favorable circumstances that we obtain valuable data. Nevertheless, it must be borne in mind that in doubtful cases any positive symptom is of value.

In pursuance of our plan **auscultation** follows percussion. Normally in the adult this is usually without result, i. e., no endocranial murmurs are heard. In infancy the case is different. In nurslings and small children in whom the fontanelles are not closed, as well as in cases of rickets, a systolic murmur is said to be present. Oppenheim also heard this murmur in an adult with marked anemia. In pathologic conditions auscultation of the skull is under some circumstances of great value. Murmurs synchronous with the pulse have been observed in arteriosclerosis, and in an adult case of acquired hydrocephalus by Fuchs; in the latter case the murmur apparently originated in the ear and was perceptible to the patient. We know that these subjective murmurs are very distressing to the patient and difficult to control therapeutically. Sometimes the murmur can be heard by placing the ear upon the head, or even at a little distance from it. This is the case with tumors very rich in vessels or tumors which press upon a large vessel, especially if near the surface of the skull; rhythmical murmurs synchronous with the pulse are then heard, and most distinctly when the patient holds his breath. They are most common in aneurysm of the basal cerebral arteries, for example, in aneurysm of the internal carotid and the vertebral, sometimes even in the form of musical murmurs which are audible at a considerable distance. Of course, in such cases the murmur disappears if we press the common carotid artery. The point at which this murmur is most distinctly heard sometimes

corresponds to the seat of the aneurysm; but the presumable locality should coincide with the other symptoms. The findings must indicate a tumor, therefore a progressive affection which limits space, before we can make a diagnosis of aneurysm.

In utilizing the history, diagnosis may go even further, as is proven by a case Karplus observed in which, accompanied by severe pain, a musical murmur appeared which was noticed by the patient, but which was also objectively heard over the left anterior half of the skull, was isochronous with the pulse, and disappeared on pressing the carotid artery. Karplus diagnosticated rupture of an aneurysm of the internal carotid artery in the cavernous sinus, and had the common carotid tied whereupon the murmur disappeared: The autopsy (death occurred in consequence of consecutive softening) showed the correctness of his reasoning.

We now turn to a method of investigation which, although of recent date, has become of great importance—**lumbar puncture.** Originally employed for therapeutic purposes, this method invented by Quincke soon became very valuable in diagnosis because it permitted the product of secretion of the central nervous system, or of its membranes, to be investigated directly.

The technic of lumbar puncture and its results under normal conditions have been described in an article on the subject in this volume (see Lumbar Puncture, by Quincke) and we at once turn to pathologic conditions and the diagnostic employment of lumbar puncture. The most frequent change noted is an increase of pressure; it may appear when the composition of the fluid is otherwise unaltered, while in other cases there is a change, both macroscopical and microscopical, in the cerebrospinal fluid. Increase in pressure often becomes so great that the fluid which would normally exude drop by drop actually flows out in a stream. On estimating the pressure in these cases, it has been found to equal 300–500 mm. of water, and even 1,000 mm. (normally 40–150 mm.). Such simple increase of pressure is found in hydrocephalus, with rare exceptions in cases in which the communication of the ventricles with the subarachnoid spaces of the spinal cord is disturbed, in brain tumor, and in abscess, here also with the above mentioned limitations. Lumbar puncture has enabled us to recognize a new pathologic picture invariably accompanied by greatly increased pressure of the arachnoid fluid, and which Quincke has designated as *serous meningitis.* In this condition a rapid increase of the ventricular fluid is said to take place, so that it also includes cases of acute acquired hydrocephalus. The affection sets in primarily with symptoms of cerebral pressure, and if there is fever, tuberculous meningitis or epidemic meningitis is closely simulated; in non-febrile cases the symptoms resemble those of brain tumor. Etiologically the cases vary; the disease may arise spontaneously or after infectious diseases or intoxications; its occurrence in purulent otitis, particularly on account of the difficulty in diagnosis, must be especially mentioned. What, however, stamps this serous meningitis characterically is its curability, either spontaneously or after lumbar puncture; for here, as well as in some cases of chronic hydrocephalus, lumbar puncture is of great therapeutic value, which originally was not expected to be the case, or at most only transitorily. In a pathogenetic and pathologico-anatomical respect the subject of serous meningitis still requires elucidation. In a symptomatologic sense, however, we not infrequently observe

its clinical picture. Many a case formerly obscure, many cases of tuberculous meningitis apparently cured, of cerebral tumor, etc., belong in this category. Increase of pressure is also found in certain forms of severe headache, especially in chlorosis, in sinus thrombosis, and in spasmodic attacks, of epilepsy, uremia, etc.

Increase of pressure is often accompanied by changes in the appearance and composition of the fluid, and these we must briefly discuss. The most conspicuous admixture is with blood, which may, however, be accidental. Otherwise, provided hemorrhage has occurred in the subarachnoid space, pure blood is not found but it is admixed with the subarachnoid fluid; for example, after trauma of the skull or vertebral column, in hemorrhages of the hemispheres which have ruptured into a ventricle, or in hemorrhage upon the surface, in ruptured basal aneurysms, and in pachymeningitis which is not encapsulated in the subarachnoid space. By diffusion of hemoglobin from the surrounding area without a direct macroscopic admixture of blood, the fluid may show a slight yellow discoloration, and erythrocytes may be recognized with the microscope, or hemoglobin with the spectroscope.

The findings in various forms of meningitis are of great diagnostic significance; the macroscopic and microscopic composition of the fluid, its chemical alterations, and its bacteriologic investigation may furnish valuable data as to the etiology. The appearance of the fluid varies in different forms of meningitis; sometimes it appears to be absolutely clear; in tuberculous meningitis, as shown by Lichtheim, Schiff and others, the subsequent formation of a coagulum from this clear fluid is characteristic. Besides, as some authors have demonstrated, this occurs also in rare cases of brain tumor, while, according to Orglmeister, it may be absent in tuberculous meningitis. In other forms of meningitis small coagula are found even in the native fluid, some so large that they may occlude the cannula. Finally, the fluid may show turbidity from the slightest degree to actual pus, as, for instance, in some cases of epidemic cerebrospinal meningitis. The chemical examination of the fluid obtained by puncture may enable us to reach certain conclusions; for instance, in different forms of meningitis the amount of albumin is generally increased to 3 per 1,000, while a decrease in the amount of reducing substance does not form a reliable guide.

The microscopic examination of the fluid obtained by puncture is of especial importance and is best made after centrifugation. Normally only isolated leukocytes are found, but in meningeal processes their number is greatly increased, and in acute processes the polynuclear, in subacute and chronic cases the mononuclear, forms predominate or are exclusively present. This *cytodiagnosis* of the fluid in lumbar puncture, as shown by many recent investigations of French physicians, has acquainted us with a far more interesting fact all the details of which are not as yet clearly defined. Widal, Siccard and others—among German authors so far only Schoenborn, Frenkel and Mayer have studied the question—have shown that in syphilitic and so-called metasyphilitic processes of the nervous system, above all in tabes and paretic dementia, an increased number of lymphocytes has been found in the cerebrospinal fluid, occasionally also large mononuclear elements. While normally with the microscope only three to four lymphocytes are found in the fluid, under the previously mentioned conditions this number is increased to

30, 40, or even more. Many French authors regard this symptom as an important guide in the diagnosis of tabes, as it may appear very early; for example, in cases in which there is only reflex rigidity of the pupil, where, therefore, the diagnosis of tabes or paralysis cannot be made with certainty but only with some degree of probability. They refer this increased number of lymphocytes to an irritative state of the meninges which accompanies tabes and paresis, and produces a mild or severe meningitis. This lymphocytosis also occurs but to a lesser degree in metasyphilis without nervous symptoms, a fact which sometimes nullifies its diagnostic importance; in the cases of isolated reflex pupillary rigidity, above mentioned, this condition is especially vitiated, for we know that reflex pupillary rigidity without the development of tabes or paralysis may occur as an isolated symptom in persons who have had syphilis. Lymphocytosis is also found in chronic alcoholism and in certain cases of herpes zoster, in which it is produced by the same cause as in tabes or paresis. On the other hand, it may be absent in tabes or paresis (Déjérine). In this lymphocytosis, therefore, we are dealing with a new condition which even to-day is worthy of notice, but many phases of which still require elucidation and explanation.

A case published by Hartmann shows that under some circumstances lumbar puncture may reveal other conditions; for instance, the discharge of a cysticercus permitted the diagnosis of cysticercus of the brain which otherwise would not have been thought of.

There is another modern development of diagnosis: *Sero-diagnosis*—the agglutination of tubercle bacilli by the cerebrospinal fluid in tuberculous meningitis—which was attempted by Donath, but as yet has given no results. That sero-diagnosis may lead to errors, even when all precautions have been observed, is shown by two cases of tuberculous meningitis in which the fluid from lumbar puncture gave the Widal typhoid reaction.

The *demonstration of pathogenic microörganisms* in the fluid obtained by lumbar puncture, either from the sediment obtained by centrifugation or from the coagula by staining methods, or, finally, by inoculation upon culture media or in animals has much greater practical value and is sometimes decisive. Among the organisms so far found is the tubercle bacillus—according to recent reports, with proper methods this is almost invariable in tuberculous meningitis, particularly when the coagula is employed for staining—also the meningococcus intracellularis, the pathogenic agent of cerebrospinal meningitis, pneumococcus, streptococcus and staphylococcus, the typhoid and coli bacilli, the influenza bacillus, etc. Notwithstanding the presence of microörganisms the fluid may be clear, as in the case of tuberculous meningitis. More frequently, however, it is slightly turbid, or even appears purulent. On the other hand, with a distinct purulent appearance the fluid may be sterile, apparently because the pyogenic microörganisms have disappeared; we know, for example, that microörganisms directly and experimentally introduced into the circulation and thence passing to the spinal cord or the subarachnoid fluid may disappear with relative rapidity after a few days. This experience was gained in producing myelitis experimentally, and in part explains why microörganisms are so rarely demonstrated in infectious or post-infectious forms of myelitis. It also explains many of the very common relations between acute infectious diseases and the grave nervous symptoms which accompany

or follow them, above all, those of a cerebral nature. We see that acute infectious diseases, whether caused by microörganisms or by a so-called secondary infection, perhaps by staphylococci or streptococci, may produce severe, even purulent inflammatory processes in the nervous system, particularly in the meninges, as well as in other parts of the body, while in other cases the presence of microörganisms may cause merely an irritation without a true inflammatory process. But, of course, all such cases are not directly due to microörganisms; often their toxins cause the condition.

Having briefly described the findings in lumbar puncture, we now turn to its differentio-diagnostic importance, i. e., let us consider how and when we may base our diagnosis upon the results of lumbar puncture, and how we can make a differential diagnosis. Lumbar puncture, we must state at the outset, is especially valuable in the differential diagnosis, difficult and often impossible from the clinical symptoms, of abscess and meningitis, especially purulent meningitis. This question arises in the cases with cerebral complications from disease of the ear, also in traumatic metastatic processes with cerebral symptoms in the course of infectious diseases, in which conditions encephalitis and cerebral tumor must under some circumstances be considered in the differential diagnosis. While in abscess which has not ruptured nor led to secondary purulent meningitis, also in tumor and in encephalitis, lumbar puncture shows an increase of pressure, yet clear fluid is obtained, in purulent meningitis we find the changes mentioned above; macroscopically there is turbidity, an apparent coagula, upon microscopic investigation many cells are noted, above all polynuclear leukocytes which often furnish an etiological explanation by the demonstration of microörganisms. Tuberculous meningitis also must be considered, and may be thus determined. It is true that the results of lumbar puncture are not absolutely conclusive. There are cases of purulent meningitis in which the process is at first local, the collection of pus in the meninges becomes encapsulated; in these, therefore, lumbar puncture may reveal clear fluid. On the other hand, to many surgeons purulent meningitis is no longer a *noli me tangere,* for here also favorable results may be expected from the early evacuation of pus. At all events, in these difficult cases lumbar puncture makes our diagnosis more certain, and indicates the proper therapy.

The diagnosis of tuberculous meningitis becomes more positive by lumbar puncture, for tuberculous meningitis, especially in the adult, by no means always runs a typical course, showing the familiar stages of irritation and paralysis so frequently represented in text-books. I do not refer solely to the cases in which acute cerebral focal symptoms, paralysis, aphasia, etc., appear, or to cases with long intermissions, etc. The question, so long disputed, whether tuberculous meningitis is curable, has also been solved by lumbar puncture, for with each succeeding year the number of recoveries in cases in which the affection was proven by the finding of tubercle bacilli in the fluid steadily becomes larger; in some cases this demonstration was subsequently verified by autopsy findings.

To demonstrate the importance of lumbar puncture, I must refer to what was previously said in regard to serous meningitis, many an obscure case of which has been made clear by lumbar puncture. Hemorrhages, particularly of traumatic origin, or cerebral hemorrhages with external rupture or rupture

into the ventricles, may also be diagnosticated, and thus, under favorable circumstances, the differential diagnosis between hemorrhage and softening, which sometimes can only be assumed, becomes certain.

Much more might be said of lumbar puncture, but the foregoing is sufficient to show that it enables us to determine very interesting and even important facts, and that it is a method to be employed whenever possible.

In conclusion I must devote a few words to a method frequently resorted to by internal clinicians in the differential diagnosis of purulent processes, a method which may also be of use in nervous affections when the differential diagnosis between purulent processes (abscess and meningitis) and non-purulent ones (encephalitis, tumor, etc.) is perplexing. This is the **examination of the blood**. It is well known that in suppuration there is an increase of the polynuclear leukocytes; the iodin reaction, which depends upon the amount of glycogen produced by the leukocytes during suppuration, may also be of value. Raymond recently diagnosticated local secondary suppuration after traumatic pachymeningitis—the patient was trephined—by a leukocytosis which subsequently occurred; the case was cured by a second operation.

If I were asked to state comprehensively the gist of this article, I should say that it set forth the methods which, in common, form physical, chemical, or microscopical aids, by the help of which, combined with the consideration of the neurologic symptoms, it is possible for us to arrive at more certain conclusions, and wherever possible also to define the nature of the underlying process. Partly new, hence varying in results, these methods have nevertheless greatly facilitated the diagnosis of cerebral affections. On continued use we may hope that these methods may become more certain and reliable.

We have not yet reached the goal of a clear and precise diagnosis of diseases of the brain, but we hope that this will be possible in the not far distant future, for the path of advance in our science is not marked out by well laid plans based upon what has already been acquired. What seems most distant may suddenly become attainable, and advancing knowledge may come with rapid strides.

LUMBAR PUNCTURE

By H. QUINCKE, Kiel

PUNCTURE is performed for diagnostic or therapeutic purposes when pathologic collections of fluid exist, or are presumed to exist, either in pathological and newly formed cavities, such as cysts and abscesses, or in anatomical cavities, such as those in the joints, the pleura, the peritoneum, etc. The cavities of this latter group normally contain only capillary layers of fluid between the serous layers; when puncture reveals fluid, this indicates a pathologic increase. Such cavities are similar to the spaces in the subcutaneous cellular tissue from which in anasarca decidedly large amounts of fluid may sometimes be removed by puncture and drainage.

There is only one cavity of the body *which in the normal state contains a considerable amount of fluid, the cerebrospinal cavity.* The central nervous system is so suspended in the fluid that its surface nowhere touches the osseous capsule. Even in a healthy person some of this fluid can invariably be obtained if the cerebrospinal cavity is punctured at a suitable point in the lumbar portion of the vertebral column.

(1) For the better understanding of such puncture and its action and results I must refer briefly to the **structure of the membranes of the central nervous system.**

The *dura mater*, the fibrous covering of the central organ, is closely adherent to the cerebral portion of the inner surface of the skull, and at the same time forms the periosteum of this bony surface, while in the vertebral canal the dura mater forms a loose sac resembling a hollow cylinder, and separated from the inner periosteum and the fibrous walls of the vertebral canal by a loose connective tissue which contains many large venous plexuses. At the height of the second or third sacral vertebra the sac of the dura terminates abruptly, narrowing in the form of a tenpin.

The delicate covering of the central nervous system, the *leptomeninx*, is formed by two membranes designated in descriptive anatomy as the *pia* and *arachnoid*; in most areas these may be detached by a gross anatomical method from each other, but in reality they form the internal and external boundaries of the delicate membrane of the brain, the two layers being united by loose connective tissue. The internal, the pia, rich in vessels, the connective layer of the delicate membrane of the brain, covers the surface of the spinal cord just as it does the brain, and follows the convolutions on the upper surface of the latter deep into the furrows, while the arachnoid follows only the general surface, enclosing the brain like a loose sac, covering the fissures like a bridge,

and being in close contact with the convolutions only at their convexities. These layers, the pia and arachnoid, are united by a very delicate web of connective tissue strands with wide communicating spaces, the *subarachnoid spaces,* between. On the surface of the convolutions the connective tissue strands are short, the subarachnoid spaces very shallow; in the furrows the latter are deep, the connective tissue strands longer and more loosely arranged. The latter is also true of the under surface of the brain, the arachnoid extending over its varied convolutions as a smooth, tense membrane, so that here we find wide, communicating, subarachnoid spaces (cisternæ).

While, therefore, the pia is closely adherent to the brain and spinal cord, the arachnoid forms a sac-like covering separated from it by the subarachnoid fluid. This sac is continuous with the sac of the dura mater; its external surface and the internal surface of the dura lie in close contact, while in the cavity of the skull they are separated from each other by a thin layer of fluid. This space in the skull between the dura and arachnoid, the *subdural space,* is a few tenths of a millimeter in thickness, while in the cavity of the cord it is exceedingly thin, and becomes apparent only in consequence of pathologic accumulations.

Autopsy does not accurately show what the relations are during life, since there is no turgescence due to the pressure of the blood and secretions. The varying relations of the subdural space in the cavity of the skull and in the spinal cord can be determined with certainty only at operations and in vivisections. As Hitzig has demonstrated, the subdural fluid in the skull is absorbed soon after death by the substance of the brain, so that even a few hours after life is extinct the cerebral subdural space seems no longer to exist.

The diagram on the opposite page, modified from the "Topographical Anatomy" of Merkel (page 78, Fig. 38), shows these relations. The subarachnoid spaces, the ventricles of the brain which communicate with them, and the aqueduct of Sylvius are blue, the subdural space between the dura and arachnoid is red, but in the cavity of the spine where the arachnoid and dura are in closest contact, this normally no longer exists.

The subdural space may be regarded as analogous to the pleural or peritoneal cavity; the fluid it contains may be estimated at a few cubic centimeters. The greatest bulk of the cerebrospinal fluid ("subarachnoid") is found in the connective tissue meshes of the delicate membrane of the brain which Henle has accurately described morphologically as "the physiologic, water-seeking, connective tissue." The meshes of this tissue throughout the brain and spinal cord communicate with each other; and this continuity which normally exists is such that, aside from special conditions, pathological processes from the surface of the brain are very readily transmitted to the spinal cord and *vice versa,* not quite so smoothly as from the serosa of the thoracic and abdominal cavities, but much more readily and more rapidly than in the normal, or even in the dropsical, subcutaneous cellular tissue.

(2) **The fluid in which the central nervous system** almost seems to float, is in many ways protective.

Its displacement with alterations in the position of the body which might mechanically damage it prevents marked changes in the form of the spinal canal; by its deviations, by its being subjected periodically to a greater afflux and efflux notwithstanding the slight relaxation of the capsule of the skull, alterations in the amount of blood and in the circulation of the blood through

the brain become possible; at the same time the narrowness of the outlets prevents this change from being rapid or sudden.

The cerebrospinal cavity contains the following elements: (*a*) Firm tissue which largely forms the central nervous system; (*b*) Blood; (*c*) Cerebrospinal fluid. Of these component parts the tissues undergo a change in volume to the least extent and only after a long time.

The amount of blood depends upon the general and local circulation. In this respect the most important factor is the height of the blood pressure in

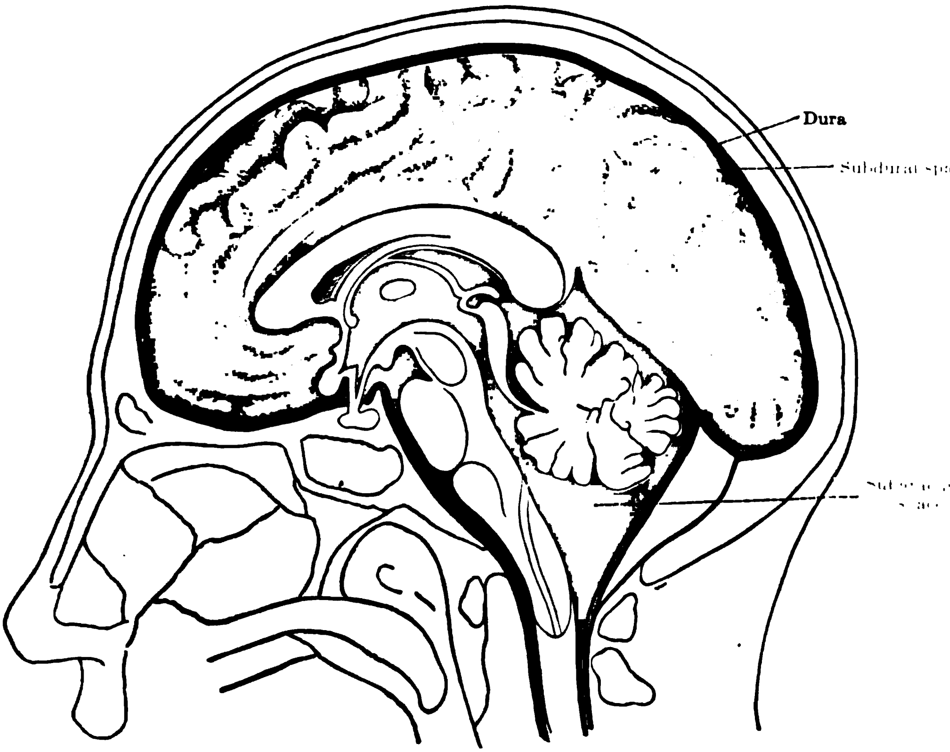

Fig. 127.

the aorta and vena cava, the caliber of the arteries which enter the cerebrospinal cavity, of the veins which emerge from it, and the caliber of the cerebral and spinal cord vessels; the latter no doubt varies because of general conditions and on account of the richness of the muscular coat of the arteries of the brain. It is immaterial whether vasomotor activity, or the composition of the blood or the hemic requirements of the nervous substance are the determining factors. It is obvious that the amount of blood in the central nervous system and in the cerebrospinal cavity must depend on the caliber and number of the afferent and efferent vessels, and that this is subject to great and rapid variation, provided there are no other modifying conditions.

The third element in the cavity, the cerebrospinal fluid, serves mainly as compensation for the first and second. Its amount is adapted to the varia-

tions in volume of the brain substance; when this is decreased it increases as meningeal edema or ventricular dropsy (hydrops ex vacuo); more rare and less conspicuous is its decrease in neoplasms and hemorrhages.

The *regulating and controlling effect of the cerebrospinal fluid* on the variations in the amount of blood, especially within the skull, is much more important. It is true the conditions here are complicated, for this fluid is itself a *secretion from the blood* and, moreover, is secreted from diverse areas: From the choroid plexus of the ventricles, from the subarachnoid spaces on the surface of the brain and the spinal cord and, finally, from the lymph tracts of the central nervous system which empty into it.

The *outlet* for the cerebrospinal fluid is by means of narrow channels through the villi of the arachnoid to the venous sinuses, thence through the subdural space and the dura to the lymph vessels of the neck and throat (possibly also to the nasal mucous membrane and the labyrinth of the ear). and probably along the sheaths of the nerves of the brain and spinal cord.

The cerebrospinal fluid, therefore, resembles a stream which receives tributaries from several sources, the importance of which, even physiologically, may vary, and of which pathologically sometimes one, sometimes another, may be paramount. As an example of this variation I may cite the action of ventricular dropsy in obliterating all of the subarachnoid spaces, of acute meningeal edema upon normal ventricles, and of diffuse edema of the brain. The increased exudation of fluid (whether in one of the previously mentioned forms or in their combination) may decidedly affect the caliber of the blood-vessels, so that the amount of blood they contain is decreased.

(3) The general **conditions under which the brain receives its supply of blood and nutrition** are peculiar from the fact that the organ is enclosed in a bony capsule which yields but little to pressure, although it contains spaces and has outlets which have a wide external point of communication in the foramen magnum. The skull of the child, prior to complete ossification of the sutures, still yields to a certain extent, but the capacity of the cranial cavity of the adult is practically unchangeable, and consequently the amount of blood it contains depends upon the position of the body. If a greater volume of blood flows to the brain and a dilatation of its vascular tracts therefore becomes necessary, this can only be brought about by a corresponding decrease in the amount of cerebrospinal fluid, either by its actually flowing into the veins and lymph vessels, or by its passing into the subarachnoid spaces of the yielding spinal sac; the latter mode of compensation would appear to be much more rapid, and therefore, in the frequent and slight functional variations in the amount of blood in the brain, is probably the usual process. The capacity of the spinal dural sac is limited, although there are variations due to its natural firmness and the resistance of its external surfaces. Of these surfaces the firmness of the bony and ligamentous vertebral canal and the varying pressure of the peridural venous plexuses may be especially mentioned.

While the conditions regulating the volume of blood in the cavity of the skull may be compared with those of the medullary cavity of a bone, in the spinal cord this bears a certain but imperfect analogy to the process in organs which, like the testicles or the kidneys, are surrounded by a firm fibrous capsule. A crude idea of these conditions is conveyed by an inverted glass bulb, the neck of which is turned downward and connected with a wide rubber tube closed at the end. The volume of fluid within the

skull (the bulb) is the same under all circumstances. The pressure which it sustains depends upon its fulness and the elastic tension of the dural sac (the walls of the tube). This pressure is the same at all points upon the same horizontal plane, but different at varying heights because *hydrostatic pressure* must be added to the *elastic pressure.* It varies, therefore, at a given point with the position which the body assumes. If the distance between the floor of the bulb and the end of the tube is 60 cm., on placing the model in a vertical position the pressure at the lower end of the tube will be respectively + 60, + 80, + 40 cm., if the pressure upon the floor of the bulb is respectively 0, + 20, — 20. With a horizontal position of the body the pressure at both points is the same, and with an inverted vertical position (standing upon the head) it will be 60 cm. less at the end of the tube than in the bulb.

(4) **The amount of pressure in the cerebrospinal cavity** depends in the concrete case upon the following factors: 1. The mass of tissue present (central nervous system + pathologic products); 2. Upon the blood-pressure; 3. Upon the amount of cerebrospinal fluid secreted.

The effect of blood-pressure is due to the arterial pressure in so far as this is not conveyed by the walls of the vessels and by the tissues, also by the extremely varied pressure in different veins. The result is designated by Pfaundler as "*transmissible blood-pressure.*"

The effect on the cerebrospinal fluid of the pressure of secretions is further modified by the rapidity of secretion and the caliber of its outlets.

In order to understand these conditions it is absolutely necessary for us to bear in mind that the amount of fluid in the cavity is not invariable but subject to continued variations, and that the three factors previously enumerated alternate with one another in a certain rhythmic measure. By their combined action pressure is exerted upon the internal surface of the cerebrospinal cavity, and its walls so far yield that the spinal dural sac becomes distended and tensely elastic. We may designate pressure produced in this way as "elastic pressure." Because of the free communication of the subarachnoid spaces with each other, this elastic pressure is equal throughout the cerebrospinal cavity, and, except under special conditions, alterations in pressure in one part of the cavity must always, even though somewhat modified, be transmitted after a time to the entire tract; thus the increased pressure transmitted from the contents of the skull to the spinal dural sac (which as an elastic supplement simultaneously forms a safety valve) may be compared with that of the air chamber of a pressure syringe.

(5) In man the **only point at which we may gauge the pressure in the cerebrospinal cavity, and perhaps decrease it** by evacuating some of the fluid, is *the spinal portion of the dural sac.* As the spinal cord of the adult with its conus medullaris extends only as far as the top of the second lumbar vertebra, below this we find only the loose bundle of descending nerve roots, the cauda equina, the individual filaments of which are somewhat motile and extend through the cerebrospinal fluid with such wide interspaces that, on inserting a cannula at this point, it freely enters the fluid.

At most points above the second lumbar vertebra, the subarachnoid space is wide enough (1 to 3 mm.) to permit the introduction of a cannula. Practically, however, we might either insert it beyond the dura and readily injure the spinal cord or by accidentally displacing the cannula even to the extent of 1 mm. we might glide beyond the dural sac. Therefore, puncture of the spinal sac can be practised only in the lumbar portion, and to make this clear, instead of the more general expression *spinal*

puncture, I have chosen the term *lumbar puncture.* Of the subarachnoid spaces of the brain, those which lie between the cerebellum and the medulla, on account of their width and position, permit the introduction of a cannula. In animal experiment this has been done by exposing and puncturing the membrana atlanto-occipitalis; in man such a puncture through the skin is impracticable on account of the close proximity of the vital centers. The pressure within the skull cannot be determined by the introduction of a cannula, even in animal experiments, owing to the narrowness of the subarachnoid spaces, and even if, as v. Leyden has proposed, a pressure tube is screwed into the skull through a trephine opening, the measure of pressure may readily be disturbed by a protrusion of the brain.

Puncture of the lumbar arachnoid space (**lumbar puncture**) may be readily performed in man and without danger; the needle should be inserted through the skin and the muscles of the back into the soft parts between the arches of the vertebræ. I first advised lumbar puncture as a therapeutic measure, but it has at the same time given us an insight into the causes of pathologic processes which take place in the cerebrospinal cavity, and into the pressure condition of this *cavity which,* as mentioned above, *embraces both elastic and hydrostatic pressure.*

The *height of the pressure,* which we should never neglect to ascertain when performing lumbar puncture, is estimated as follows: The cannula, after insertion into the subarachnoid space, is attached to a small rubber tube with a glass tube at its end, and this is raised perpendicularly just as soon as the first drop of fluid appears. The fluid is received in this vessel, and its height, measured vertically above the point of puncture, indicates the pressure existing in the subarachnoid space. If lumbar puncture is performed in the horizontal lateral position, we generally estimate the "elastic pressure," not only as it exists at the point of puncture, but approximately also within the cavity of the skull, for with this position of the body the difference in height between the seat of the puncture and the highest point of the skull is very slight, and the proportion of hydrostatic pressure to the entire pressure estimated is also very low. The conditions are different with an erect position of the body; here the vertical difference between the vertex of the skull and the point of puncture amounts in the adult to about 60 cm. In this position we invariably find higher values in lumbar puncture because of the increase of hydrostatic pressure—of course, this is not so great as to correspond with the altered position and the height of the vertex of the skull.

Pfaundler has made comparative estimations on this line; he first measured the pressure during lumbar puncture of children in a sitting posture, then in a recumbent position, and in the former position he determined the vertical difference between the point of puncture and the vertex of the skull. In children whose cranial bones had united the difference in these pressure values averaged only 21 per cent. of the ideal, hydrostatic components (H); in children whose fontanelles were open, 33.9 per cent. From this it is evident that the system of subarachnoid spaces does not act like a wide communicating tube, but that some of this pressure (certainly, some time after a change of position has occurred) is borne by the walls, and is, perhaps, also compensated for by the dissemination of fluids to other higher tracts.

The hydrostatic pressure in children with hydrocephalus was somewhat greater (namely, 27 to 53 per cent.); in these cases the spinal cavity is usually more or less dilated.

Krönig found the lumbar pressure in adult healthy persons who were in a horizontal position to be 125 mm., in a sitting posture (where muscular tension adds something to the increase in pressure) 410 mm. If the difference from the point of

puncture to the vertex of the skull (about 60 cm.) is expressed by H_1, and that from the foramen magnum (about 40 cm.) by H_2, the difference (285 mm.) equals 48 per cent. of H_1 and 71 per cent. of H_2.

(6) **The position of the fluid in the pressure tube almost invariably shows variations** which may be ascribed to different causes. Most distinct is the *influence of respiration*: The pressure falls with inspiration and rises with expiration, being greater after prolonged respiratory movements; the difference amounts to 20 mm. More slight (1–4 mm.) and less invariable are the *variations in pressure due to pulsations of the heart.* In some cases there is a rise which is synchronous with the systole of the heart, and in others a fall of the fluid; I am unable to state the amount of this variation. In addition to these respiratory and arterial variations others are sometimes observed which amount to from 10 to 30 mm., and vary in duration from 10 to 30 seconds. The cardiac systolic and the respiratory variations in the pressure of the arteries and veins are transmitted throughout the entire course of the cerebrospinal cavity to the subarachnoid fluid; in lumbar puncture the narrowness of the spaces scarcely permits those generated in the cavity of the skull to be noticeable in the pressure tube; the variations in pressure which are here visible are probably produced mainly from the spinal cavity, and especially by the varying fulness of the voluminous, peridural, venous plexuses. Great increase in pressure (several hundred millimeters) is caused by forced expirations as in cough, straining, or screaming; the pressure is still further increased by increasing muscular resistance, particularly in epileptic spasms. Nawratski and Arndt measured the pressure in the tonic stage and found it to amount to 750–870 mm., while previously and subsequently it was normal. Muscular action for the development of this brief but marked increase in pressure may be directly transmitted through the ligaments and other tissues to the spinal dural sac.

If the continuity of the spinal subarachnoid space be interrupted at any point, the cardiac and respiratory variations will probably be lessened, since they are only transmitted to the lower portion of the dural sac. Henneberg believes that this may be utilized in the differentiation of myelitis and compression myelitis.

In practice it is, of course, necessary to determine the height of the cerebrospinal pressure while excluding all of these disturbing factors which may increase it, but the numberless influences which must here be considered probably account for the variations in the height of the normal pressure in lumbar puncture. In our experience, *normal pressure* may vary between 40 and 130 mm. water; 150 mm. is the limit, and 200 must always be regarded as abnormal.

With cerebrospinal fluid of a low specific gravity, 1.007–1.010, the height of the column of fluid may be read off accurately as water pressure; it is much easier to use these figures directly than to calculate them as mercury pressure (dividing by 13.6). The average blood-pressure in man (120 mm. mercury) amounts to 1,620 mm. water.

(7) Under pathologic conditions the **cerebrospinal pressure** frequently **rises**, and this is of importance for the function and nutrition of the brain. The conditions within the skull are decisive for the height of the pressure, since here are the principal secretions and here also their chief outlets. The

spinal cavity, so far as we know, is as little concerned actively in the increase of pressure as the spinal cord appears to be susceptible to it.

Pathologic processes which may produce pressure within the brain are the following:

(*a*) *Proliferation of tissue* which decreases space, tumors of the brain substance or those in the soft or hard membranes which compress the organ, inflammatory swelling of different areas of the brain, and fragments of bone which mechanically compress it;

(*b*) *Effusions of blood* in or between the membranes of the brain or in the brain substance;

(*c*) *Purulent and serous exudates,* generally of inflammatory origin. The more liquid, hence the more displaceable these are the more likely they are to produce general pressure or local symptoms; they are frequently combined with the processes enumerated under 1 and 2, which at the same time increase the *normal secretion of the cerebrospinal fluid.* The *principal source* of the latter is evidently the *venous plexuses* of the ventricles of the brain; thence the fluid finds its way through the aqueduct of Sylvius and the foramen of Magendie also through spaces in the tissue, along Galen's veins, and laterally past the tonsils of the cerebellum into the subarachnoid spaces. Hypersecretion of the venous plexuses is generally to be regarded as *inflammatory.* Sometimes *microörganisms* are found in this secretion, for example, tubercle bacilli, but more frequently they are absent and a *chemico-toxic action* must be assumed. Accordingly we may separate ventricular meningitis into simple and bacterial forms. Serous exudates sometimes form acutely but are so transitory that they cannot be regarded as inflammatory but of *angio-neurotic* origin.

In compression of the vena magna Galeni (by tumors, etc.) *stasis hyperemia* arises in the venous plexuses of the three anterior ventricles, and this causes increased transudation.

When the aqueduct of Sylvius or the foramen of Magendie is obliterated, even though the secretion be normal in amount, the ventricles are dilated because of the *stasis* of the secretion and hindrance to its *outlet.*

The effect of profuse serous transudation is most obvious in the simple hydrocephalus of children; here the sutures and fontanelles which have not yet closed permit us to recognize the pressure existing; if the exudation is permanently increased, these appear tense and stiff, but again become flaccid after the pressure is relieved.

In internal hydrocephalus the lateral ventricles are most markedly, and the fourth ventricle the least, distended; when the aqueduct of Sylvius is closed the distention may be limited entirely to the three anterior ventricles.

In internal hydrocephalus the contents of the ventricles of the brain, which may normally be estimated at from 20 to 30 cc., may amount to several hundred or even to more than a 1,000 cc. The ventricular dropsy due to hypersecretion presses upon the brain, and forces it to the inner surface of the skull. The convolutions are flattened and the fissures obliterated.

The *subarachnoid spaces* on the surface of the brain are implicated in the bacterial inflammations of the pia, especially so in the purulent ones, and here serous exudations are found in the form of meningeal edema; but the number of observations proving the frequency with which these "edemas"

accompany an increase in intracranial pressure is insufficient. Some cases, for example in uremia, in plague, and in influenza, may be of toxic or angioneurotic origin; and in the transitory, rapidly cured cases in which transudation causes an increase in pressure, we cannot differentiate those of subarachnoid exudation from those chiefly of ventricular origin.

The *absorption of the fluid* into the *general circulation* might just as readily take place by diffusion into the blood-vessels of the cerebral and spinal cord membranes as by the previously mentioned outlets. In how far the former vent is to be at all considered we do not know; the preformed outlets are found particularly in the cavity of the skull just as in the organs of secretion.

Whether occlusion (other than occlusion of the ventricles themselves) may alone produce an increase in pressure is unknown to us.

On the other hand, in every case of dropsy of the ventricles with distention, from pressure of the cerebral convolutions against the skull, the subdural space, the subarachnoid spaces, the path to the Pacchionian granulations, and the remaining outlets are narrowed. Thus a new course of increase in pressure arises, and the spinal sac perhaps absorbs fluid to a greater extent than under normal conditions.

According to Bönninghaus (l. c., pages 345 et seq.), ventricular dilatation in itself ("automatically") brings about the transference of pressure from the foramen of Magendie to the occipital bone, or an acute exudation by kinking of the aqueduct of Sylvius.

Finally, as will be more minutely explained later, in enlargement of the brain by internal hydrocephalus or other causes, the foramen magnum is often closed as by a plug, and thus communication between the spinal and cerebral subarachnoid spaces is cut off. Not only the complementary spinal outlets for the liquor cerebralis, but the cavity of the skull becomes an absolutely rigid capsule which no longer acts by its yielding and elasticity as a safety valve for the spinal dural sac.

Intracranial increase in pressure, therefore, leads to a number of sequelæ which, the higher the pressure rises, make compensation increasingly difficult, and thus produce a true vicious circle. The process may be compared to the kinking and incarceration of an intestinal coil, perhaps even better to acute glaucoma, in these conditions at one time one, at other times another, factor is predominantly active.

We might extend the use of the expression "*automatic closure*" still further and include all of these active factors.

In the pathogenesis of these conditions the observations made by Nölke in the Clinic at Kiel are especially interesting and are as follows: In four cases in which, on account of a more or less complete closure at the foramen magnum, the cerebral pressure could not be reduced by lumbar puncture the ventricles of the brain were directly punctured by a small opening through the fontanelles into the skull. The fluid thus obtained invariably showed much less albumin than that previously obtained in the same case by lumbar puncture, the amount of albumin per thousand being:

	In Case I.			II.	III.	IV.
In the lumbar fluid	2.7	3.0	4.7	2	1	1
In the ventricular fluid	A trace.	A trace.		1	0.2	A trace.

There are, so far as I can see, only two possible explanations of these findings: Either communication between the skull and the cavity of the cord had been perma-

nently cut off, in which case a fluid much richer in albumin would have been secreted in the spinal subarachnoid spaces than in the ventricles—or communication existed but was incomplete, in which case the fluid secreted in the ventricles during its passage to the lumbar portion would have become richer in albumin by absorption. The latter condition is similar to that in the kidney where, in the vascular nodules of the glomeruli, a very thin fluid is excreted which develops into urine from absorption of solid constituents in the uriniferous tubules; only additional comparative investigations of the spinal and ventricular fluids in cases where there is open communication will decide this question. It is certain that the cerebrospinal fluid is not uniform, but is a mixture the origin and composition of which varies.

(8) The **rises in pressure observed in lumbar puncture** frequently equal 300 mm. of water; 500 mm. may be designated as high, 700 as extremely high pressure and is not frequently seen; 1,000 mm. is very exceptional. In children the normal as well as the pathologic figures are upon the average somewhat less than in adults—about three-quarters.

Lumbar pressure is generally in proportion to the clinical symptoms of brain pressure (headache, slowing of the pulse, vomiting, etc.), but, of course, to this there are exceptions. We know that characteristic pressure symptoms in severe and even in fatal cases may be slight or entirely absent, and in such cases the estimation of the pressure is supplementary and corrective, and of more significance in deciding upon the condition than the functional symptoms. Generally *in acute diseases there is a moderate increase in pressure with severe clinical symptoms, in chronic diseases markedly increased pressure with slight pressure symptoms*; in the latter there is also frequently a gradual compensation of the brain.

A series of estimations of pressure permits more certain conlusions than a single puncture, because extraneous influences are less potent, and we obtain some idea of the progress or decline of the disease.

While the sutures of the skull are open (open communication of the ventricles with the spinal subarachnoid spaces) the visible and palpable tension of the fontanelles is a measure of the height of the lumbar pressure; and we may convince ourselves that the latter is increased by mere external palpation.

(9) **Lumbar pressure** probably **becomes subnormal** with lowered blood-pressure in collapse and chronic debility, but this is of no practical utility, and has therefore not been investigated in man. But the *finding of low lumbar pressure* with an undoubted *existing intracranial increase in pressure* is significant. Except in those cases in which the cannula is occluded by nerve roots, by pus flocculi or blood coagula, and the observation is consequently erroneous, when the cannula directly enters the spinal subarachnoid space, and shows low pressure the passage to the upper part of the cerebrospinal cavity is wholly or partly obliterated. For example, this occurs when the subarachnoid space in the upper part of the spinal cord is obliterated by a gelatinous exudate or cicatricial connective tissue, when the foramen Magendie is impermeable, or *when the foramen magnum is occluded by the brain itself*; the latter may be the case in hydrocephalus of all of the four ventricles or when the brain mass itself is enlarged; the brain then lies in the cavity of the skull like a tense bladder. This occlusion may happen in acute serous effusions and in edema of the brain, as well as in chronic conditions (for example, in tumors, especially of the cerebellum); in these cases the tonsils are frequently seen at either side of the medulla oblongata, where with the

neighboring convolutions of the cerebellum which are elongated and in shape more or less like a cone they occlude the foramen magnum like a conical plug (Fig. 128).[1] Owing to these conditions, the subarachnoid communication between the cavity of the skull and the spinal cord is at first narrowed, but with an increase of intracranial pressure it may be periodically or permanently obliterated.

Under such circumstances, and in spite of high intracranial increase in pressure, lumbar puncture gives only slight or normal pressure values. It sometimes happens that when we begin to measure, the pressure is high, but after the evacuation of one or several cubic centimetres it falls very rapidly to normal or below; this is because there was at first only limited communication with the cavity of the skull, but after the evacuation of some of the fluid the spinal pressure declined so markedly because of intracranial pressure completely occluding the foramen magnum. Attention is drawn to this threatening occlusion by two circumstances: (*a*) When conspicuous differences in the height of pressure are revealed upon repeated punctures in spite of the other symptoms remaining the same. (*b*) When there is an extremely rapid decline in pressure during puncture. If we suspect a tumor of the posterior cavity of the skull, this observation may confirm the diagnosis. It is practically important because it shows how very cautious we should be in performing puncture, for, in those cases in which puncture has been followed by severe cerebral symptoms or even a fatal result, the occlusion spoken of was probably the cause.

FIG. 128.—TWO-THIRDS NATURAL SIZE.

Under such circumstances the fluid must be evacuated with great care. When there is a rapid decline of the pressure, or if functional brain symptoms appear, the withdrawal of fluid must be interrupted (perhaps the fluid should be returned). In these cases lumbar puncture should not be attempted, and it would perhaps be wiser to puncture the ventricles of the brain directly (compare Nölke, l. c.).

(10) **Composition of the Fluid Obtained by Puncture.**—The quantity of fluid obtained by puncture varies from a few drops to several hundred cubic

[1] Chiari has described in congenital hydrocephalus frequent and conspicuous anomalies in the form of these parts, and has illustrated these without discussing the mechanical consequences mentioned above. The same deformities, however, also occur in adults and, without being so marked, may produce complete occlusion. My colleague, Heller, has made a full report of these.

The illustration is from the brain of a girl, aged eighteen, in whom meningitis developed from otitis media, and subsequently diffuse inflammatory edema of the brain.

centimeters; large quantities are found when the effusion is clear and purely serous.

The *normal cerebral spinal fluid* is perfectly transparent and as colorless as water; it has the same appearance but is more profuse in serous meningitis and hydrocephalus. There are all transitional stages from slight turbidity up to a sero-purulent state; as a rule, pus is uniformly distributed; occasionally there are small flocculi in an almost perfectly clear fluid. On standing, the suspended cells form a sediment even though the fluid at first appeared perfectly clear; occasionally a clear fluid precipitates fibrin in the form of small flocculi, more rarely as an extensive network. In the fibrin flocculi, the deposit of which may be increased by the introduction of a small piece of cotton, scant cellular elements and microbes accumulate in large numbers.

Braunstein alone mentions a purely molecular turbidity without morphotic constituents which could be determined microscopically.

Microscopically the normal fluid shows no cells, but sometimes isolated endothelia; turbidity is usually due to pus corpuscles. In acute inflammations only polynuclear elements are found; in subacute or more prolonged processes we may find these, or even mononuclear lymphocytes (Bendix) exclusively.

The fluid obtained by puncture frequently contains small *admixtures of blood*, which, without distinctly coloring it, produce the faintest turbidity, and are only recognizable when deposited. These small admixtures of blood are due to the introduction of the needle, and generally appear with the first few cubic centimeters. This permits a differentiation from blood which is a constituent of the fluid itself, which is usually admixed in larger quantities, and distinctly colors the fluid. We observe this in hemorrhage from the subarachnoid space; for example, after injuries of the skull or of the vertebral column, in ruptured aneurysm of the cerebral arteries, and when intracerebral blood foci rupture into the ventricles.

In intermeningeal (subdural) hemorrhages (therefore in pachymeningitis) no blood is found in the fluid unless the arachnoid is simultaneously injured.

Sometimes blood flows from the cannula when its point is before or behind the dural sac in the venous plexus. It differs from the hemorrhagically stained cerebrospinal fluid by its slower flow and greater discoloration. Upon sedimenting, the cerebrospinal fluid admixed with blood is but slightly stained, on standing in a tube it deposits blood, but usually shows no, or very slow, coagulation. If only a few drops of blood are exuded, we can generally decide whether the fluid previously contained blood or it was admixed during the puncture.

Often the fluid contains *pathogenic microbes*, and up to the present time the following have been found: Tubercle bacilli, the meningococcus intracellularis, pneumococci, streptococci, staphylococci, the pneumobacillus, typhoid, coli, and influenza bacilli, bacterium aërogenes meningitidis, thick movable rods (Stadelmann), the bacillus tetragenes, and actinomyces (Sicard). Since microbes produce inflammation in the cerebral membranes, the fluid which contains them is usually more or less rich in cells, sometimes of a distinctly purulent character; but pneumococci, streptococci, and typhoid, coli and influenza bacilli may also be present in purely serous exudates. The microbes are found in the sediment, after centrifugation, and in the fibrin coagula.

Microscopic investigation and cultures do not always give like results. Where nothing is revealed microscopically, culture may sometimes give a positive result; on the other hand, microbes that have perished may be found in stained preparations but it is impossible to develop these in culture; this is particularly true of the meningococcus.

Purulent fluids are not rarely free from microbes; and, moreover, a clear fluid may be obtained by puncture, yet, further up, the pia may show purulent infiltration.

The *specific gravity* of the cerebrospinal fluid is normally 1.006 to 1.007, in pathologic cases it is sometimes higher, 1.008 to 1.009, exceptionally it may be 1.013, but these figures are inconstant and therefore of slight diagnostic importance; in the estimation of small quantities at hand, and because the temperature must be considered, the use of the scales or the areopycnometer is necessary, and the process thereby becomes laborious.

The normal cerebrospinal fluid contains *albumin*, and in chronic hydrocephalus this is 0.2–0.5 per thousand; sometimes there may be merely traces. According to Halliburton, it consists exclusively of globulin, and only when there is inflammation is serum albumin found. When pus and blood are present, the amount of albumin increases to 8 per thousand and more, and even when the fluid is clear it may amount to 0.5–2.0 per thousand, which usually indicates inflammation; in a case of hydrocephalus due to venous stasis I saw 7 per thousand of albumin.

A reducing substance which most authors regard as *sugar* is found in the normal fluid and in chronic hydrocephalus. It is inconstant in tumors; it is absent in the fluid of meningitis, but may be found in slight inflammatory conditions; therefore, the diagnostic importance of its absence is uncertain. When only a small quantity of fluid is at our disposal this test in particular is often omitted.

Fibrin coagula point with great likelihood to existing inflammation, but may also sometimes be produced secondarily by a tumor.

Hemoglobin from a transudate may give to the fluid a yellow color (in extradural effusions of blood, Rendu and Géraudel).

The cerebrospinal fluid also contains normally a diastatic ferment, cholin (Cavazzani, Grober), which is increased in paralytics and in meningitis (Gumprecht).

The virus of hydrophobia was found by Denigès and Sabrazès, and the tetanus toxin by Gumprecht, in the puncture fluid, but other authors failed to find these; the agglutinins of blood serum (for example, of typhoid) are either not found or exist only in very small quantities in the cerebrospinal fluid; the hemolysins and alexins are also absent.

The secretion of the fluid is composed of selected materials, as is proven by the fact that it contains more potassium salts than the blood serum (A. Schmidt), and that ferrocyanid does not pass from the blood, and iodin only very slowly, into the cerebrospinal fluid (Cavazzani). Sicard never saw iodin and methylene-blue pass into the normal cerebrospinal fluid. According to Widal and Monod the same condition occurs in tabes, paralysis, and inflammation due to the meningococci, while in tuberculous meningitis iodin passes from the blood to the fluid.

In uremia, in tuberculous meningitis, and in enteric fever, the fluid may have a toxic effect (Dircksen).

As a *guide in diagnosis* the pressure and the amount of albumin in the fluid should be measured with each lumbar puncture, and it should be microscopically examined for cellular elements and microbes.

(11) **Method of Performing Lumbar Puncture.**—The patient should be placed upon his left side near the edge of the bed, the body inclined a little forward, the chin as nearly as possible approaching the knees, the operator sitting beside the bed; the needle should be introduced between the third and fifth lumbar vertebral arches. When the dural sac is reached and the fluid begins to flow from the cannula, the mandrin should be removed and a hollow metal cone should be closely attached to the needle and this again to a narrow rubber tube with a glass tube at its end.

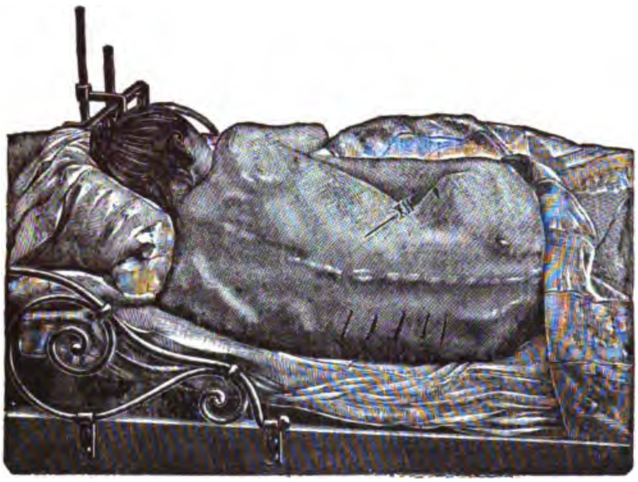

Fig. 129 *a.*

As soon as fluid appears, the glass tube is held to such a height that the fluid column is in equilibrium, when its vertical position from the point of opening will show the existing pressure; after this has been noted (naturally taking into account the previously mentioned secondary influences) the glass tube is lowered (10 to 20 mm.) below the height of pressure so that the fluid may slowly drop into a graduated cylinder. At the conclusion of the operation, the needle is withdrawn, and the fluid which is seen to exude from it is allowed to trickle into the vessel containing the rest of the fluid, and the point of puncture is closed.

Fig. 129 *b.*

I shall now describe the details.

A hollow needle, 4 to 10 cm. long and 0.8 to 1.6 mm. in thickness, serves as the *instrument* for puncture; all of my needles except the smallest sizes have a steel mandrin which fits them exactly and extends to the point, being ground so as to match the slanting surface of the needle. I believe the mandrin very useful because, if the needle does not at once enter the dural

sac and while we are moving its point about, particles of tissue may easily slip into the opening and occlude it. After passing the interarcual space the mandrin may be removed, and the position of the needle may be shifted a little until fluid appears. The handle is attached to a well fitting cone with a rubber tube and a glass tube; both should be of narrow lumen (1.5–2.0 mm.) so that too much fluid shall not escape before the pressure is read. The adjoining illustration shows the proper shape of the glass tube for the outlet of the fluid; this should be from 10 to 15 cm. long, the length of the rubber tube being 20 to 40 cm. Its length should correspond with the expected pressure, and if insufficiently long it may be extended.

To measure the pressure we use a metallic rule half a meter in length, this being held vertically with the zero figure at the point of puncture; or a metallic tape-measure may be used.

To prevent the outpouring fluid from filling the tube, Wilms attaches the cannula to a small mercury manometer by a short connecting piece. Krönig has a stop-cock on his puncture needle, attaches this by a short piece of rubber tubing to a T-shaped glass tube, one of the parts serving for the outflow, the other for measuring the pressure, and this may be lengthened if necessary. I found no special advantage in these modifications, and prefer my own more simple method, especially as it permits the reading of the pressure at any moment during the evacuation.

Needles of varying length and thickness must be chosen according to the size of the body and the thickness of the soft parts; but, as a rule, three sizes only are used. Of course, the instrument must be absolutely clean and sterilized. To prevent rust, the needle and mandrin after cleansing should be finally rinsed with alcohol and placed in glass tubes, the mandrin not being inserted in the needle. The needles of iridium are very satisfactory and durable.

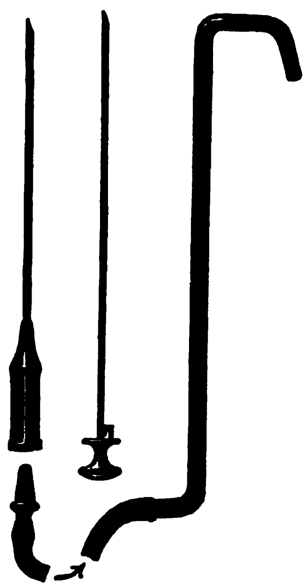

Fig. 130. — One-half Natural Size.

The *left horizontal lateral position* described above is the best position for lumbar puncture. This permits the greatest possible curvature of the spinal column anteriorly, and produces a separation of the arches of the lumbar vertebræ. The patient's condition may make it desirable to choose the right lateral posture, but this is always less convenient for the operator. A sitting posture would afford us a better view of the anatomical landmarks and help to maintain the mid-plane, but this is for many patients very exhausting and even impracticable. With this position the pressure figure read off indicates the hydrostatic pressure as well as the elastic, but does not accurately show the amount of the former; besides, if the outflow is not very carefully regulated, a sudden fall in pressure may easily occur, and serious or even dangerous consequences ensue because the fluid is then actually drawn out of the cavity of the skull. For these reasons I never perform lumbar puncture in the sitting posture.

Careful disinfection of the skin is of course necessary, especially in unconscious or uncleanly patients.

Anesthesia is not necessary in lumbar puncture except in quite rare cases of mania, severe agitation, or spasm; even local anesthesia I rarely employ. The pain from the cutaneous wound is but momentary, and needling the musculature is absolutely painless if the patient is quiet; in passing through the periosteum and into the dura itself there is some pain; the latter factor is, under some circumstances, a valuable guide.

Anatomical Relations.—As the conus medullaris in the adult terminates between the first and second lumbar vertebræ, the four lower interarcual spaces of the lumbar vertebræ are best adapted to lumbar puncture. The needle should enter the dura exactly in the middle line; the dura (including the adjacent arachnoid) is readily penetrated on account of its tension. The point of the needle rests in the median plane between the nerve trunks of the cauda equina, which, united into two strands upon the right and left side, leave a space in the middle about 2 to 5 mm. in width; this space is generally wider and better developed in children, in whom the cauda equina is also much more lax.

Fig. 131 *a*.—Cauda Equina of the New-born. (Natural size.)

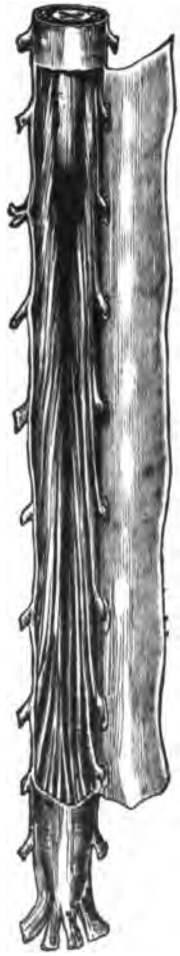

Fig. 131 *b*.—Cauda Equina of the Adult. (One-half of natural size.)

The median space is more distinct near the conus at the height of the second and third lumbar vertebræ than further downward.

Figs. 131 *a* and 131 *b* show these relations in the new-born and the adult (but they vary also in individuals).[1] Lumbar puncture is very simple in children in whom the large vertebral arch shows wide rhomboid spaces between the vertebræ. Here where the spinous processes, the ligaments and muscles are but slightly developed, we pass directly to the median line in the plane of a horizontal transverse trunk section, therefore directly from before backward, and readily penetrate the dural sac to the depth of 1–2 cm. In adults the conditions are more complicated, since with growth and muscular development (besides varying racial and individual peculiarities) the bones also enlarge, and the intervertebral arches change in width and form; the possibility of reaching them also depends upon the shape of the spinous processes; these it is true usually extend horizontally; occasionally, however, they incline downward, or have at their point a hook-shaped process so that the posterior free interarcual space is partially covered.[2] If, in the first case, we penetrate to the middle line somewhat below the lower border of the spinous process with the needle directed horizontally, the bone will be encountered, while, in the latter case, the needle must be slightly inclined upward toward the head. In muscular persons the ligamenta interspinalia are frequently so strong and tough that a needle punctures them only with great difficulty; in such cases I believe it better to make the puncture through the skin 0.5–1 cm. to the right of the median line, and to give to the needle such a direction that it enters the dura in the median line. The *rule for puncture,* therefore, is the following: Hold the needle horizontally or slightly inclined toward the head and insert it immediately below the spinous process in the median line; if the muscles are firm and the ligaments tough, the puncture should be made through the skin 0.5–1 cm. to the right of the median line, and after inserting the needle deeply we should give it a slight inclination toward the median plane—depending upon the presumable thick-

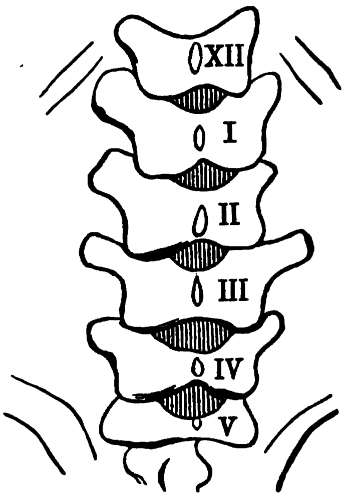

FIG. 132.—LUMBAR VERTEBRAL COLUMN OF A CHILD ONE YEAR OLD.

[1] These preparations have been made in the following manner: After removal of the brain, the upper part of the cadaver is raised, and inclined forward upon the abdomen; the sacral canal is then opened and at the apex of the dural sac a 10 per cent. lime solution is injected into the subarachnoid spaces of the spinal cord. When the body cools and rigidity has set in, the vertebral column from the sacrum up to the middle thoracic vertebra is removed and placed in a solution of formalin. After several days and when the lime has hardened, the vertebral arches are removed and the dura opened. In Fig 131 *b* the vertebræ are not illustrated.

[2] These relations may be perceived in Figs. 133 and 134, which have been copied after Braun by permission. In a geometric drawing they show the relations of the lumbar vertebræ in a lateral and posterior aspect. Fig. 133 is a preparation with a horizontal, Fig. 134 with a downward, direction of the spinous processes.

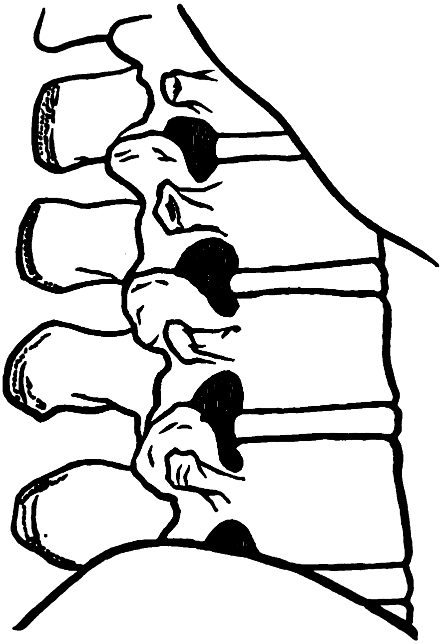

FIG. 133 *a*.

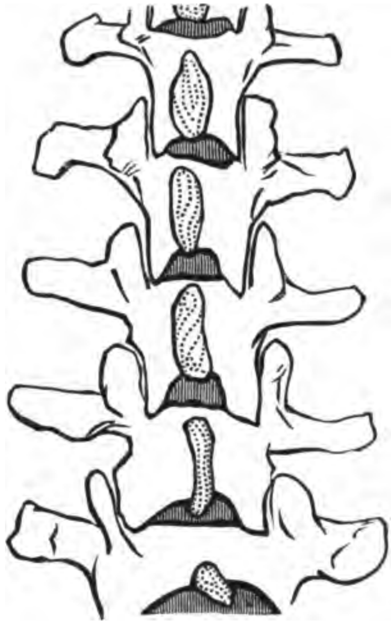

FIG. 133 *b*.

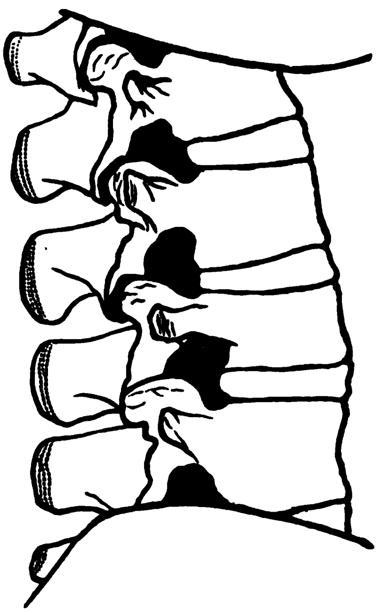

FIG. 134 *a*.

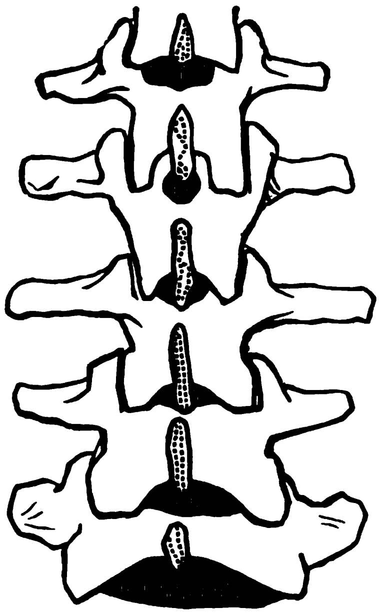

FIG. 134 *b*.

ness of the muscles. In following this rule the dural sac will not be penetrated immediately, but the needle will meet the bone or tough periosteum. It must then be withdrawn and carefully inserted in another direction. We can often feel the needle enter the dural sac, and sometimes the patient is conscious of this by the pain he feels. To determine the location of the point of the needle, we must notice the depth to which it has entered; if correctly inserted, in adults it must penetrate from 4 to 6 cm. before fluid exudes, the slighter depth being in delicate, weak persons or women, the greater depth in cases the reverse of these. In large, muscular men the depth to which the needle penetrates may be from 7 to 8 cm., in a stout person even to 10 cm. In the rare cases in which there is cutaneous edema, this must be considered and an effort made to remove it by pressure before the puncture.

It is advisable always to note the depth to which the needle penetrates, so that on repeating the operation, this may serve as a guide.

In regard to the question *which interarcual space is to be used for puncture*, there has been much unnecessary discussion. In my investigation of the skeletons of 30 adults and 12 children, I have generally found the first and second lumbar intervertebral spaces to be larger than the third or fourth. The fifth (also known as the hiatus lumbosacralis) is usually lower but somewhat broader; transversely the intervertebral spaces measure from 18 to 20 mm., and vertically 10 to 15 mm. *The third intervertebral space is generally the best* for puncture since it is broad, is sufficiently distant from the conus medullaris, and the space between the bilateral bundles of the cauda equina is here sufficiently large; but the fourth and fifth intervertebral spaces, also in adults the second, may be used for puncture. Hence, in the individual case, the choice must be made according to the palpable form of the bones, particularly of the spinous processes. With repeated punctures it is generally better to vary the point of insertion of the needle.

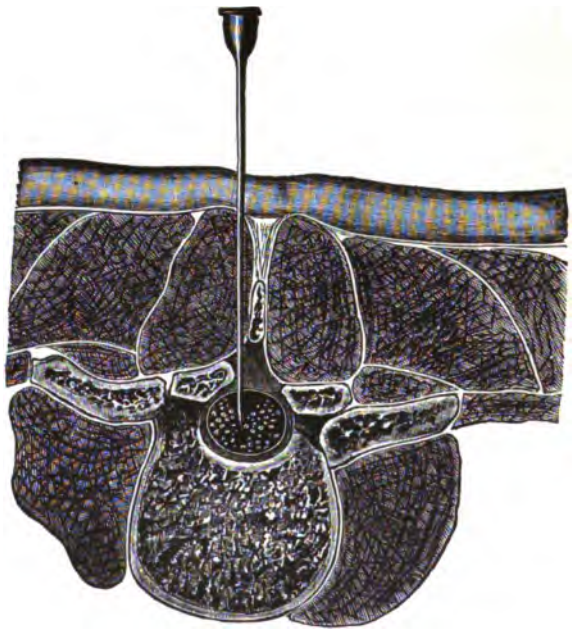

FIG. 135.—TRANSVERSE SECTION THROUGH THE LUMBAR VERTEBRAL COLUMN, THE PUNCTURE NEEDLE *in situ*. (Two-thirds of natural size.)

In the cadavers of eight children I found the end of the conus to be at the height of the third lumbar vertebra up to the end of the first year; at the end of the third year, at the height of the second lumbar vertebra, and only once, in a child four years old, at the height of the fourth lumbar vertebra.

In thin people the lumbar spinous processes may be easily counted. They are broader and may be outlined more easily than the dorsal which sometimes deviate laterally (although the twelfth dorsal vertebra frequently resembles those of the lumbar region). It must be remembered that the twelfth rib is joined to the *upper* margin of the twelfth thoracic vertebra. This is difficult to find in stout people, in whom also it is difficult to feel the ribs. The fourth lumbar spinous process is in these cases a good landmark; it is on the same horizontal plane as the upper border of the iliac crest. After determining the height of the five spinous processes by palpation I have found it of great assistance to outline these by five horizontal lines on the skin (compare Fig. 129 *a*).

In *estimating the pressure*, the behavior of the patient must be considered; sometimes, on account of pain, a change in respiration, or muscular tension, the fluid will at first rise, often decidedly (up to 100 mm.) above the true average. This should be read off only after the patient becomes perfectly quiet. The respiratory variations depend greatly on the type of respiration; those due to pulsation, as in the skull itself, are greater with high pressure. The fluid must be evacuated slowly and carefully while constantly observing all the symptoms. The operator by raising or lowering the glass tube can control the rapidity of the flow, and even instantaneously stop it; small quantities can even be returned to the sac.

How much is to be withdrawn depends upon the individual case; and the pressure and other symptoms, not the quantity of fluid discharged, form the indication for its cessation; this, however, must be read from time to time during the outflow; it should not be allowed to decline below the normal, therefore it must equal 100 mm. of water; if the pressure is high at the start, we must stop the sooner; if after a little waiting we feel assured that the figure first read shows the existing pressure in the cranial cavity, this is certainly not true of the subsequent figures, because the intracranial pressure accommodates itself slowly to that at the point of puncture, and secretion is constantly going on in the ventricles. In fact, when low pressure is finally attained by the evacuation, we see that the fluid in the glass tube tends to rise more or less rapidly.

A *comparison between the amount evacuated and the change in pressure* at the beginning of puncture is of special significance considering the previously discussed possibility of existing or threatening "automatic" closure of the cranial cavity; in this condition there is no addition from the skull to the small contents of the spinal dural sac; hence, after a few cubic centimeters have been drawn off, the spinal pressure falls several hundred millimeters. If the outflow is at first profuse with only a moderate fall in pressure, we cannot absolutely exclude such an automatic closure, but it is not very likely.

The rapidity of the outflow is, of course, increased with the height of the pressure. If, prior to attaching the tube, the fluid pours from the cannula in a stream this naturally shows high pressure, but never reveals its degree; the rapidity of outflow depends upon other important factors as well as the height of pressure, namely, the caliber of the cannula, and the condition of the subarachnoid spaces.

On attempting to remove the cannula at the conclusion of lumbar puncture, a palpable resistance is sometimes met with as if it were retained by

tense ligaments. If blood or clear fluid flows from the point of puncture this should be immediately controlled by compression. The opening should be closed by the application of a little iodoform, or, preferably, by collodion and a cotton bandage; in uncleanly patients several layers should be employed. I have never seen complications from the puncture of the canal.

When the condition of the patient does not make it impossible, he should always remain in bed for at least twenty-four hours after lumbar puncture, so that the disturbances of equilibrium in the cerebrospinal cavity may be adjusted.

With an open fontanelle the tension ceases during puncture; this indicates a fall in the lumbar pressure.

Otherwise, no noteworthy symptoms appear in the patient during or after puncture, the pulse and respiration remaining unchanged.

Sometimes during the operation, usually a short time afterward, headache is lessened, the mind becomes clearer; on the other hand, in isolated cases headache appears or increases, or general malaise sets in; in this case the puncture should at once be interrupted, the patient should be kept quiet, and an ice-bag be applied to the head. Only very exceptionally do these symptoms portend danger; as stated, they may be due to automatic closure of the cavity of the skull, but usually are attributable to distention of the brain with blood on account of the decreased pressure; in twenty-four hours at most these symptoms generally disappear. In very rare cases an elevation of temperature for a day or two follows puncture.

Difficulties in Lumbar Puncture.—The pain in lumbar puncture is usually no greater than in pleural aspiration, since in this condition also it varies according to the individual susceptibility of the patient; pain is somewhat greater if he is restless, or if the space between the bones is not immediately found; it is sometimes due to the peculiar hyperesthesia which exists in many patients with disease of the brain.

In rare cases they complain of pain or painful tension in one leg during the puncture, occasionally the latter condition becomes objectively noticeable; usually this disappears at the termination of puncture, at all events in the course of the next day; the sequels never persist. In such cases a nerve root of the cauda equina has probably been touched or disturbed by the needle. This explains the occasional anatomical finding of a fine, thread-like coagulum of blood along the nerve root (in cases in which none of the previously mentioned irritative phenomena had before appeared).

If, on withdrawing the mandrin, blood exudes from the needle in drops the point of the needle has probably penetrated the anterior or posterior venous plexus, and we must try to find the proper position by pushing the needle forward or by withdrawing and reinserting it. If no fluid appears, the same maneuver is repeated, or rotary or lateral movements are made with the needle to free the opening from occluding nerve roots, coagula of blood, or pus flocculi. In fact, during the entire operation of puncture, it is advisable to keep the needle in the position which is most favorable for the exudation of the fluid. If we believe that we are in the dural sac yet no fluid appears, we should not be afraid to draw off a few drops of fluid with a syringe; if the opening is occluded merely by pus or blood coagula, this maneuver will clear the outlet; if not, the drop which has been aspirated may

give us points in the diagnosis. To aspirate more than a few drops is unwise since there is danger if the pressure is low; and if nerve roots occlude the opening or the stoppage is due to the gelatinous composition of the exudate aspiration is useless. Thick fluid pus is not found in the dural sac. Therefore, when for these reasons or because the opening between the bones cannot be found, there is no result from puncture, we should withdraw the needle at once, and on the following day we may try to puncture another intervertebral space.

(12) **Lumbar Puncture is Performed in the Following Conditions:**

(*a*) When there is brain pressure with threatening symptoms which render the decrease of this pressure imperative;

(*b*) When there is moderate, but long-continued brain pressure which we may hope to relieve by puncture;

(*c*) When an increase in pressure is assumed or suspected, and a gauge of this pressure is desirable;

(*d*) In cases in which, aside from the question of brain pressure, we wish to examine the fluid, or to determine the existence of meningitis, or to decide its nature;

(*e*) In cases in which the fluid is diagnostically examined for possible admixtures (blood, toxins, agglutinins, etc.);

(*f*) When we wish to inject fluids into the spinal canal for therapeutic purposes.

Like other punctures, for example, in the pleural cavity, lumbar puncture is resorted to *both for diagnostic and therapeutic purposes.* In this description, just as in practice and in the concrete case, these two indications cannot be distinctly separated from each other—less so in puncture of the spinal canal than in puncture of the pleura. As the symptoms, especially the objective signs of meningitis, are by no means so positive as the signs of exudative pleurisy, puncture of the spinal canal is frequently practised as an aid to diagnosis; its results, however, are more important, because the pressure of the exudate may at this point be much increased and is of greater significance. Spinal puncture is less serious than pleural because, in passing through the serosa, even under normal conditions, the needle encounters fluid, while in the pleura, if there is no exudate, the lungs or other organ may be injured.

In the diagnostic puncture of the pleura the needle inserted is usually withdrawn and replaced by a larger one for the evacuation, but in spinal puncture the same needle serves for both purposes.

(13) **Lumbar Puncture in Various Diseases.**—(*a*) SEROUS EFFUSIONS are most readily influenced by puncture. The cerebrospinal effusions termed hydrocephalus are of less significance, even if of the same volume, for the pressure which the brain suffers, not the amount of fluid, is the important point.

This is most evident in the hydrocephalus of children; here we have cases with only a slightly enlarged cranium yet severe brain symptoms, and, on the other hand, enormous heads with but slight disturbance in function. If the sutures of the skull are still open the first group will show decided, and the second group but slight, tension of the fontanelles. But the condition may change; in the first group the brain symptoms may lessen (although the skull may perhaps increase in size) if the tension of the fontanelles is de-

In order to lose none of the data obtained by lumbar puncture, we should note the findings on a printed diagram (such as is here depicted). This enables us to compare very readily the results of several punctures in the same patient.

<table>
<tr><td colspan="2">NAME</td><td rowspan="3">DISEASE (Duration, Symptoms)</td></tr>
<tr><td>Age</td><td>Sex</td></tr>
<tr><td colspan="2">Admission to Hospital</td></tr>
</table>

<table>
<tr><th colspan="5">LUMBAR PUNCTURE</th></tr>
<tr><td colspan="2">Date...............</td><td></td><td></td><td></td></tr>
<tr><td colspan="2">Place..............</td><td></td><td></td><td></td></tr>
<tr><td colspan="2">Degree.............</td><td></td><td></td><td></td></tr>
<tr><td rowspan="3">PRESSURE</td><td>At the begininng of puncture.....</td><td></td><td></td><td></td></tr>
<tr><td>At the end of puncture........</td><td></td><td></td><td></td></tr>
<tr><td>Variations..........</td><td></td><td></td><td></td></tr>
<tr><td rowspan="7">FLUID EVACUATED</td><td>Amount............</td><td></td><td></td><td></td></tr>
<tr><td>Appearance........</td><td></td><td></td><td></td></tr>
<tr><td>Specific gravity.....</td><td></td><td></td><td></td></tr>
<tr><td>Albumin per 1,000..</td><td></td><td></td><td></td></tr>
<tr><td>Sugar.............</td><td></td><td></td><td></td></tr>
<tr><td>Cells...............</td><td></td><td></td><td></td></tr>
<tr><td>Microörganisms.....</td><td></td><td></td><td></td></tr>
<tr><td colspan="5">REMARKS:</td></tr>
</table>

creased, while in the second group symptoms of cerebral irritation and pressure may sometimes persist for weeks or longer if the tension is increased.

"*Chronic hydrocephalus*" of children often begins acutely with meningeal symptoms, and not rarely may subsequently show acute or subacute exacerbations, both anatomic as well as symptomatic. With the closed skull these processes are not marked by increase of tension and circumference, but only by functional disturbances, which, however, are readily produced because with the same volume the exudate exerts a much stronger pressure. The volume of the effusion and the symptoms more clearly explain the varying picture of hydrocephalus (except hydrocephalus *ex vacuo*) if we regard the underlying process as an inflammatory increase in exudation. The clinical picture varies because the affection sometimes appears acutely, sometimes insidiously, and then again after long intervals in exacerbations; also because with an open skull this is difficult to demonstrate by palpation with the hands, and in the closed skull without lumbar puncture; to this we may add that unlike the condition in other serous cavities, after removal of the inflammatory exudate the former anatomical relations are restored; but the rigidity of the walls of the skull and deficient elasticity of the brain often cause the ventricles to be distended with fluid although the secretion is only of normal amount.

In addition to *acute serous* forms of *meningitis* we meet the symptom-picture of acute and transitory exudation which I have designated as angioneurotic hydrocephalus. Other pathologic processes—neoplasms, parasites, effusions of blood—may cause increased transudation and subsequently hydrocephalus.

As in other serous cavities the restoration of the equilibrium by the outflow of the secretion is of decisive importance; if the outlets become too narrow or too large, the absolute quantity of the fluid increases and the pressure rises; the latter circumstance often at first increases the outflow, the tracts widen, and compensation is brought about; but a certain increased internal pressure may also occlude the outlets, most readily in the cavity of the skull, where distention of the ventricles presses the surface of the brain against the bone, and the subarachnoid and subdural spaces, also the channels to the Pacchionian granulations, are narrowed. These are the conditions in which spinal puncture beneficially decreases the pressure; it clears the outlet of obstacles and thus breaks up a vicious circle. Whether this benefit is permanent, depends upon other factors.

When the secretion continues to be profuse because of the return of the old condition, repeated punctures are necessary, and these may possibly retard the damage to the brain from pressure until the disease is on the decline. *Spinal puncture, therefore, may be beneficial, especially in acute and subacute serous meningitis* and in the acute exacerbations of chronic cases, but this will be only transitory or very slight when the exudation, although perhaps not great, is uniformly persistent; success, therefore, depends upon the nature and study of the pathologic process—just as in that of the pleural cavity and of the joints. In these regions puncture is merely a remedy which, combined with other treatment, we use in certain stages and for certain indications. On account of the importance of the organ, the danger of an increase in pressure in the cerebrospinal cavity is greater than anywhere else. At the

present time, we can often determine the cause of an existing pressure in the brain, but we can only guess at the nature and stage of the exudative process; hence, the conditions being to a great extent unknown and varying, the benefits from lumbar puncture in serous effusion also vary; in adults as in children the results of one puncture have been even more surprising than in pleural aspiration, and have suddenly changed the course of the disease. This is particularly evident in the acute, inflammatory, and angio-neurotic forms where it is only necessary to free the outlets as soon as the exudation has reached a high mark.

The cases in which the initial improvement did not continue, and in which repeated punctures were necessary, are more numerous. Here the exudation decreased only after the lapse of weeks or months, either "spontaneously," under other treatment, or because puncture produced a different condition, perhaps because irritating products in the fluid had been removed. In such cases many punctures (10 to 25) have been made at intervals of days (Grober).

When the secretion is continuous and profuse, puncture is of little benefit or only transiently. It is useless also in those cases of chronic hydrocephalus in which we must assume that larger or smaller areas of the brain have been changed or atrophied by long continued pressure. This statement must be modified in regard to the moderate hydrocephalus of small children, in which, even under such circumstances, repeated punctures are sometimes followed by a marked improvement in mental and other nervous functions, probably because the diminution in pressure permits a greater development of the brain.

As the development and the stages of serous effusion differ, so the result also varies.

In addition to other data, the puncture throws light upon certain conditions: High pressure denotes a still existing hypersecretion; the removal of large quantities of fluid indicates a decided dilatation of the cerebrospinal cavity, therefore probably a somewhat longer existence of the hydrocephalus; an amount of albumin below 0.5 per thousand generally denotes a favorable prognosis.

It may also be stated that an absolutely clear fluid may contain a small number of morphotic elements which can be recognized only upon sedimentation, centrifugation, or coagulation. The microscopic examination for cells and microbes should never be omitted; serous exudates may show a series of conditions from that truly and morphotically free through all grades of turbidity up to a purulent one.

Pneumococci and streptococci, the bacilli of typhoid, influenza and tubercle, and coli bacilli have been found in the serous fluid, as well as in other serous exudates, i. e., those poor in cells, as in the pleural cavity.

(*b*) Purulent Meningitis.—In purulent meningitis puncture is rarely followed by the evacuation of thick pus, but of a cerebrospinal fluid with more or less purulent admixture, and this varies from slight turbidity to a distinctly yellow and purulent discoloration. On standing, the deposit of pus is slight and only exceptionally is it coagulated by excreted fibrin; macroscopically (and this is diagnostically important) the fluid may sometimes be perfectly clear, while, further upward, the meshes of the pia are filled with purulent coagula.

Microscopically the cells of fresh purulent exudates contain leukocytes with divided nuclei, while in chronic exudates, in which the fluid is usually less turbid, lymphocytes also are found; in very late stages of protracted purulent or tuberculous meningitis these cells may be found alone.

The microbes most frequently noted are meningococci, pneumococci, and streptococci, as well as tubercle bacilli; in stained preparations the meningococcus and pneumococcus, although recognizable, are sometimes attenuated, and no longer grow in the culture. If they are absent from the fluid, particularly the meningococcus intracellularis, they may be found in the adherent purulent exudate.

But there are also actually sterile purulent inflammations of the membranes of the brain (in syphilis) in the exudate of which no microbes can be demonstrated, in which, therefore, a dissolved virus from the blood or from the cerebrospinal fluid has perhaps produced the inflammation.

Purulent meningitis is always a dangerous disease, and the results of therapy are necessarily less satisfactory than in the serous form. Since the malady is a purulent inflammation of the cell tissue, we can expect but little from puncture, even from repeated punctures; nevertheless these sometimes produce most certain results which of course differ according to the genesis of the disease. The greatest benefit appears to follow puncture in the forms due to meningococci, next those due to pneumococci, and after one puncture, especially in the former, we occasionally observe improvement both in regard to the fever and the cerebral symptoms. In other cases several punctures are necessary (in a case of Netter's, eleven); in such a series of punctures the turbidity of the fluid gradually decreases. In this form of meningitis success probably depends upon the fact that the virility of the meningococcus suffers, the disease is thus checked, and it is then only necessary to form an outlet for the escape of the toxins with the dead bacilli.

When thick, fluid, flocculent pus is found, puncture is followed by irrigation with a sterile normal salt solution (Jakob, Krönig; see page 42).

Even when therapeutic success cannot be expected from puncture, either because the symptoms are too mild or too severe, by determining the nature of the meningitis this operation may give us *indications for the diagnosis as* well as for treatment. It has often been resorted to in *diseases of the brain* in order to decide whether operation on the skull was justifiable or not. After injury to the skull, in brain abscess, and in otitis media, cerebral pressure symptoms may be due to purulent meningitis and may be either circumscribed or diffuse, but they may also be due to serous meningitis and of varying importance in the prognosis and treatment. Here lumbar puncture clears the situation. If it shows serous fluid, operation upon the skull is likely to be successful, although it by no means excludes circumscribed purulent inflammation.

Surgeons as well as otologists have practised lumbar punture on these principles. Braunstein (Schwartze) excludes purulent cerebral meningitis only when a clear fluid flows off so profusely that we may suppose it to originate from the cranial cavity. Aside from the fact that this is not an infallible guide, such a profuse evacuation appears to me not devoid of danger, because it may mobilize a circumscribed pus focus and diffuse the inflammation. Nor does an operation upon the skull appear to me to be absolutely contraindicated because purulent fluid is obtained by puncture; certainly there is less hope of success, but other circumstances also must be taken into considera-

tion. It is quite conceivable that a purulent meningitis limited to the cerebellum and spinal cord might heal if the abscess in the cerebellum, or suppuration of the bone from which it originated, were given an outlet.

(*c*) TUBERCULOUS MENINGITIS.—Although tuberculous meningitis is really included in the previous description, its frequency and its clinical picture merit special consideration. As a rule, the fluid obtained by puncture is only slightly turbid. It may be perfectly clear with small floating flocculi. On standing it forms fibrin coagula, and lymphocytes are microscopically present (either with pus corpuscles or alone). At the stage of onset the fluid may be perfectly clear, in rare cases it is markedly purulent (for example, in mixed infections with meningococci or pneumococci).

The amount of albumin is usually greater than normal, and varies between 0.5–2 per thousand. The fluid, therefore, according to the individual case and stage, may show all the transitional stages between a purely serous and a purulent meningitis. Lumbar pressure, corresponding with the distinct pressure symptoms, is usually high, over 500 mm. water.

Pfaundler once observed it to be 1,500 mm. (= 110 mm. Hg); he found in his cases in the stage of irritation an average of from 650 mm. to 700 mm. of pressure, and in the stage of paralysis 320 mm. pressure. The "pressure curve" resulting from this corresponds also with the other pressure symptoms. It can scarcely be called typical, but varies greatly in the individual case.

The *finding of tubercle bacilli* in the fluid is noteworthy and often an important aid to the diagnosis; these increase in the terminal stages of the disease, and are invariably found in the fluid post mortem. Some investigators found them in more than 75 per cent. of the cases. The greater fluidity of, and sparsity of cells in, the liquor compared with other serous exudates may favor their dispersion; and the shape of the dural sac promotes their sedimentation in the lumbar portion.

In addition to other methods, tubercle bacilli may be demonstrated by inoculating guinea-pigs either subcutaneously, intra-peritoneally, or intra-durally; Martin[1] produced tuberculous meningitis by occipital injection, Hellendahl induced by spinal injection not this disease but general miliary tuberculosis, and more readily than by any other means.

Langer used the fluid itself for culture; 4 to 5 cm. were dropped into sterilized culture tubes and placed in the incubation oven; after five or six days the sediment was profuse, and after several weeks the culture showed more tubercle bacilli, granule and nodule forms. Langer was thus enabled to demonstrate bacilli when none could be found in the fresh specimen.

In some cases of tuberculous meningitis this demonstration of tubercle bacilli may be of great significance; I will merely mention that Freyhan, Henkel and Gross proved that the disease does not always terminate fatally.

Although only in the rarest cases does recovery from tuberculous meningitis follow lumbar puncture, there is often a transitory improvement, consciousness returns at least for a time, and restlessness, moaning and spasms cease; repeated punctures bring about the same amelioration.

(*d*) TUMORS.—Brain tumors at first press upon the surrounding areas, subsequently by displacing the brain substance they flatten the subarachnoid

[1] *Soc. de biologie*, 5. Mars, 1898.

spaces; this impedes the passage of the cerebral spinal fluid toward its outlets, and lymph stasis may produce hydrocephalus. The secretion of the venous plexuses may be increased by the influence of the tumor; this may also be caused by venous hyperemia from pressure upon Galen's veins. Besides the lessening of space from the existence of the tumor, other factors may increase the pressure.

These complicating conditions vary according to the size, the seat, and the nature of the tumor; hence lumbar puncture in brain tumor reveals varying heights of pressure.

The evacuated fluid is usually clear and without cellular admixture; the amount of albumin varies from 0.5–8.0 per thousand.

Lumbar puncture can be therapeutically successful only in those cases in which hydrocephalus is marked besides producing pressure on the brain. It is obvious that this result can be only palliative, as in the case of neoplasms of the pleura.

I have stated that in brain tumor an automatic closure of the cranial cavity readily occurs, and for this reason puncture must be most carefully performed. This is particularly true when the seat of the tumor is presumably the cerebellum. Here the rapid decline of lumbar pressure after the removal of but a small quantity of fluid is an aid to the diagnosis.

(*e*) ABSCESSES.—Abscesses like tumors act mechanically upon the contents of the skull. Here lumbar puncture aids but little in the diagnosis except in determining the existing cerebral pressure. In abscess it may help us to decide the question whether rupture has taken place and produced meningitis.

In cases of abscess, lumbar puncture is as little beneficial as in tumor; at most it may temporarily lessen the pressure on the brain, which is due to an accompanying ventricular effusion.

That free outlet may favor a threatening rupture of the abscess is obvious, but no case of this has as yet been reported.

(*f*) VARYING CONDITIONS.—Lumbar puncture proves that the cerebrospinal pressure in *sinus thrombosis* (Braunstein) and *compression of the superior vena cava* (up to 420 mm., Noelke) may be increased by stasis transudation into the subarachnoid spaces and the ventricles. This may also be presumed, although to a less extent, in valvular lesions with *general venous stasis*.

The same circumstance is the chief cause of the *temporary* but *marked increases of pressure* in the *spasmodic conditions of epilepsy and hysteria*, in the spasms of paralytics, and also in attacks of mere *screaming* with *pressure* amounting to from 300 to 800 mm. (Navratzki and Arndt, Stadelmann, Quincke). Except during the attacks, pressure has been found normal in epileptics. In two epileptics who had severe attacks, Chipault saw improvement after several punctures.

Vidal and Le Sourd found lymphocytes in the spinal fluid in progressive paralysis; probably these were the expression of subacute inflammation.

The serous transudations in uremia, lead colic, and in some infectious diseases are of somewhat doubtful pathogenesis.

In *uremia* the spinal pressure is sometimes unchanged and, even when there are no convulsions, it is sometimes increased. In these cases, as in

arachnoid and cerebral edema, there is increased transudation, and to this as well as to toxic influences do we attribute the varying symptomatology of uremia. After spinal puncture there is sometimes a decrease in the cerebral symptoms. In a case of *lead colic,* and during the coma following eclampsia, Segelken and Brasch saw the pressure amount to 310 mm. and after evacuating 50 c.c. of fluid, partly clear and partly turbid, there was improvement.

In some infectious diseases, such as *enteric fever, scarlatina, pneumonia,* a moderate increase of pressure has occasionally been found on lumbar puncture (Stadelmann, Salomon and others), no true meningeal symptoms being present. As the diseases in question are sometimes intercurrent with microbic meningitis, it is possible that there may have been a very mild grade of this affection, or perhaps only a toxic irritation of the meninges. The same conditions were found in *influenza* and in *acute leukemia* (Stadelmann), perhaps also in *plague,* for marked meningeal edema has been described in the cadavers from this disease.

In *chlorosis* with severe headache and meningeal symptoms lumbar puncture frequently shows an increase of spinal pressure, and the symptoms are often favorably influenced by puncture. Although sinus thrombosis may in some cases be the cause of the condition (Lenhartz), yet, on account of the brief duration of most of the symptoms, I am inclined to assume an angioneurotic increase in secretion. Moreover, lumbar puncture sometimes produces the same results and is just as beneficial in the severe *migraine* of those who are not chlorotic.

The meningeal affection of *syphilis* is most frequently gummatous or indurative and circumscribed; when localized in the spinal cord the latter form may produce such a condition that a normal discharge of fluid does not follow spinal puncture. In the secondary as well as the tertiary stage of syphilis we have the picture of serous meningitis; here, under high pressure, a clear or slightly turbid fluid flows profusely, and improvement follows (Quincke; Chipault, a typical case). I have no knowledge of the anatomical cause of these forms.

(*g*) HEMORRHAGE.—Copious effusions of blood into the cranial cavity are produced by spontaneous cerebral hemorrhage, by aneurysms from the large basal cerebral arteries, or by trauma; the latter are usually meningeal. Traumatic hemorrhages in the skull as well as in the spinal canal often, but not necessarily, accompany fractures. Hemorrhages into the cranial cavity may increase not only the intracranial, but also the spinal, pressure; as in tumors the disturbance is caused primarily by an impediment to the outflow of lymph, but often it may be due to an inflammatory increase in secretion.

If there has been a rupture into the ventricles or the subarachnoid spaces blood alone is mixed with the fluid from puncture; but the blood may coagulate further up and the spinal fluid remain clear. The blood usually coagulates when it is effused into the almost empty subdural space. Partly for this reason and partly because the physiologic current sets in the opposite direction, blood effused subdurally does not, as a rule, reach the subarachnoid spaces and subsequently the fluid obtained by puncture; the conditions are different if the arachnoid has been injured. When the seat of the hemor-

rhage is extradural we may still less expect to find blood in the aspirated fluid, but by diffusion hemoglobin may transude and give to it a yellow color.

In obscure cases, after accidents, and in unconscious patients who can give no history, lumbar puncture from positive as well as from negative findings may be valuable in diagnosis. But we must be cautious, for if the hemorrhage is recent a marked decrease in pressure may increase it or start it anew, especially in the skull. After a few days this danger is lessened, and if some of the blood is evacuated, perhaps simultaneously the transudate also, the remainder may possibly be more rapidly absorbed. After traumatic hemorrhage between the membranes of the cord, puncture and evacuation of the effused blood has frequently had a very beneficial effect (Kiliani, Jacoby, quoted by Braun, Bugge).

(*h*) Subdural Effusions.—Between the dura and arachnoid, just as may occasionally happen between the layers of the pleura or of the peritoneum, pus forms in a thin layer; for example, in bone suppuration which may extend from the ear to the dura, the pus may flow toward the spinal sac and separate the arachnoid from the dura; the subarachnoid spaces are then compressed and the arachnoid is forced against the pia. In lumbar puncture, after passing through the dura, we reach a subdural space which normally does not exist.

In a case of this kind I found 500 mm. of pressure and evacuated 20 c.c. of foul, creamy pus. At the autopsy the pia-arachnoid of the spinal cord was merely hyperemic, and showed no purulent infiltration; the surface of the cerebellum was covered by pus which could be washed away, and the subarachnoid tissue in the area of the pons and the chiasm was already pus stained.

Serous effusions into the subdural space do not appear to occur; a secretion into the ventricles and subarachnoid spaces seems to predominate, normally as well as pathologically; for this reason the arachnoid becomes tense and separates from the pia; in the spinal canal hydrostatic pressure is added to this, and the arachnoid is closely pressed against the dura.

(14) In conclusion, in **puncture of the spinal sac the diagnostic and therapeutic conditions are analogous to those of puncture of the pleura, of the joints, and of other serous cavities.** The fluid in these cavities is not a fixed and constant amount, but an ever varying one and ever forming; it cannot be compared with the contents of a bottle which may be emptied, but to a natural reservoir with an inlet and outlet; by pumping the level may be reduced for a short time, but this will be permanent only when the afflux is decreased or the outlets are enlarged. So the immediate results of a puncture will be permanent only when the conditions which have promoted the accumulation of fluid are changed, either by evacuation, by the natural course of the disease, or by other means; this is also true of lumbar puncture. The peculiarity and vital importance of the enclosed organ, the narrowness of the cavity, and its distended shape are important. The first two factors give to each of its diseases greater significance; the third enables us to differentiate from each other the distant cranial cavity and the lumbar region, as well as to attempt therapeutic procedures.

(15) **Slitting the Dura.**—When, after the puncture of serous effusions, the cerebrospinal pressure persistently rises, it is often advisable to evacuate

the fluid continuously for some time. This has been done in hydrocephalus by inserting a drain of catgut between the lateral ventricle and the subdural space, or a gold cannula between the ventricle and the subcutaneous tissue. In the lumbar region the needle (Lenhartz) or cannula (Sahli) is permitted to remain for some time, but on account of the danger of displacement and infection this cannot be permitted for a long period. I have, therefore, *tried to dissipate the fluid into other tissues and fluid tracts by slitting the dura;* this is accomplished *by a long-bladed lancet,* the tip having a breadth of 3 to 6 mm., which is held in the median position and introduced exactly like a needle; the passage through the dura is distinctly felt, and the depth of the longitudinal section may be gauged by the further introduction. The incision may be enlarged by a lever movement. Sometimes fluid exudes from the wound and along the handle of the knife, but shortly after the removal of the knife this flow ceases, the wound is packed with iodoform gauze, and collodion and a bandage are applied. The cerebrospinal fluid now flows into the canal which has been artificially made and into the tissue spaces; this is sometimes recognizable from an edematous swelling of the subcutaneous cellular tissue and of the deeper soft parts in the lumbar region. This swelling may continue for one, two, or even eight days; but even when it is invisible the drainage may continue, especially into the loose, peridural cellular tissue.

Fig. 136.—One-half the Natural Size.

In this process there must necessarily be some injury to the veins and the peridural plexus; but, under the pressure on the exuding fluid, hemorrhage soon ceases; I have never seen serious consequences from this, nor from an injury to the bundles of the cauda equina. When a necropsy was subsequently performed, the slit was found in the desired area or very near it in the median line, and always shorter than I expected (1 to 3 mm.) probably because of the elasticity of the dura; hence there appear to be neither danger nor sequelæ; as a rule, the slit closes more rapidly than is desirable, probably because of a slight displacement of the arachnoid. Lately I have several times attempted to produce a transverse slit by withdrawing the knife from between the arches, turning it at an angle of 90°, and then reinserting it. But with this method it is probably wiser to make the transverse slit at first.

Slitting should be performed only after a preceding puncture when we are familiar with the conditions, and also have some idea of the depth to which we wish to penetrate. It may happen that the narrow, intervertebral space will admit the needle but not the lancet; then the adjoining space will be chosen. While we should puncture and gauge the pressure, as a rule, immediately before slitting, it is not wise to diminish this by an outlet because the tense dura permits the knife to enter more freely. To facilitate the insertion of the knife, I have had a furrow made in the side of the lancet, and the pedicle bent like a bayonet, so that the knife may be inserted alongside the needle which is already in the dural sac; but this is no great improvement, and it necessitates that the blade of the lancet be somewhat thicker.

I have performed slitting about twenty times, and my opinion is that it should be further tested; it is beneficial in chronic and subacute cases of serous exudation when the pressure is permanently increased; I have not refrained from employing it in tuberculous meningitis, because the bacilli which are evacuated are manifestly less dangerous outside than within the spinal canal.

(16) *Lumbar Puncture* has also been employed **therapeutically**, namely, **to introduce fluid into the spinal sac.** Jacob and Krönig,[1] after performing puncture in purulent meningitis, injected a physiologic *salt solution* which diluted the *pus*, and then *evacuated it.* The much smaller dimensions of the subarachnoid space naturally make this less suitable for such irrigations than the cavity of a joint or serous sac, and the benefit from this procedure will of course be limited to merely a few segments of the spinal cord; successive trials will show whether by this means we cannot occasionally obtain results. From 5 to 25 c.c. of fluid were repeatedly injected, and allowed to flow out.

Drugs, too, have been injected into the spinal canal on account of their direct action on the spinal cord and nerves.

Injections of *cocain,* first made by Bier, show to-day the best results and the most manifold application of this process. Within four to eight minutes after 0.01–0.02 of cocain has been injected into the subarachnoid sac, there is decreased sensation (for tactile and temperature impressions) and complete analgesia in the lower half of the body from the navel down, so that operations may be performed upon the lower extremities and pelvis without the patient feeling pain.

The *analgesia* lasts from an hour to an hour and a half; motility is completely retained but the muscles are flaccid (in 5 per cent. of the cases flatus and feces were involuntarily passed, but never urine). In the majority of cases (80 per cent., Tuffier), there are *sequels*: Headache, general discomfort, paresthesia of the legs, difficulty in respiration, a sense of heat, sweating, nausea or vomiting (in 40 per cent.). After a few hours there is sometimes a brief rise in temperature (up to 102.2° F.).

At the expiration of an hour cocain can no longer be demonstrated in the cerebrospinal fluid (Tuffier).

There is some uncertainty in regard to the degree of analgesia and its relation to the dose, as well as concerning the appearance and duration of the secondary effects.

Bier injected 0.01–0.02 of cocain in a one or two per cent. solution; lately he has preferred to inject 5 c.c. of a corresponding dilute solution after having previously evacuated the same amount of fluid (5 c.c.). Jacob considers the most suitable menstruum a 0.1 per cent. salt solution. Guinard regards the headache which often appears after Bier's process as a slight aseptic meningitis, since lumbar puncture diminishes the pain, and the lymphocytes discharged in the fluid are in proportion to the intensity of the pain. Guinard succeeded in averting the headache by adding cocain to the cerebrospinal fluid previously obtained by puncture, and then reinjecting it. This is very easily done; instead of using a glass tube for lumbar puncture a small burette with a capacity of 5 to 10 c.c. may be employed by attaching it to a rubber tube (see Fig. 129 *b*); after about 5 c.c. of the cerebrospinal fluid have drained into this, we add the cocain with a graduated syringe or pipette, and then by raising the burette the fluid passes back into the spinal sac. Beta-eucain (0.02–0.06) or tropa-cocain (0.05–

[1] *Berl. klin. Wochenschr.,* 1902, Nr. 8, p. 174.

0.06) have been employed instead of cocain. Of course, the solutions must be sterilized by boiling.

Besides in operations upon the lower extremities and the pelvis, subarachnoid injections of cocain have been used to diminish pain in sciatica, in herpes zoster, in epididymitis and in gastric crises, also other pain eccentrically projected as in priapism due to myelitis. They had no efficacy in lead colic.

The question as to where the effects of cocain are produced brings into consideration the posterior roots and the sensory ganglion cells of the spinal cord; probably the latter are chiefly affected. Contrary to the usual course of the current, the cocain must reach them by way of the lymph tracts. With Nissl's stain, Carini found changes in their chromatin and cell nuclei 8 to 24 hours after the injection.

Neugebauer investigated very minutely the time and place in which analgesia developed; as a rule, he found the first sensory area to be in the fourth sacral segment (perineum, etc.), thence gradually rising to the third, second and first sacral segments; above this the trace was lost. In 50 per cent. of the cases a certain diminution in the sensation of pain was perceptible also in the upper extremities and the head, including the mucous membrane.

The differences in the action and the sequels of the same dose may be attributed either to the fact that the cocain solution in a relatively concentrated form acts only upon the lower portion of the spinal cord, or that higher up it is more admixed with the cerebrospinal fluid.

Tetanus antitoxin, as well as cocain, has frequently been injected into the subarachnoid space, apparently with success.[1] Here the active substance is injected directly into the diseased spinal cord; after a few hours there is a decided rise in temperature.

The marked effect of this direct introduction appears to depend not only upon the short distance to be traversed but also upon the fact that some substances very slowly reach the nerves by way of the blood and through the walls of the vessels; for instance, Lewandowsky observed that strychnin introduced into the subarachnoid of animals was effective in one-tenth of the subcutaneous dose, and that sodium ferrocyanid in doses of but a few centigrams produced motor irritative symptoms which could not be attained through the circulation by doses one hundred times as great.

Sicard made similar observations concerning morphin.

In regard to further absorption by way of the subarachnoid spaces, reports are contradictory. While E. and A. Cavazzani, after a few attempts, declared the absorption of combinations of iodin and ferrocyanid to be slow, Lewandowsky found the absorption of ferrocyanid to be but little slower than through the subcutaneous connective tissue, the substance appearing in the urine after the expiration of 15 to 35 minutes. After the injection into dogs of one gram of KI in a 4 per cent. solution, Jacob found the iodin in the urine six hours later; fulminant symptoms, however, showed that the meninges were markedly irritated by the salt, for even a 0.7 per cent. salt solution has an irritating effect both in dogs and man, while a 0.1 per cent. solution does not irritate. Of iodin (sodium iodid?, Quincke) a solution of 0.04 per cent. is most suitable. In three cases of cerebrospinal syphilis Jacob injected 25 c.c. of this solution (therefore 0.01 of the salt) once or twice into the cerebrospinal sac, and believes that the results were good.

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[1] Sicard, Schultze, Leyden, Blumenthal and others. See also volume on "Infectious Diseases," pp. 842 et seq.

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Very complete literature up to 1899 is embraced in Neurath's reference. In the preceding we have mentioned only the more recent articles and those of the earlier ones which were especially referred to in the text.

CEREBRAL HEMORRHAGE AND EMBOLISM

By R. GEIGEL, Würzburg

When a person is seized by apoplexy, in by far the great majority of cases the attack is due either to hemorrhage from, or embolism of, an artery of the brain. These affections differ in many features, yet they have also much in common, and it is worth our while to make a few comparisons in their description.

In the first place both are typical focal affections of the brain. In one case we are dealing with an effusion of blood, a hemorrhagic focus, in another with the consequences of embolism, softening of the brain substance, an encephalomalacic focus, both occurring at the same points of preference and both being markedly circumscribed local affections. Common to both is a series of diffuse brain symptoms culminating in a more or less rapid disturbance of consciousness which may terminate in complete coma, symptoms which have been included under the name of the "apoplectic insult." It is impossible to decide from the symptoms alone whether rupture of the vessels or an occlusion has taken place. The statement is frequently made that in the first case redness, and in the other pallor, of the face is present—an unreliable differentiation upon which we should never depend in practice—and it must be confessed that without a general examination, without a history, the differential diagnosis is absolutely impossible from the objective cerebral phenomena; even including everything diagnostically valuable it is exceedingly deceptive, and in many cases cannot be accurately made.

These initial **symptoms** which introduce cerebral apoplexy as well as embolism may be referred to a disturbance of the entire brain function, and since these are of mechanical origin they must also be explained mechanically, and this we shall now attempt to do.

It must be remembered that after ossification of the sutures of the human skull and the closure of the fontanelles, the skull resembles a capsule in which two fluids, the blood and the liquor cerebralis, also the semi-solid brain mass, are hermetically enclosed. These three constituent parts, as well as the capsule of the skull, are insusceptible to pressure, and in a physical sense may be regarded as absolutely rigid. We know that there is pressure—the intracerebral pressure—within the cavity of the skull, that this may vary even in normal conditions, and that it usually attains a height of 100 mm. water pressure. If the blood-vessels, the arteries of the brain, are assumed to empty freely into the cavity of the skull, necessarily in the interior of the skull the same pressure—the arterial pressure—must be present. The theory of the simple propagation of this pressure is opposed by the vascular tension; there-

fore we may state it as a law that the intracerebral pressure at all times equals the difference between the arterial pressure and the vascular tension. When the arterial pressure remains the same, an increase of vascular tension leads to a diminution, and a cessation of vascular tension to an increase of intracerebral pressure. This process is of vital importance for the blood supply of the brain, therefore also for the normal processes of life. Not the quantity of blood constantly present in the cavity of the skull, but the quantity propelled in a unit of time through the capillaries of the brain determines the nutrition and proper function of the living cerebral substance.

When the intracerebral pressure increases, the thin walled portions of the vascular system—the veins and capillaries—must necessarily be compressed, thus producing a slowing of the circulation which we designate by the term adiæmorrhysis cerebri—deficient circulation in the brain. Hence, from what has been stated, even a decrease of arterial tension while the arterial pressure remains the same represents a causative factor in the production of this condition.

Any sudden arrest of the circulation in the brain may produce the symptoms which we observe during an attack of apoplexy—vertigo, nausea, vomiting and loss of consciousness—and also the clinical symptoms which we designate by the term "preeminent pressure symptoms"; rigid pupils, retraction of the abdomen, and slowing of the pulse are merely the consequences of a greatly increased intracellular pressure, a sudden, acute adiæmorrhysis cerebri.

How this occurs in apoplexy may be easily understood. If the arterial wall ruptures at any point, the arterial pressure may irresistibly be transmitted to the cavity of the skull, the intracerebral pressure increases, the veins and capillaries are compressed, general adiæmorrhysis occurs, the apoplectic insult takes place, and only gradually, after the blood and lymph have had time to flow off, is the equilibrium within the skull reestablished, the rupture in the vessels becomes occluded, the brain begins to recuperate and to function normally.

The description of the process in embolism is not quite so simple. When a plug produces occlusion of a branch of a cerebral artery, the propagation of the arterial pressure in the vessel to the periphery ceases, and the vascular tension brings about a constriction by contraction of the walls.

Since there is never a vacuum in the normal skull the effects of this constriction must be conveyed to the entire brain substance, and cause a sudden dilatation of all of the cerebral arteries in which no embolism has occurred.

Therefore, at the moment when a vessel of the brain is occluded by embolism, the blood coming from the heart pours into the widened vessels of all the other vascular areas and dilates them, not, however, flowing from the arteries through the capillaries into the veins, and thus supplying the brain substance with freshly oxidized blood. This theory demonstrates that even in the veins a regurgitating stream must arise, and that in the capillaries —as we must infer—the circulation is for an instant arrested; they are filled with blood from the veins, and this acute disturbance of the circulation is nothing more than a sudden impairment of the circulation of the blood in the brain, an adiæmorrhysis cerebri acuta.

In comparing these affections it at once becomes obvious that no differential diagnosis between the apoplectic attack in hemorrhage and that in em-

bolism can be made, since in both, notwithstanding the difference in cause, the same mechanical factors are operative—an acute, general impoverishment of the supply of blood to the brain.

While the same general and grave cerebral symptoms may be observed in other conditions, in intoxication, in cardiac asthenia, in uremia, in diabetic coma, they are distinguishing features of the apoplectic insult in that, in spite of the similarity of the brain symptoms, each represents a focal affection of the brain, and even during the continuance of the severe general picture produced by the phenomena of the attack, we must search for symptoms which will furnish a clue to the localization in the brain.

Of course there is as yet no sign of the circumscribed symptoms of paralysis which subsequently appear when the patient has regained consciousness.

The four extremities are apparently flaccid, and uniformly show that they are no longer under the control of the patient. The expression of the face and the very rapid drop of an extremity which has been lifted scarcely reveal the severe implication of *one side* as compared with the other.

Very soon, even while profound unconsciousness continues, peculiar changes in the reflexes occur, and among these none is more important than the well known cremaster or—more aptly speaking, since it can be tested also in the female—the inguinal and oblique reflex. If we stroke the skin upon the internal side of the thighs toward the pudenda with a pointed instrument, the fibers of the internal oblique muscles contract toward Poupart's ligament; this reaction is perceptible in females, while the contraction of the sluggish cremaster muscle, which also takes place, is peculiar to males.

On testing this reflex in a case of apoplexy we find it present, as a rule, on one side of the body and absent on the other. Upon the latter side paralysis of the extremities is to be expected, and crossed with it upon the other side we must assume the hemorrhagic or encephalomalacic focus in the brain.

The position of the eyes may aid us in the **diagnosis**; we refer to their conjugate deviation. Both are turned toward one side and upward, and in this position may oscillate so as to simulate nystagmus. In by far the great majority of cases this conjugate deviation is very transitory, and disappears with the symptoms of the attack. The lesion in the brain may be assumed to be on that side to which the eyes have turned.

Before discussing the further course of these diseases we must consider the **etiology**, the **prognosis**, and the **treatment** of the attack.

Here, besides many points of resemblance, we find also many differences.

Apoplexy is a disease of advancing age; atheroma of the arteries of the brain forms a predisposing cause by the development of miliary aneurysms of the finest branches. It is also favored by cardiac hypertrophy—for instance, by a valvular lesion of the aorta—and by a contracted kidney; but it may also be the consequence of the hemorrhagic diathesis, as in purpura or scurvy, or may arise during the course of a severe blood affection such as leukemia or pernicious anemia. In embolism, under some circumstances, degeneration of the aortic valves may permit an embolus to be detached and subsequently to reach an artery of the brain. This accident occurs much more frequently in the young than hemorrhage, and we may therefore state the general law to be as follows: When an attack of apoplexy occurs in a youthful person who is not suffering from a disease of the blood (and this can be readily

determined), and who is also non-syphilitic, embolism is very likely, particularly if there is coexisting cardiac disease.

The suddenness of the apoplectic attack and the rapidity with which loss of consciousness ensues is usually greater in embolism; a protracted form is, as a rule, common to cerebral hemorrhage, in which form, under low pressure, instead of streaming out the blood oozes from a narrow slit. The more profound the unconsciousness and the longer it continues the less hope is there usually that the patient will survive the attack. If in cases of hemorrhage consciousness does not return within the first twenty-four hours—although it may be but momentary and incomplete—the prognosis may be declared hopeless. It is also very unfavorable when tracheal râles or Cheyne-Stokes respiration appear, when the pupils are rigid, when there is decided slowing of the pulse or this becomes very rapid, when the abdomen is retracted and the initial vomiting recurs; when, therefore, those signs are added which we have learned to regard as special symptoms of pressure on the brain. Recovery is possible in embolism even when unconsciousness lasts for twenty-four or forty-eight hours or longer. On the other hand it is a favorable sign if consciousness is not completely lost, or if it soon returns; if intellection is clear from the first day, even though for a few moments only, we are justified in hoping that consciousness will return after a short or long period, that it will constantly become more clear and, finally, that regular alternations of waking and sleeping may be established, the sleep at the onset being still very profound.

Unfortunately, during the apoplectic attack little can be done therapeutically. It has taken place, the lesion has been effected. But a mechanical consideration of the injury furnishes at least an indication as to the treatment.

When the blood has ruptured through the wall of the vessel, nothing prevents its further distribution except the coherence of the semisolid brain mass and the intracerebral pressure. When the former is broken, and the blood finds its way to the free surface of the brain or to a ventricle, a massive hemorrhage always follows and to this the patient invariably succumbs in a very brief time. It is the increase of the intracerebral pressure which produces the symptoms of the attack, and it is the increase of the intracerebral pressure which—apart from the mechanical resistance of the brain substance itself—prevents the further extent of the hemorrhagic focus; and from the fact that these conditions combined—resistance of the brain and increased cerebral pressure—equalize the arterial pressure, not a drop of blood can leave the vessels.

Thus we see how incorrect it is to attempt to decrease intracerebral pressure during the acute attack, as most physicians do, by the application of an ice-bag to the head. This should never be employed until the time when severe brain symptoms show that an improvement of the circulation to relieve the greatly compressed brain is urgently necessary. If we are called to the bedside of a patient and find that an ice-bag has already been applied to the head, we may permit it to remain in order to keep in check the symptoms of the attack.

Venesection which was formerly so generally employed has fallen almost into disuse, and perhaps not quite justly. When the carotids pulsate, when the pulse is full and tense, when the face is flushed and congested, and other

severe symptoms appear, venesection may be performed as a last resource; but it should be thorough so as to give immediate relief to the circulation, diminish the venous pressure, and in this way secure a better circulation in the brain. Of course, with this procedure there is always danger that the power of the heart may be severely damaged, and this cannot fail to lessen the recuperative power of the cerebral activities.

In most cases, therefore, we merely loosen any clothing that may constrict the body, raise the head moderately high if the patient is in bed, empty the rectum by an enema, and await further developments.

Attention must be given to the bladder; many semi-unconscious patients cease moaning only when the paralyzed bladder which is distended to the utmost is emptied by catheterization. After the first twenty-four hours the patient may be cautiously raised to a sitting posture so that the lungs may be examined, and we then note whether hypostatic pneumonia, which so frequently sets in on the side opposite to the lesion of the brain, is present. If this has occurred the patient must be placed upon the healthy side; therefore, with a right-sided pneumonia, upon the left side. At the same time we must take the utmost precautions to prevent the formation of bed-sores upon the sacrum; in many cases these develop very rapidly and severely, and if practicable it is always wise to put a patient who has been attacked by apoplexy upon an air or water bed.

In embolism, theoretically and apart from general measures, entirely different methods are necessary.

It has been demonstrated that in embolism of the cerebral artery the circulatory disturbance is less when the venous pressure is increased; therefore a low position of the head, besides stimulating its power, is always indicated in embolism of an artery of the brain.

It is unfortunate that in the rarer cases of these diseases it is impossible at first sight to make a differential diagnosis with the slightest degree of certainty!

It has already been stated that hemorrhages and encephalomalacic foci have their preferred seats. These are found in the areas of the artery of the Sylvian fossa which supplies the internal capsule and the basal ganglia, and in accordance with well known laws, destruction in this region brings about crossed hemiplegia, a paralysis of the arm and leg and the lower branches of the facial nerve, probably also of the hypoglossal nerve.

Embolism manifests a peculiar preference for the right side. Occasionally the paralysis may rapidly and totally disappear in days or weeks, and it is this fleeting character of the focal symptoms which preëminently distinguishes it. On the other hand, paralysis due to hemorrhage may often markedly improve, even late in its course.

The hemorrhagic focus not only destroys and displaces everything in its immediate area but also presses upon its surroundings, and as it decreases in size the pressure diminishes still more, and those tracts may then recuperate which—lying in the internal capsule—resupply the arm and leg which have been paralyzed for weeks. This distant action does not arise from the encephalomalacic focus; only the regions which it immediately occupies are destroyed, and this damage is absolute, permanent, and irreparable if, within the first forty-eight hours, a collateral circulation does not bring a sufficient

amount of blood to the brain substance. It may be remarked in passing that this absence of distant action is the reason why the encephalomalacic focus is such an excellent object for investigation in topical diagnoses of diseases of the brain.

The subsequent treatment of apoplexy, the treatment of the remaining paralysis, should be about as follows: After from two to three months an attempt may be made by galvanization of the brain to repair the damaged tracts and to decrease the hemorrhagic focus. Only large, well moistened electrodes should be employed. The anode is usually applied upon the side where the hemorrhagic focus exists, the cathode upon the opposite side, so that the focus is in any case within the current. The current must be very weak; the physician himself should test its strength by placing the two well moistened electrodes upon his temples while his eyes are closed, and the current is then slowly increased until a distinct effect of light is produced in his eyes. Even this current is much too strong, and should not be at once applied to the diseased brain. On the contrary, we should apply electrodes without any current, and then use a rheostat so that the previously determined strength may be increased or diminished at will. The duration of the treatment should be from two to three minutes, and this is to be repeated at first twice, subsequently four times a week. Systematic exercises in walking and in movements of the arm are about the only measures by which we can hope to restore motion in the paralyzed limbs; exercises in speech may also be practised. The atrophy of inactivity may be combated by faradization of the muscles which may be begun about four weeks after the attack; above all, we must endeavor to prevent what proves in most cases a great hindrance to the restoration of motion; namely, secondary contracture of the paralyzed limbs.

After a few weeks rigidity appears in the paralyzed extremities of the hemiplegic and the arm is adducted, rotated inwardly, flexed at the elbow, the forearm being pronated, the fingers and thumb flexed.

In the same manner the leg becomes stiff, flexed at the knee, the foot assuming the equinovarus position.

Heinrich Munk recently conferred a great service upon mankind in proving by experiments upon monkeys that this contracture is chiefly the consequence of inactivity, and that it may be prevented by systematic passive movements. If, as soon as stiffness in the muscles becomes noticeable in an animal that has been operated upon, the affected parts are extended twice daily for about five minutes, but slight resistance is afterward present, and contracture does not follow. If we suspend these extensions, the stiffness at once increases, and subsequently passive movements have no effect in producing the former slight relaxation. Therefore in man, soon after the attack, say in about two to three weeks, we begin systematically to extend those muscles which are liable to contract, stretching them twice daily for from five to ten minutes, and we persist in these endeavors for months because, fortunately, it has been proven, of course in only a few instances, that contractures which according to experience would have certainly followed may in this way be actually prevented.

A person who has suffered an attack of apoplexy is, as a rule, prone to be anxious, and the advice which we give him to avoid all excitement, all bodily exertion, all errors in diet, constipation, any extreme effect of heat to the head,

particularly the use of alcohol in concentrated form, is probably followed in most cases as carefully as possible. Although relapses occur so frequently, and the patients succumb in the second or third attack, this is not due to the indiscretion of the patient or the physician but rather to the nature of the case, to the atheroma, to the valvular lesions, to those conditions which produced the first attack, and are just as likely to cause a second and third, and the final, fatal one.

THE SYMPTOM-COMPLEX OF APHASIA

By C. WERNICKE, Breslau

I. DISTURBANCES OF PHONETIC SPEECH

A woman, aged 46, the wife of a tailor, presented on examination a remarkable defect. She had received the average education of people of her class; at least, it was certain that she had been able to read fluently and to write. All signs indicated a focal disease of the left half of the cerebrum. There was right-sided hemiplegia combined with severe sensory disturbance of the entire right half of the body. In the course of nine months, at first slowly developing, an increasing weakness of the right hand had appeared, followed by three acute exacerbations of the disease, the second being followed by a periodical loss of the power of speech. After the third and last exacerbation her condition remained unchanged. Only a slight disturbance of speech was found, namely, some difficulty in articulation, which did not affect the distinctness of expression. This condition of moderate *anarthria* could not be left entirely out of consideration, yet at the present time it can be disregarded in the investigation of her case. The woman was thoughtful, attentive, and apparently of normal mental grasp. She answered all questions intelligently, a proof that she had understood what was spoken and had followed a definite train of thought. She read aloud fluently, and it was evident that she understood what she read. Every letter, every figure, was read correctly without the least hesitation, and she recognized and immediately understood all pictures, diagrams, and outlines. On the other hand she had completely lost the ability to write. She was under prolonged observation which gave her abundant opportunity for practice of this kind; the only result was that once, under urgent persuasions and with visible subjective difficulty, she wrote upon a slate at dictation the small letter *a* and the figures 2, 3 and 4. The art of writing spontaneously without dictation was wholly lost. This was, therefore, a case of quite isolated loss of a previously possessed power of writing, a classical case of so-called pure or isolated *agraphia*.[1]

Here we must naturally consider that the right hand, although not wholly paralyzed, was generally incapacitated. There was complete tactile paralysis as far as all the finer movements were concerned, so that only gross massive movements could be performed. Nothing further was noted. But the art of writing, as we know from experience, is by no means entirely confined to the right hand. Normally every person who has learned to write can also learn to write with the left hand; less easily of course than with the right, much more slowly, and in a more painstaking way, as if drawing. In this writing, as we must admit, there is a different technic of individual movements, but it may be done without extreme difficulty, and each letter and word may be legible. Usually awkwardness is noted only upon the first attempt, and comparatively good results follow a little practice. In the case of this woman, however, there was an almost absolute lack of power to write syllables, words, or even letters alone. As the power of massive movements of the right hand had been retained she was persuaded to grasp with the right hand a small block of wood to which chalk was attached. Standing before a blackboard with this block of wood in her hand the patient could write nothing, although she was able to lift the right hand and guide it with the left.

[1] *Monatsschr. f. Psych. u. Neurol.*, Aprilheft, 1903.

The peculiar psychical condition of the patient on any attempt to write deserves mention. Her expression and posture denoted extreme embarrassment and helplessness. She seemed lost in thought for a few minutes, was coaxed, made several efforts, and finally exclaimed that she was tired and could try no more.

The apparently perfect analogy of this peculiarly circumscribed mental defect with the more frequent and familiar pathological picture of *aphasia* is so remarkable that the case is especially suitable as an introduction to the questions concerning this realm of disease which are still so much discussed. That in this case the faculty of writing was lost in consequence of a local disease of the brain is at least no more noteworthy than the familiar experience that the power of speech may be similarly lost; if we adhere to the definition given by Broca (the discoverer of a *circumscribed center in the brain for human speech*) of the resulting speech disturbance, the analogy appears complete, for in his patient the power of articulate speech, the faculty of speaking, which we know must be acquired like the art of writing, had been lost, and this isolated defect could no more be explained as a paralysis of a group of muscles than in our case. Therefore, if we may assume that these defects are of kindred nature, we are also forced to assume a *writing center* as well as a special *speech center* in Broca's convolution. As a matter of fact many authors have drawn this conclusion from their clinical experience; among them we find no less a one than Charcot—the man who, as long as he lived, was regarded as the intellectual leader in Medicine in France.

Nevertheless, a closer study of the entire symptom-complex of aphasia as well as of the symptoms of the case under discussion shows that if we admit an actual writing center we have already passed beyond the most obvious assumption, and that this assumption is by no means borne out by autopsy findings. We should be particularly cautious in the study of aphasia, for a too hasty and erroneous generalization of facts in themselves strictly correct and comprehensive is dangerous, and has caused every attempt at localization in aphasia to be regarded as Utopian. I must call to remembrance the fact that the discovery of Broca (1861) occurred at a time when the principles of cerebral pathology taught by clinicians such as Bouillaud and Andral were directly opposed to those of physiology as taught by Flourens. Where the first exponents pointed to facts which apparently favored the varying importance of different parts of the brain, the latter was inclined to ascribe these to the effect of Gall's teachings of phrenology which have quite properly been discredited, and experiments in animals were referred to as proof of the resemblance of the animal cerebrum in all of its parts to that of man. Hence a clinician who disputed the truth of Broca's discovery found a soil well prepared by skepticism. When Trousseau expressed his doubts, he found scientific opinion easily swayed to his side. The celebrated discussion of this subject which arose in 1864 in the French Academy of Medicine is an instructive illustration of this. But it was at the same time evident that in the defect which Broca called aphemia he meant something very different from what his opponent, Trousseau, understood. Pure cases of Broca's aphemia, as Broca expressly maintained, are characterized only by the "impossibilité de parler" without other disturbance. This patient, according to Broca, lost "la faculté d'articuler les mots," "le souvenir du procédé, qu'il faut suivre pour articuler les mots." Broca expressed himself still more clearly when he characterized the process by which

the child learns to speak, as follows: "Par le développement d'une espèce particulière de mémoire, qui n'est pas la mémoire des mots, mais celle des mouvements nécessaires pour articuler les mots." Now there was no reason why Trousseau should not develop a picture of aphasia differing entirely from that characterized as aphemia, which is very rarely seen in a pure type; and he pointed out the many remarkable and perfectly incomprehensible phenomena, at that time observed in these patients. But these cases were of entirely unlike character; it is true they seemed to belong to the same aphasic symptom-complex, as we understand it to-day, but they were vastly different from the condition described by Broca as aphemia. If the necropsy findings in many of these cases showed Broca's convolution to be uninjured, we to-day are inclined to regard this as favoring rather than opposing Broca's localization. The presumable proof of the contrary, which Trousseau evolved from the varying necropsy findings, therefore related to an entirely different clinical picture. In addition, numerous cases in which Broca's symptom-complex was observed as an indirect focal symptom could not at that time be satisfactorily explained, and these also appeared to contradict Broca's discovery. This led to the inevitable and undesirable result that the symptomatology of aphasia was increased by the addition of many individual points; but these, although a fundamental gain, and although at first welcomed with great enthusiasm as marking an epoch of advance in the knowledge of cerebral functions, were finally considered to be unproven and were discredited. Not until an enormous impetus was given to the study of the anatomy of the brain by Meynert, and to the understanding of the experimental physiology of the brain by Fritsch and Hitzig, was a more profound interest awakened in the clinical picture of aphasia in Trousseau's sense, as well as a correct appreciation of Broca's discovery. Even the name aphemia, employed by Broca in its restricted sense for the previously described clinical picture, had been relegated to oblivion.

If we recall that it was chiefly the hope of discovering a localization for cerebral functions that introduced Broca's discovery, which was vitiated by Trousseau's influence, we will be inclined to ascribe a paramount rôle to the experimental proofs furnished by Fritsch and Hitzig that there are motor points in definite areas of the animal brain which may be stimulated, and that this led to the consideration of aphasia in the sense of cerebral localization. But even Hitzig's experiments would have proven incomprehensible, and he would probably have found no adherents, if Meynert had not given us such ingenious and convincing descriptions of the anatomical structure of the human and animal brain as to enable us clearly to understand the function of the cerebral cortex. We are indebted to Meynert for our conception of "the projection systems" which in a physiologic sequence embrace, on the one hand, all of the sensory and motor tracts leading from the organs of special sense to the musculature, and, on the other hand, also to the cortex of the cerebrum. While stimulations from the body can be conveyed to the "sensory sphere" of the cerebrum only through this tract, the impulses which arise in the latter are projected to the muscles. According to Meynert, a transverse section of the cerebral peduncles would include the entire organism, which "would be devoid of the senses of smell and sight." The ganglion cells of the cortex of the cerebrum have everywhere the same elementary function,

and only from the variation of their combinations with the periphery of the body by means of the projection systems are their specific qualities arising from different localities of the cortex manifested. From anatomical investigations, Meynert came to the conclusion that the cerebrum may be divided into two large territories, an anterior one of motor, and a posterior of sensory, importance. Memory pictures produced by this function, and which fill the cerebral cortex, are motor in the anterior structures and sensory in the posterior.

Viewed from this standpoint, the movements which Fritsch and Hitzig produced by galvanic irritation of the anterior portion of the brain of a dog appeared in a new light. They did not resemble those evoked by irritation of special peripheral nerves, but involved several muscle groups, and produced distinct movements simulating those which are voluntary. Both of Hitzig's extirpation experiments, which were at once published, permitted the interpretation that the resulting disturbance of movement depended upon loss of motor memory pictures or conceptions of movements, and Broca's aphemia also appeared to be a loss of motor memory pictures, particularly for the movements of speech which had been acquired. The time at which a child learns to speak is especially the time for this functional acquirement of motor memory pictures, or, more definitely stated, "du développement d'une espèce particulière de mémoire, celle des mouvements nécessaires pour articuler les mots." It will be observed that Broca, with the remarkable acuity of great discoverers, had taken into consideration only those functions of definite parts of the brain which (in Meynert's opinion this was subsequently fully confirmed) more nearly corresponded to the motor area of the cerebral cortex.

Nevertheless, the remarkable variety of the clinical forms of aphasia constituted a fact which demanded consideration and explanation. Here also the ground was broken by Meynert, for this author believed he had found in this peculiar structure (in his opinion the claustrum was entirely composed of association cells, its area of distribution being in the island and its cortical convolutions) the central termination of the auditory nerve, and at the same time a sound area for speech. Although the auditory nerve could not be traced up to the island of Reil, nevertheless, the significance of this nerve and of the island for the function of speech called attention to their previously ignored connection.

It is evident that, after Meynert's investigations, we had all the prerequisites for the full and comprehensive understanding of aphasia. My work upon "The Aphasic Symptom-Complex" which appeared in 1874, was merely a minute explanation of Meynert's theories and their special application to human speech. In the anterior region of the brain, Broca's convolution, we possess a *motor speech center* which we regard as the region of motor memory pictures of speech, or conceptions of speech movements, and the origin of speech impulses; in a posterior region we assume a *sensory speech center*, the region for pictures of speech sounds, and at the same time the terminal point of the auditory nerve. The child's brain receives and stores up memory pictures of the sounds of speech which it has heard and gradually imitates these; thus speech becomes the functional acquirement of every individual. Combined fiber masses, "association tracts," between the sensory and motor speech centers, transmit this act of imitation, and also the power of speech.

From general anatomical considerations which permit us to regard the entire island covered by the first embryological convolution arches as an anatomical entity, I believe the sensory speech center to be situated in the first temporal convolution, and the postulated association tract in the association organ described by Meynert as located in the fiber system extending through the cortex of the island, the deepest layer of which loses itself in the claustrum. Fortunately the findings at two autopsies confirmed this presumption that the sensory speech center was in the first convolution. In both cases the posterior half of the longitudinal course of the first temporal convolution and a closely adjacent area of the second temporal convolution were destroyed by a focus of softening. This, of course, permitted the differentiation of three clinical forms of aphasia, a *motor*, a *sensory*, and a third form which I designate *conduction* aphasia. The motor form had already been clearly described by Broca; it was the aphemia of this author. Sensory aphasia was chiefly characterized by the fact that the sounds of speech were heard, and the power of articulate speech was retained, but the speech sounds were not understood. This symptom, the confounding of words (the *paraphasia* of Kussmaul), was explained by the hypothesis that the sound pictures of phonetic speech presumably regulated the speech. The clinical existence of conduction aphasia appeared to be proven by cases of paraphasia in which both the power of speech and its understanding were retained. The article published in 1874 explained the seeming contradiction, which, according to Kussmaul, still existed in a description regarded as classical, and which was opposed to any localization of speech. After the opinion had been expressed that Broca's convolution was the only speech center, and this was assigned its proper position as being exclusively the center for motor speech, skepticism, which had opposed further efforts at localization, at once gave way. With the appearance of Kussmaul's book, which in a clinico-symptomatologic sense is still valuable, skepticism was routed. And as Meynert's point of view in regard to *one* realm of the pathology of the brain was accepted, that one which had heretofore been regarded as especially difficult and impenetrable, this opened the way to further revelations of the secrets of the brain, and to expectations which have not failed of realization.

The clinical picture of sensory aphasia with its localization in the first temporal convolution and the adjacent parts of the second temporal convolution was soon generally recognized. Kahler and Pick, subsequently Lichtheim, also French, Italian and English authors, helped to bring about this recognition, and we maintain to-day that the clinical picture of this special form of aphasia and the necropsy findings are just as surely based upon facts as is Broca's motor aphasia. It, therefore, becomes my duty to emphasize that the chief symptom of sensory aphasia, arrest of the understanding of speech with retention of the power of hearing, had already been recognized by two authors, by Schmidt[1] upon the basis of his own observations, and by Bastian[2] after reports of strange cases. These authors did not ascribe defective speech to a definite locality of the brain; hence we are constrained to admire their acuity which enabled them to grasp the true

[1] *Allg. Zeitschr. f. Psychiatr.*, 1871, XXVII, p. 304.

[2] On the various forms of loss of speech in cerebral disease. *British and Foreign Med. Chir. Review*, April 1869.

condition while ignorant of Meynert's point of view and the process of its development.

As already stated, the most important point is proof (which I attempted to demonstrate) of the fact that the aphasic symptom-complex embraces the conflicting clinical pictures of motor and sensory aphasia, and the anatomical localization of these contrasting conditions has been proven to be in two entirely different areas of the brain. Historic retrospect here teaches us but little, and is therefore unnecessary. But an essential and very important advance was made in this direction when we arrived at a clear understanding of the relation which must be assumed between the diametrically opposite functions of these two centers, and upon this standpoint we base the following description.

The *sensory speech center* is the region in which, corresponding to the central termination of the auditory nerve, the memory pictures of speech sounds that have been heard, sound pictures (Helmholtz), have their anatomical substratum in the ganglion cells of the cerebral cortex, "cortical units" (H. Sachs).

Hence this transmits the *understanding of word sounds* (Liepmann), the recognition or the "identification" of the perception of sounds which form the word, a function which must be strictly differentiated from the understanding of the "sense of the word." The mere thought of a foreign language enables us to understand the necessity of this differentiation, for we learn to comprehend our mother tongue in the same way in which we learn a foreign language. Most children understand spoken words, and even their meaning long, even years, before they acquire the power to articulate these words. From this, as we shall soon see, a certain independence of the sensory speech centers arises, in comparison with the much more dependent motor center. But we must admit individual variations in this duplex relation, since undoubtedly there are children whose power of articulation keeps pace with their understanding of word sounds. The words which the child learns to speak have at first nothing to do with the artificial sound formation of written language. There are complicated words the sound picture of which, as well as the speech movement conception thereof, must be slowly acquired by practice. The possession of both of these memory pictures, intimately combined for each word, is what the French authors have designated as "*internal speech*," which suggests also a mental power, a kind of memory —and this may be likened to Broca's definition given above. We will subsequently see that a firm combination of associated memory pictures constitutes for us the nature of conception. For this reason I proposed the designation *word conception*, recognizing the acquirement of word conceptions to be the most important process in learning to speak. Déjérine has accepted this view and employs the phrase "*notion du mot*." The expression "speech conception of words" (Caro) appears to me to be happily chosen, for the correct articulation of words, the intactness of both components of word conception, the sensory as well as the motor, is absolutely necessary. Hence, if only the method of learning to speak is unquestionable, it is nevertheless very doubtful whether subsequently the impulses for movements of speech are not referable to the tract through which speech was originally learned by imitation. A number of authors hold this to be so self-evident (I shall

mention only Kussmaul and Bastian) that they scarcely deign to discuss the question. Yet clinical experience up to the present time is overwhelmingly opposed to this view. As we shall soon see, these are chiefly experiences of sensory aphasia attributable to destruction of the sensory speech centers. We must therefore assume that the main impulses of speech are directly transmitted from the remaining cerebral cortex to the word conception, and when this is deranged by an absence of the sensory center the impulse is transmitted to the speech movement conceptions; hence the power of articulate speech is retained, but is disturbed only because of the lack of a regulating influence of the sensory speech center which determines the choice of the correct conceptions of movement.

This description enables us clearly to understand the clinical picture of **sensory aphasia** as well as its principal characteristics.

(1) The comprehension of the sound of words is defective, and as this comprehension by way of the hearing is a prerequisite to the understanding of the sense of words the comprehension of spoken words is also defective. In any case the defect is purely acoustic, and the investigator is very liable to fall into error if he attempts to facilitate the understanding of the sense of the word by signs, looks or gestures. If, however, by refraining from this, it becomes evident that none of the speech sounds heard by the patient have been understood, he may prove by means of these signs and gestures that the hearing is still intact or, if defective, that the degree of deafness present by no means explains the defective comprehension of the sense of words. As is well known, even with extreme peripheral deafness the ability to understand the sound of words is usually unimpaired.

(2) The power of articulate speech is retained, the patients' speech even being remarkably voluble, perhaps in consequence of the frequent misunderstandings to which they are exposed. For, although they speak rapidly, use a comparatively rich vocabulary, and form their phrases properly, they frequently make mistakes in the choice of an expression, or employ incorrect or distorted words without observing it. Under excitement speech is sometimes much better than at others, and entire sentences may be spoken perfectly. Objects shown them may be incorrectly named, or distorted words are frequently used in naming them. Their confusion of words in spontaneous speech may so increase that it is impossible to understand them, and we then designate their speech as *jargon aphasia*. On attempting to answer questions these patients never use words in the same sense in which they have heard them. Their inability to comprehend the sounds of words apparently also makes them incapable of imitating the sounds they have heard.

(3) I shall not here discuss written language, since it is not yet the common property of mankind, and consequently it often fails to throw light upon the clinical picture. We shall see later that this ability to write is combined with an intact power of internal speech, what we have called the conception of words, and therefore is invariably and severely damaged in sensory aphasia.

Sensory aphasia in the overwhelming majority of cases is due to the occlusion of vessels, therefore, usually has an acute beginning. As a rule the symptoms of the attack are slight, sometimes entirely lacking, and there may be no sign of unilateral paralysis. As to the prognosis of the disease, views

differ. Déjérine concluded from his observations that there is usually a permanent defect; the majority of authors, with whom my experience leads me to coincide, are of the opposite opinion, and believe that a comparatively rapid compensation of the defect is the rule. In fact the difficulty appears to remain at its acme only for a few weeks, when, provided no intercurrent condition appears, it gradually disappears in the course of a few months. Thus, by means of a returning sense of hearing, pictures of word sounds are newly acquired, and those sound pictures are permanently lost which the daily intercourse of the patient gives him no opportunity of using. I have repeatedly succeeded in demonstrating traces of the preceding disease by proving the patient's absolute inability to understand words or phrases in rare use, such as vertebral column, popliteal space, axilla, and the like. The inability to name correctly any objects presented to view appears to persist for a long time. The conditions in regard to the restitution of written language have not been thoroughly studied.

No less easy is the description of **motor aphasia**, the aphemia of Broca. In adhering to the preceding train of thought the following symptoms are prominently revealed, so that the clinical picture is easy of recognition.

(1) The power of articulate speech is wanting. The patients have forgotten the process, the mechanism, which they formerly called into action to produce its sounds. They are therefore mute—at least, they have only a minimal power of speech. In some typical cases even this is lacking, and nothing remains but the power to make and repeat a few inarticulate sounds. The defective speech most frequently observed consists of senseless syllables, perhaps of a few words or phrases, or it may be some profane or emotional expressions.

These few words, monotonously repeated, to which speech is limited are not articulated voluntarily and exclusively to express what they mean, but they appear as an invariable reaction to all the demands which are made upon the patient's power of speech. It may happen after some exceptional emotion that these patients, who are usually mute, will give utterance to a natural expression, as a rule merely an interjection, yet subsequently they are unable to do this voluntarily. It has also been observed that during sleep words and phrases are uttered which the patient at other times is incapable of enunciating. As a rule, the faculty of speech is entirely lost. This loss of speech cannot be ascribed to any paralysis of the muscles, for a test of their functions excludes all forms of bulbar paralysis.

Often we find as an accompanying phenomenon a hemiplegia or hemiparesthesia, a unilateral weakness in the right lower facial or lingual region, sometimes only in the region of the right lower facial or right hypoglossal branches. A clinical symptom of the latter is the deviation of the tongue to the right whenever it is protruded. As these signs of paralysis also accompany articulation which is really unimpaired, they have merely the significance of an accompanying symptom, as has been sufficiently demonstrated by clinical experience.

A certain impairment of the mechanism of speech which we must assume to be analogous to defective speech is so common as to demand attention. This is manifested by the impossibility of producing complicated movements in the region of speech muscles. Thus, some of these patients cannot put

out their tongues, cannot inflate their cheeks, cannot gnash their teeth, or they are unable to open their mouths upon being bidden to do so, without simultaneously protruding the tongue, etc. These futile attempts at speech movements, which could be previously performed with ease, permit us to recognize most distinctly the patient's inability to perform the required maneuvers. According to Broca's excellent definition they lack for this "le souvenir du procédé qu'il faut suivre." Sometimes these symptoms occur only during the early and acute stages of the disease.

(2) In the main, the power of understanding speech is retained; at least this appears to be the case on ordinary tests. The faulty reaction may in part be referred to an impossibility of movement, analogous to that in the case just described and of the same significance. Therefore all orders given may be promptly carried out, objects asked for may be produced, questions, to judge from the expression of the face, are clearly understood, and the like.

There is almost invariably a certain inability to understand complicated constructions and the finer differentiations of speech, as was first pointed out by Déjérine, and illustrated by numerous convincing examples. Here the contrast to sensory aphasia is so marked that a sharped differentiation is justifiable, and the sensory defect is amply compensated for as soon as we furnish the necessary mental stimulus and corresponding surroundings. I no longer am of the opinion that in pure motor aphasia the ability to understand speech always remains unimpaired, because I consider the acquirement of word conceptions an important phase in the process of learning to speak. Since, in this respect, we must admit a decided individual difference, proper allowance must be made for this in examining the patient and in estimating his case.

(3) What is true of sensory aphasia is also true of written language. If the individual in question had previously possessed this faculty it is lost simultaneously with articulate speech.

Motor aphasia like sensory is a pathologic picture which is most frequently of acute development, usually with the symptoms of apoplexy and more or less profound right-sided hemiplegia.

The prognosis *quoad restitutionem* is generally unfavorable unless the affection is transitory and functional, i. e., unless there is an embolism which is compensated for by the immediate production of a collateral circulation or is an indirect focal symptom in which motor aphasia is only the secondary effect of a focus in the vicinity of Broca's convolution. This is a frequent occurrence; we sometimes even observe that motor aphasia appears indirectly as a focal symptom with left-sided hemiplegia if extremely severe symptoms of apoplexy accompany it. If there is an improvement in motor aphasia without the previously mentioned exceptional conditions, and this is rare, speech is permanently impaired. I had under observation a patient who, by very painstaking efforts and by methods similar to those used with deaf-mutes, was in the course of years taught to speak and, in the main, could be understood. But the force and exaggeration of his speech processes, the constrained movements like grimaces which involved the entire face, and his slowness of speech, were remarkable. In my experience this helplessness, this recognizable and, to the expert, extremely characteristic want of control

of the "procédé qu'il faut suivre pour articuler" is permanent even in the most favorable cases. On repetition the speech continues to be almost as faulty as when spontaneous. But Déjérine and Thomas have recently reported that under the systematic optical instruction usually practised with deaf-mutes we may often achieve remarkable results even in long-standing cases (fifteen years!) in a surprisingly short time (six weeks!). I shall revert later to this theoretically important point. In comparatively rare cases motor aphasia is not so complete, or after some time it becomes evident that considerable power of speech remains. In such cases it is possible to induce the patient to repeat easy, simple words, although always with evident effort, but he never succeeds with longer or more difficult words or with complicated sentences, and he becomes greatly confused so that, for example, instead of saying "I" he says "you," instead of saying "good" he pronounces his own name, no matter how dissimilar; therefore the most unlike conceptions of movement, if retained in any appreciable degree, are confounded on attempting to speak. An unexpected interjection such as "O God!" may sometimes be ejaculated by these patients. I admit such occurrences, but the clinical resemblance to motor aphasia extends no further, and the contrast between sensory and motor aphasia is evident even to the tyro in medicine.

Of **conduction aphasia**, the third clinical form which I attempted to differentiate from the many aspects of the aphasic symptom-complex, there are as yet but few reports, and even these fail to coincide, hence it is impossible to describe a uniform clinical picture on empiric foundation. I was at first inclined to regard the majority of the cases which did not show the Broca clinical picture but only the symptom of paraphasia or confusion of words as sensory aphasia, because in these forms the power to comprehend speech was retained. This opinion was shattered by finding that I had much over-estimated the possibilities in these cases, and the view was shown to be untenable. Of course certain positive characteristics are necessary, particularly the loss or impairment of word conceptions, the clinical signs of which I shall subsequently minutely consider, as well as the impossibility of repeating words; according to the views developed above, the tract which serves for the repetition of acoustic word sounds is especially implicated. In both directions positive reports are wholly lacking, and I therefore limit myself to one important observation. If, under some circumstances, the mere sound of a word is sufficient for its full understanding—and, as shown above, this is true of the majority of words frequently used—and if, on the other hand, words can be spontaneously spoken without the previous stimulation of the sound picture, after interruption of the association tracts between the sensory and motor speech centers it should still be possible to repeat at command words whose meaning is understood. But this repetition will not be so infallibly successful as in the tract developed in childhood while learning to speak; it will be partially correct and partially paraphasic. When paraphasic, the patient will notice his error and endeavor to correct it. It is positive proof that this earlier tract is unimpaired if the patient can immediately repeat on request, so-called *echolalia*, which is purely automatic and often evoked without command at the moment of speaking, and also if he is able to repeat incomprehensible, meaningless words or phrases, for exam-

ple, words from a foreign language. If this particular form of repetition is impossible, yet on the other hand the power of speech and its understanding be retained, also paraphasia and the ability to criticise errors that have been made, I consider the clinical requirements to have been sufficiently fulfilled. I cannot, however, refrain from emphasizing that the autopsy findings (for example, exclusive or predominating destruction of the island) are not calculated to support the view of conduction aphasia postulated by me.

If, as I believe I have demonstrated, the assumption of a sensory and motor speech center has been corroborated by anatomical findings, as well as by clinical observations, it follows in logical sequence that still other clinical pictures must be embraced in the rich aphasic symptom-complex. Lichtheim was the first to recognize clearly this logical principle, and he also deduced the necessary corollary. In rare cases, and the pure cases are always rare, it seems possible that the medullary substance which contains the projection fibers of both centers is alone interrupted by a focus, while the centers themselves remain intact. If these centers still exist in combination, a condition which of course can only rarely be possible, a new clinical type must result bearing some resemblance to the previously described ones inasmuch as it presents the fundamental symptoms, loss of power to understand spoken words and the power of articulate speech; they have, however, the differentiating factor of retained "internal speech," the intact "word conception." As a matter of fact the corresponding clinical picture in the motor region is not so infrequently met with, especially if there is simultaneously hemiplegia. The counterpart, sensory aphasia, is much rarer but has unquestionably been observed. In the light of anatomical researches the designation of *subcortical motor* or *sensory aphasia* would be applicable to these two types of disease. According to clinical criteria the designations "*pure word mutism*," and "*pure word deafness*," are more wisely chosen.

PURE WORD MUTISM differs from cortical word mutism due to destruction of Broca's convolution in the fact that efforts to speak, although futile, permit us to recognize the undamaged "internal speech" or "word conception" even though merely from the sound or rhythm. This is most apparent when the power to produce sounds is to some extent still present. Never do such absolutely dissimilar reactions occur on trying to repeat words as in cortical motor aphasia. What is spoken is perfectly understood, even when complicated demands are made upon the faculties. Moreover, the ability to write is unimpaired, the patient reading and writing without effort, the latter, in spite of the almost invariable accompaniment of right-sided hemiplegia, being somewhat clumsily done with the left hand. The mechanical power of translating the intact word conception into sounds has simply been abolished. Similar conditions prevail with PURE WORD DEAFNESS; here also the entire internal apparatus of speech, both the cortical centers, and their simultaneous action, so that the word conception remains clear, and spontaneous speech is absolutely free and coherent, the power of written expression does not suffer, and that of reading is unaltered. The understanding of the word sounds, of what has been spoken, is alone lacking, an adequate power of hearing being evidently retained.

We must now investigate somewhat more minutely both of these clinical

pictures with which we have become familiar, not only on account of their theoretic interest but on account of the diversity of opinions concerning their recognition and correct appreciation. *Pure word mutism*, or *subcortical motor aphasia*, is undoubtedly of vast importance in both of these disturbances. Long recognized as a clinical picture, just as Trousseau recognized the remarkable fact that aphasic mutes could occasionally still read and write, these rare cases were considered to be the purest examples of motor aphasia, since, according to Charcot, there was a special center for reading and writing. We are indebted particularly to Déjérine, who was the first to obtain autopsy findings which, contrary to the opinion of Charcot, absolutely proved the true nature of these cases and the existence of a subcortical form of motor aphasia, that they were brought into general recognition.

In regard to the anatomical location exclusively involved in the subcortical interruption of the corona radiata fibers coming from Broca's convolution, authors are quite unanimous. Only the medullary layers of Broca's convolution itself or its point of entrance at the centrum ovale can be considered; if the projection fibers lower down and within the trunk of the brain are affected, the picture of motor aphasia is no longer produced but disturbances of articulation prevail, while there is still a distinctly recognizable use of words; this is probably due to the position of the fibers, but of this we have as yet no exact knowledge. The uncommonly frequent right-sided hemiplegias from focal disease of the corpus striatum or the internal capsule are usually unaccompanied by motor aphasia, or at most it is of exceedingly brief duration and must be regarded as an indirect focal symptom. The facts which led me to assume a special motor speech tract extending continuously from Broca's convolution to the nuclei of the nerves of speech movement in the medulla oblongata and skipping the internal capsule, I shall refer to subsequently. Here I limit myself to the mere mention of the question of an exclusively unilateral innervation of the bulbar apparatus in speech; this question must later be discussed, but now it would lead us too far afield. The intactness of word conception is a sign which permits us to recognize this form also in those who cannot read or write. Lichtheim proposed a method by which the patient informs us of the number of syllables in a word which he understands but cannot speak because of his infirmity; for instance, on being shown an article to be named, the patients are told to squeeze the hand of the physician as many times as there are syllables in the word. The objection may be raised to this that those who do not know how to spell are often ignorant of the number of syllables in a word. For this reason the ability to count has been accepted as evidence of retained internal speech (Fränkel and Onuf). A far more reliable guide it appears to me is that the sounds which are indistinctly articulated permit us at least by their rhythm to guess the word intended. The same aids must be resorted to if, in advanced cases of progressive bulbar paralysis, we desire to demonstrate the intactness of "internal speech."

A somewhat more elaborate description must be given of *pure word deafness*, or *subcortical sensory aphasia*. That this in its perfect purity is not rare in the clinical picture above described is acknowledged by all. The objections to it which have been made refer, on the one hand, to the postulated unilateral position and the subcortical seat of lesion, and, on the other hand,

to the possibility which has been maintained of the peripheral development of the pathologic picture from disease of the terminal distribution of the cochlear nerve in the labyrinth of the organ of hearing. The question of this unilateral position will be more minutely discussed later. But there can be no doubt that the motor as well as the sensory speech center is generally unilateral and situated in the left hemisphere. For the motor speech center —and this has been universally done—acting on the principle of a saving of labor, we may assume that a facility in learning to speak, being combined with a symmetrically acting musculature, stimulated from each hemisphere, is also derived from only one hemisphere. We must also assume anatomically similar conditions and relations in the projection system and the cortex for both auditory nerves, in that each hemisphere contains a central projection field for these. But it is difficult to believe that this should occur in the act of sensory projection in only one and that the left hemisphere, and that only in this are there memory pictures of speech sounds that have been heard, as well as a center for the understanding of word sounds. The facts of cortical sensory aphasia permit no other explanation, and since we must be content with this plausible principle of a saving of labor, recent investigations have shown that for the acoustic picture memory, and even the simplest perceptions of tone, a motor component is admixed from the start so that the labor saving principle, or, more accurately expressed, the conservation of motor nerve activity, must also be operative in acquiring acoustic memory pictures. At all events, if a center for the understanding of word sounds is unilaterally located, it also seems possible that the projection fibers extending to it may be unilaterally interrupted, and the conduction path to the center for the understanding of word sounds be blocked, while the intactness of the same projection fields of the other hemisphere may permit other tones and sounds to be perceived and correctly appreciated.

A few anatomical considerations are justifiable. The clinically indisputable connection of each auditory nerve with the hemispheres must have its anatomical substratum in a semi-decussation of the auditory nerves. As such area, only the posterior field of the medulla oblongata can be considered. Later the posterior corpora quadrigemina and the internal geniculate body form primary centers of hearing (analogous to the primary centers of sight in the anterior corpora quadrigemina and the external geniculate body), hence the posterior superior peduncle, that is, the pedicle of the internal geniculate body from the first temporal convolution is certainly the portion of the tract here under discussion. Besides this peduncle of the internal geniculate body, it is possible that a special portion of the optic thalamus from the temporal lobes also belongs to the central auditory tract. This can be situated only in the immediate vicinity of that previously mentioned. It is certain that portions of both acoustic nerves are within these areas of the corona radiata fibers of the temporal lobes. That a unilateral focus in this region of the corona radiata fibers of the temporal lobe may produce pure word deafness as a permanent symptom has been proven by Liepmann's undoubted case.

S. Freund has maintained that pure word deafness must be assumed to be the consequence of peripheral disease of the auditory nerve in the labyrinth. He bases this assumption upon his observation of a case in my Clinic,

which he has, however, incorrectly reported [1] since he ignored the positive symptoms of cerebral disease which are mentioned in the history. Even less conclusive are his two other cases. However, S. Freund has done us a great service in having shown the necessity in such cases of minute examination of the ear by a specialist. Bezold [2] has proven that the majority of the fibers of the auditory nerve are not involved in the comprehension of sounds of speech, and that "in the entire musical scale there remains only the short distance from b′–g″ inclusive, therefore a large sixth, the perception of which is absolutely necessary for the understanding of speech." Liepmann [3] showed that a few tones besides these, either above or below and amounting to an octave, are also involved in the understanding of human speech, so that from the entire series of tones less than two octaves are necessary. In the case from my Clinic which Freund utilized it was deemed advisable to investigate with a continuous series of tones. This was subsequently done, and it was demonstrated that in this patient these two octaves in particular were well retained. In H. Liepmann's case in my Clinic, on which a necropsy was held, the same examination was made and proof established.

The human faculty of hearing appreciates a series of tones ranging through more than eight octaves. Since merely two of these are absolutely necessary for the understanding of speech, and all of the parts of the auditory nerve in the cochlear are uniformly distributed, we come to the conclusion that only the fourth or the fifth part of the projection fibers of the auditory nerve need find its central end in the sensory speech center. This circumstance may possibly explain the relatively slight extension of the sensory speech center; this, according to numerous autopsy findings, extends only to the posterior third or the posterior half of the first temporal convolution and the adjacent area of the second temporal convolution; hence from anatomical and physiological considerations there can be no doubt that the remaining part of the temporal lobe also serves in part as the termination of the auditory nerve.

These newly determined facts force us to assume that the sensory speech center coincides with the termination of those projection fibers which include the series of tones from b′–g″. From this results the postulate that in cases of subcortical sensory aphasia the left temporal convolution would always be actually deaf to the aforesaid tones—if its function could be separately examined, which of course is impossible. In other words, that word deafness, and not an actual although partial deafness for certain tones, results from unilateral lesions, and is explained by the fact that the affected portion of the fibers of the auditory also reach the right temporal lobes, and thus permit the perception of the same tones.

This view also explains the above mentioned possibility of acquiring new acoustic word pictures, and thus compensating for the defect. If the corresponding cortical areas of the second right temporal lobe are subsequently affected, permanent sensory aphasia supervenes, and can no longer be com-

[1] Compare H. Liepmann, "Ein Fall von reiner Sprachtaubheit." *Psychiatr. Abhandlungen*, Hefte 7–8, Breslau, 1898.

[2] "Das Hörvermögen der Taubstummen und Nachträge dazu." Wiesbaden, 1895–1896. *Zeitschr. f. Ohrenheilkunde*, XXXVI.

[3] L. c.

pensated for, as O. Berger has proven by one of the earliest observations bearing on this affection.

If we extend our researches still further, we arrive at the postulate that cases of sensory aphasia from bilateral disease of the temporal lobes run their course with actual tone deafness in the area of the previously mentioned tone heights. The future will reveal whether this supposition is actually correct. But, up to the present time, such investigations in cortical sensory aphasia, the difficulty of which we do not deny, have not been systematically attempted.

On the other hand it becomes quite clear that with distributed bilateral disease of the temporal lobes the picture of word deafness is less prominent than that of general deafness. In the majority of cases the nearest point of termination of the auditory fibers is then destroyed, and this makes possible the development of central cortical deafness. A. Pick has described two such cases in which the patients were so deaf that the almost total inability to understand words was only explained by the fact that extreme deafness from peripheral causes leads to absolute inability to comprehend words. At all events these cases do not show the clinical factors of subcortical sensory aphasia, or pure word deafness, and do not justify Pick's conclusion that pure word deafness is due to cortical destruction extending throughout both temporal lobes. A third case of Pick's[1] which presented besides extreme deafness the essential characteristics of pure word deafness may be readily explained as a combination of both these defects, which opinion was borne out by the autopsy findings.

While we have so far especially considered those forms of aphasia which are generally recognized, either anatomically or clinically, such as cortical sensory and motor aphasia, or those which, as a rule, are regarded as special clinical types, such as the subcortical forms of aphasia, we now come to debatable forms which still require the support of additional observations and anatomical findings. From a purely logical standpoint we cannot reject the conclusion that a *transcortical motor* and a *transcortical sensory* aphasia are possible; i. e., an aphasia caused by an interruption of the tracts which bring about the combination of the two previously assumed speech centers with the so-called region of conception, therefore the greater part of the general surface of the cerebrum. The discussion of these clinical varieties is justified by the fact that there are abundant clinical examples corresponding to the theoretically constructed picture. And although I attach great weight to the opinion of Déjérine, who declares these to be erected on a purely theoretical foundation, I cannot recognize as conclusive the proofs he advances. I shall soon revert to this point. We shall first attempt to develop theoretically the principal criteria of the clinical picture as has been done with the other forms.

We must clearly understand what is meant by the conception of an object or "*the concrete conception*" (Ziehen), for, as already emphasized, the conception of an object must be awakened by a sensory speech or word sound center so that the word sense may be understood. Therefore a *conception*

[1] *Arch. f. Psych.*, XXIII, p. 909.

center has been spoken of; this would be identical with our center for the *understanding of the word sense.* Naturally it is pure fiction, and merely an artifice which diagrammatically simplifies the subject, to speak of such a center. In fact, in these processes which are extensively distributed throughout the regions of the cerebral cortex, we are dealing with conditions such as appear in the following considerations: If we take Meynert's point of view described at the beginning of this article in regard to the regions of special sense, we assume the conception of concrete objects to be the function of different projection fields at the cortex; for example, the conception of a rose is composed of "a tactile memory picture" or "a tactile picture" of the rose in the central projection field of the palpating surface of the hand, of an optical memory picture in the optical projection field, and a smell memory picture in the olfactory projection field of the cerebral cortex. By the constant repetition of the same impressions of special sense so firm an association of these memory pictures is formed that even the stimulation of one sense by the object is sufficient to remind us of the sum of its essential properties; in other words, to awaken the conception of the object. Sometimes there are more, occasionally less, memory pictures of various regions of special sense which correspond to a conception, but it is always a definite and firm grouping due to the nature of the object which forms the anatomical substratum of every conception. This definite sum (which must always be determined) consisting of associated memory pictures, must "enter into consciousness," provided not only the sound of the corresponding word is appreciated but also that its sense is understood. In accordance with anatomical views we postulate also for this act an anatomical tract of fiber communication or an association tract between the sensory speech center, or center for the understanding of word sounds, and the projection fields in which the conception is produced. These tracts we may call *transcortical,* inasmuch as they extend beyond the nearest cortical termination of the auditory nerve in contrast with the generally recognized subcortical tract areas. As is obvious they can be considered only as a radiating bundle that fuses into one terminal point, the sensory speech centers, and only in this vicinity can it be focally separated.

The condition must be quite similar if, spontaneously, i. e., in consequence of internal cerebral processes, the word "rose" is spoken. First the conception must appear, then the impulse of the movement conception corresponding to the word "rose" must reach the cortex of Broca's convolution. For this purpose the analogous "transcortical" tracts are necessary which can only be conceived in the form of a converging radiating bundle. The following diagram represents the relations of a "concrete conception" to both speech centers,[1] and also possesses acoustic factors as, for instance, a bell and a dog.

[1] The definition of a concrete conception here given as the definite grouping of associated memory pictures with each other necessitates only the most necessary assumptions. In my opinion it would be superfluous to assume anything more than this: That for association between the memory pictures of various projection fields special *association centers* are necessary which therefore might anatomically be considered as collective points of association tracts from the various projection fields of the organs of special sense. Flechsig claims that he has proven the existence of such association

According to the preceding we have a perfectly clear definition of the clinical picture of *transcortical sensory aphasia*: An abolition of the understanding of the word sense with a retained understanding of the word sound. The speech sounds as such are perfectly understood. This is shown by the retention of the ability to repeat them, but the accompanying conception is not awakened. The power of speech is actually retained but is slightly impaired in that spoken language is not understood and consequently its correct appreciation cannot be tested. A paraphasia extending beyond this with an admixture of incorrect or distorted words or syllables is usually only suggested. The repetition may consist of entire sentences or may be limited to the last few words according to the degree of the retention of memory, and sometimes discloses no error; at other times there are paraphasic distortions. The repetition may be in obedience to command, sometimes it is involuntary. It may be combined with what has been heard in the form of a question, sometimes it is spasmodic, sometimes almost reflex. In the last case we have echolalia, a symptom which usually indicates a weakening of the sensorium or general intellectual decay. The unintelligible repetition of a question is characteristic of this.

The contrast in the motor realm is *transcortical motor aphasia*, consisting in the arrest or very decided impairment of spontaneous speech, while on repetition it is fluent, perfect, and there is no lack of understanding of language. The speech still at command is not made up of a few words or syllables constantly repeated, as is the case in cortical motor aphasia, but there are occasional expressions of discontent, of anger, of helplessness, therefore emotional expressions; the command of language, as may be proven by repetition, is unlimited, automatic. Series of sentences learned by rote, such as the Lord's Prayer, the multiplication table, and the like, may be smoothly recited by the patient, either at command or after being repeated to him.[1]

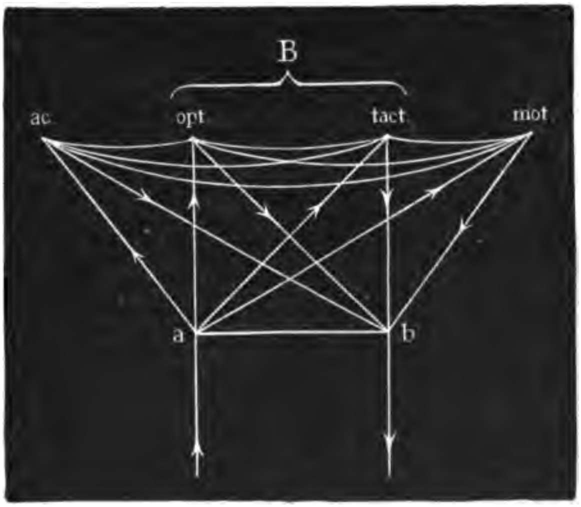

Fig. 137.

centers or "*coagitation centers*" as he calls them. They are said to be characterized anatomically by the fact that no projection fibers or corona radiata fibers enter them. Since then it has been proven that corona radiata fibers extend to all coagitation centers. I can only assume that the anatomical substratum for centers of this kind is in one cortical territory—that is, in the insula with its claustrum which Meynert regarded as an associated organ.

[1] Compare Heilbronner, "Ueber die transcorticale motorische Aphasie und die als 'Amnesie' bezeichnete Sprachstörung." *Arch. f. Psych.*, XXXIV, p. 341.

But the simplest communication, conversation, or difference of opinion, is impossible through speech; only in the rarest cases can questions be answered except by gestures; once in a while it is possible to obtain a single short answer. Articulation is absolutely faultless.

These are the clinical types. They are complemented in some cases, i. e., in those with a fair degree of education, by the no less characteristic impairment of the power to write. Here also there is a striking contrast between the perception of speech and its expression: In sensory transcortical aphasia there is no comprehension of what is read, but, owing to the integrity of word conception, reading may be fluent and without visible effort; in other cases it is more or less paraphasic. In motor transcortical aphasia spontaneous writing is impossible, while, in contrast to this, at dictation it is sometimes correct, at other times it is accompanied by many paraphasic distortions. These patients read silently with full understanding, but on reading aloud we perceive the influence of paraphasic admixtures.

In the first case the reading may be compared to the reading of a language which is not understood but is composed of the same sounds, and the same comparison can be made with the difficulty on writing at dictation, for, while not faultless, it is relatively good. On the other hand, spontaneous writing shows to an increased extent the disturbance (paraphasic) of active speech, therefore paraphasic distortions may render the speech utterly incomprehensible. It will have been observed that in proceeding from the consideration of simple and associated facts to more complicated pictures, the significance of which may still be considered as disputed, we have left far behind us what Trousseau and Kussmaul in their time had considered to be strange and entirely incomprehensible observations. A more minute study of the impairment of the power to write may even throw light upon other related points, which as yet have been merely touched upon, and enable us to complete our clinical sketches. Before proceeding it is advisable as an aid to the diagnosis briefly to recapitulate the essential factors in the various forms of aphasia which have been described. Here I follow Lichtheim who, in connecting a supposed conception center with the two speech centers, the motor and sensory, which I separated, was the first to rear a framework for the diagrammatic presentation of all aphasic symptoms. In the subjoined diagram (Fig. 138) *B* indicates the conception center, i. e., the localization of important memory pictures of a concrete object which, in fact, occupy very different cortical regions; but, as has been shown by their firm functional connection with a psychological unit, they produce harmony in the concrete conception. The sensory speech center is indicated by *a*, the motor speech center by *b*.

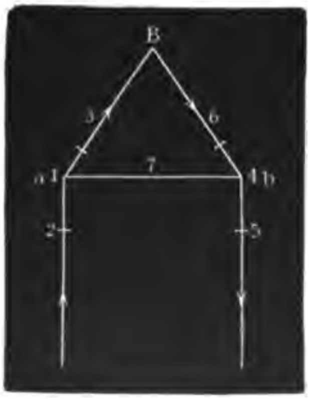

FIG. 138.

The tract from *a* to *b* represents the association tract which serves for the repetition of speech sounds. The tracts *a B* and *b B* are those association tracts which serve to connect the concrete conception with both speech centers.

In the center, *a, b,* is a centripetal and centrifugal tract; in other words, here the sensory and motor tracts enter parts of the projection system. The sensory speech tract contains at least a certain number of fibers of the auditory nerve of the same tone heights which were previously mentioned, and the motor speech tract contains the fibers running to the internal capsules (intended for the nuclei of the bulbar nerve) which function in speech. The diagram enables us to understand why the transcortical forms of aphasia are very rarely observed in their pure form; for *a B* and *b B* are, in fact, radiating bundles only the terminal parts of which, *a, b,* are identical. The separation of these two tracts by a focus can therefore only occur in the most favorable cases in the immediate vicinity of both centers, *a, b.* If the numerals in the diagram are chosen for illustration, 1–3 are the sensory forms, 4–6 the motor forms of aphasia. According to the position of the cortical centers, *a* and *b,* we find upon each side a cortical, subcortical and transcortical form. If, for reasons previously mentioned, we disregard the form of conduction aphasia designated as 7, we must with Lichtheim admit that the mere occurrence of the various forms of aphasia indicated by the diagram is a certain proof that these conditions actually occur in nature. But I can accept this proof only as positive in the case of the cortical and subcortical forms, 1 and 2 and 4 and 5; in the case of the transcortical forms, 3 and 6, there is only some degree of probability. In fact the mere coincidence of such peculiar symptoms as mark the transcortical forms is difficult to explain.

Objection has been made to the name chosen by me, especially for the transcortical forms, and, I must admit, with some degree of justice, for I find that its anatomical foundation is insufficient. But, in support of this nomenclature, I may state that I am aware of no other name which expresses the peculiarity of these cases. For the subcortical forms we possess very apt designations in the names pure word deafness and pure word mutism. But I see no particular reason for dropping an anatomical designation when, in my opinion, it has been deemed fitting. As to the designation transcortical, there are theoretic reasons for its retention to which I shall refer at the conclusion of this article.

The question of localization, that is, of the pathologico-anatomical findings, will later be considered in detail, but here I wish to call attention to certain decisive points of view. One of these has already been emphasized, for I have repeatedly referred to the general experience that pure cases are exceedingly rare. We cannot, therefore, expect that every case of aphasia will show that grouping of symptoms which is typical of the pure form which has been described. The second point, the possibility of grouping the corresponding anatomical findings of different clinical pictures, is no less important. Two such combinations must be mentioned, both of which appear as total aphasia, the one with inability to understand language, the other with loss of the power of speech, therefore as sensory plus motor aphasia; but they are unlike in their different response to the word conception, or so-called internal speech. The more common condition is associated with loss of internal speech, therefore is the sum of the above described cortical forms. It is always associated with hemiplegia. The other form in which the power of internal speech is more or less perfectly retained is rare, but, nevertheless, has been observed; it is the combination of both transcortical forms of

aphasia, and occurs without hemiplegia. Furthermore, a glance at the diagram shows that combinations of subcortical and transcortical forms of both motor and sensory aphasia are favored by anatomical conditions, and are more readily produced by foci in Broca's convolution or the first temporal convolution than by individual forms. Other combinations which must be regarded as natural groupings, and therefore somewhat more common, are met with under the explicit discussion of written language.

The clinical facts enumerated, and the consequent guiding points of view, enable us more minutely to criticise our case of pure agraphia. In demonstrating the existence of a motor writing center, analogous to Broca's motor speech center, there must be a defect just as circumscribed as in Broca's aphasia, a loss of this faculty: "le souvenir du procédé qu'il faut suivre pour écrire les mots." But internal speech, word conception, the "notion du mot" must be intact. For we do not write words but definite groups of letters, and this act, particularly the definite sequence of letters which form a word, presupposes an intact word conception.

But in our case even the first requirement was not fulfilled. The patient could neither tell us how many syllables a word contained, nor could she select letters from tables of letters handed to her and form a word, not even when only three letters, *a*, *n*, *d*, were given her to form the word "and." And this is the almost invariable condition. Experience shows that agraphia, as understood at present, i. e., when not limited to one hand, is always accompanied by and is due to a disturbance of internal speech. More explicitly stated, it is a disturbance of the influence which the "word conception" has upon the choice of letters to form a word, and is a purely transcortical function.

II. DISTURBANCES OF WRITTEN LANGUAGE AND GENERAL PATHOLOGY OF SPEECH DISTURBANCES

That the *disturbances of written language* to which we now turn are of individual importance, and that they take place not only in connection with but independently of aphasic disturbances, proves their occurrence although they are rare, as is apparently isolated agraphia, with which we began our consideration. The contrast to this, isolated alexia, has also been observed without any other disturbance of speech, and so much more frequently that it obviously tempts us to assume a special center independent of the other functions of speech, or a cortical center for written language quite analogous in a motor and sensory respect to the true speech centers, of which the first is to be regarded as the seat of conceptions of writing movements, the second as the optical field for the memory of words. Charcot has been recognized as the most prominent exponent of this conception, which simply transfers the diagram of sound language to written language. However, the untenability of this gross diagrammatic consideration has gradually become obvious, and it has now but few adherents, existing and being widely distributed only in the consideration of a special and unilateral visual speech center in the angular convolution of the lower parietal lobe (Déjérine); even this v. Monakow denies.

Correctly to appreciate the importance of written language as compared

with spoken language, we must remember first of all that the former is a comparatively late acquirement, and that it is not the common property of all persons as is spoken language. Because the possession of this faculty by different people is not invariable, it cannot be uniformly included in the mechanism of the brain, as is spoken language. Hieroglyphics, for example, presuppose a mechanical process in the brain different from that in the usual writing of individual letters among people more highly cultivated. The importance of the alphabet in written language is shown by the fact that reading is generally achieved by spelling. No matter how self-evident a sentence appears to us now, Grashey's investigations have shown that we learned to recognize it only by the previously described process. Grashey's successors, particularly Goldscheider, have found that this is not invariably the case, for combinations of even four letters are understood "at a glance," familiar words as a whole, and independently of the letters which compose them, so that we must admit a faculty for optical word memory pictures commensurate with the individual vocabulary, either printed or written; usually the written picture of the name will be considered. In the course of our description we find another limitation. As a rule which applies to the majority of readers, we must regard spelling, reading and, consequently, optical memory pictures, as being only for letters and not for words, and comparatively few exceptions to this can be admitted. When, in writing, we bring to remembrance the written picture of a word and for this purpose place letter after letter, it is evident from the process itself that we proceed by spelling. What we here produce is a manifold combination of letters similar to that we analyze by spelling when we read. Hence it follows that, aside from the previously mentioned exceptions, a direct relation of the conception of an object to written language must be denied. This point is so important that we shall subsequently discuss it more minutely.

By what process internal thought is communicated by means of speech and written language is almost unknown to us. In many persons it is probably and chiefly by means of sound pictures. That it may be by written pictures and not sound pictures appears absurd after our previous reasoning; for we possess optical memory pictures merely of letters, not of words; certainly all the phenomena of reading and writing are explained by this simple view; should we proceed beyond the most obvious and most simple assumptions, no one can tell to what extremes the pathology of the brain might lead us. The twenty-six letters of the alphabet are certainly not material for thought. Nevertheless, such an assumption is accepted in the differentiation by many psychologists and some pathologists, particularly Bastian, of three different formulas of thought, according to which human beings are classified as "moteurs, auditifs and visuels." The promulgator of this opinion was Charcot. He reported a case in which, during the act of speaking, there was a certain reading of the internal picture of the written word; far be it from me to attack his observation, although I have never seen anything similar. But I regard such an occurrence as the rarest exception, and such a generalization as not permissible. The possibility of its occurrence in deaf mutes does not disprove my theory, at least only in so far that by special training the brain may be enabled to perform such feats. But with most persons in possession of their faculties, there can be no question of such a training.

There is no doubt that intelligent deaf mutes may learn written language independently of speech. The combination of letters which form a word is for them an entirety, the separation of the word into letters is a later and artificial acquirement, analogous to the phonetic spelling of a word. If we admit that some persons usually think in word (of course not in letter) sounds, therefore, in deaf mutes this will appear in a corresponding optical combination of letters each of which in like manner is associated with a corresponding concrete conception, or is united by transcortical tracts with the corresponding pictures of word memory, as was previously explained (Fig. 137). In deaf mutes, therefore, we may distinguish between optical word images and images of letters. This particular class of unfortunates must here be omitted from consideration, since they require special and precise investigation. In all other persons the brain would be over-burdened if it retained all combinations of letters as special optical memory pictures of words, the corresponding word sound pictures being already present, and we must therefore assume that this is not the case. It is positively disproven by the fact that in rare cases (Rieger and Sommer) a permanent loss of power to use certain letters of the alphabet in writing has been observed, the patients having no conception of the form of certain letters, yet there was no corresponding speech disturbance affecting the sounds represented by these same letters. In Rieger's [1] celebrated case this was true of three letters of German text, also of seven small and fourteen capital Roman letters, the identical letters in both the German and Roman alphabets. These cases are further remarkable from the fact that aphasic symptoms were merely indicated; they are the purest cases of disturbance of the function of written language alone which have yet been reported.

It is quite remarkable that Sommer,[2] who published one such case, having investigated it by Rieger's method, comes to the conclusion of an immediate connection between object conceptions and writing movement conceptions. In Grashey's case he believes this explanation to be absolutely necessary. Here he goes a step further than the adherents of a substantive optical word center, some of whom (Déjérine, for instance) deny a motor writing center. His hypercritical tendency and his apparently unprejudicial standpoint here become apparent; for in reality there could scarcely be more positive proof of the connection of an optical word picture with its components, the pictures of letters, than the way in which Grashey's case found his words, and the explanations which we owe to the subsequent investigation of this case by Sommer [3] and Wolff.[4]

Grashey's patient showed the peculiarity that he could only find words for objects shown him, and, as was observed later, only for objects and their properties brought within his psychical conception, by writing. Without this artifice it was impossible for him to express himself in words, but after he had finished writing the word he was able to speak it; if he was disturbed while doing this, each part of it remained discon-

[1] "Beschreibung der Intelligenzstörungen infolge einer Hirnverletzung." S. A. aus *Verhandl. der Phys. med. Gesellschaft zu Würzburg*. N. F. 22. u. 23. Bd.

[2] Sommer, "Zur Theorie der cerebralen Schreib- und Lesestörungen." *Zeitschr. f. Psych. u. Phys. d. Sinnesorgane*. V, p. 305.

[3] *Centralbl. f. Nervenheilk. u. Psych.* Märzheft, 1894.

[4] Gustav Wolff, "Ueber krankhafte Dissociation der Vorstellungen." *Habilitationschrift*, Leipzig, 1897.

nected from the object and useless. But only the complete word bore any resemblance to the word conception of spoken language which corresponds with the concrete object; parts of it, letters, a combination of letters, or even an unfinished word, have no connection with the object that is seen, or felt, etc.

Grashey's case is an example of the possibility mentioned above of special training of the brain. Here the choice of the word independent of the object is impossible, a defect which we will consider later. To compensate for this defect he resorts to the artifice of utilizing written language, as, for instance, a deaf mute would, but, unlike the deaf mute, he must proceed by spelling. His writing is only a result of the intensely stimulated optical memory pictures of letters in which the motor components of "conception of direction" are the main thing, as we shall see later.

In one respect I agree with Sommer: The defect in Grashey's case cannot be explained by assuming a loss or diminution of memory for recent impressions. Its vast importance for the conception of written language and its relation to spoken language have not been shattered by subsequent investigations, but have rather been strengthened.

After these preliminary remarks I consider myself justified in assuming the same cause for disturbances of written speech as those contained in my report of 1886, to which I refer the reader for details.[1]

Ziehen's "concrete conception," as well as all abstract conceptions and, as we shall see, the entire material of thought, has no immediate connection with written language, being essentially a language of letters communicated only through the centers for speech. Therefore, reading and writing are transcortical subordinated activities from the centers of spoken language, and nothing more positively proves the necessity of separating focal symptoms with a transcortical seat than the fact that there are isolated disturbances of written language due to a focal disease of a definite area.

Hence the faculty of writing depends upon spoken language, and is lost as soon as the word conception or internal speech is damaged; it is retained, and furnishes a valuable criterion, when word conception and internal speech remain uninjured.

Integrity of the power of articulation is an important sign in subcortical motor aphasia or pure word mutism, as well as in subcortical sensory aphasia or pure word deafness. In the transcortical disturbances of speech, the intactness of the word conception is also evident from the fact that purely mechanical reading and writing reveal no disturbance. The patient may read aloud either at dictation or from copy. Some individual points in the symptom-complex of written language are not yet sufficiently proven. As a rule, we may maintain that a defective power to write is analogous to defective speech, for with the loss of the power of speech the comprehension of what is read is also lost, with the loss of the spontaneous power of speech, the faculty of spontaneous writing also disappears; in reading aloud and in writing at dictation paraphasic admixtures and distortions are as numerous as on repetition. This is particularly true of transcortical sensory aphasia, provided that the paraphasic symptoms are involuntarily increased by the act of writing so that writing becomes even more difficult. In the discussion of such cases, the terms paralexia and paragraphia are objectionable, and the expression paraphasic disturbances in reading and writing is preferable. In transcortical motor aphasia, it is evident that spoken language is characterized by an absence of spontaneity, and this becomes more marked on trying to write. It also appears in writing at dictation, for this form of writing, although

[1] *Fortschr. d. Med.*, IV, pp. 371 u. 463.

possibly to a less extent, presupposes the spontaneous coaction of the writer. "Word finding" for the act of spontaneous speech, the actual function of the tract *B b*, necessitates for writing the further exercise of finding the letters, as the sounds of the letters, in our sense, belong to the word conception.

The power of writing, as previously stated, is most severely damaged in the cortical forms of aphasia, for here the word conception itself sustains a lesion. Cortical motor aphasia, as a direct focal disease, usually results in just as persistent an alexia and agraphia. But the letters continue to be optical structures, and may be written individually, as is proven by the retention of the ability to copy. For reasons to be subsequently more minutely considered, in an intact brain which still responds, there is a greater probability of the restoration of the receptive faculty of reading than of the expressive faculty of writing. The sequence in which reading returns after cortical motor aphasia has been studied by Thomas and Roux under Déjérine's direction; the inverse relation of learning to read was revealed by the circumstance that at first entire words, then syllables, finally the individual letters which compose a word, were read with confidence. Besides mutism, isolated or very marked agraphia may persist for years; an agraphia, however, which is characterized by the retention of the power to copy as well as to change letters from one form into another form, and this power prevents confusion with the substantive agraphia of our case detailed at the beginning of this article. Generally the ability to write improves just as does articulate speech; it is slower at dictation than on spontaneous writing (Déjérine). In writing, the integrity of the word conception is absolutely necessary; on this point I agree with Déjérine and differ with v. Monakow. My experience does not permit me to coincide with the observation of v. Monakow that motor aphasics often find written expression easier than speech. This probably refers to cases of predominant subcortical motor aphasia, for I believe that v. Monakow's clinical picture of cortical motor aphasia has been based on too broad a conception; he recognizes a partial motor aphasia of much wider extent than I do. At all events there are exceptional cases—a case of Banti's is a proof of this—in which cortical motor aphasia leaves intact the faculty of written expression as well as the understanding of written expression. Monakow assumes that this can be due only to a partial disease of Broca's convolution, a condition to which we shall refer when describing the pathology.

Sensory aphasia of cortical origin is generally admitted to occur without severely damaging the power to write. On the restoration of the power of speech the art of reading is sooner learned than that of writing; in a word, according to universal experience, agraphia usually persists as a severe permanent disturbance. This corresponds with the fact that we attach the highest value to the acoustic components of the word conception during the time of its attainment. Perhaps, however, purely anatomical conditions are not unimportant, since the posterior and upper end of the first temporal convolution almost forms an important association tract for the motor act of writing.

Moreover, there is a lack of casuistic material consisting of well observed cases of inability to write to show that in disturbances of speech the symptoms disappear during the stage of improvement.

The relation of written to spoken language enables us to appreciate in

a double sense the clinical symptoms of alexia, agraphia, paralexia and paragraphia whether they are the sequels of a damaged word conception or whether they appear independently. The former may be designated verbal, the second literal. The substantive disturbances of written language, which are observed with intact word conception or internal speech, relate also to the form of letters so that, for example, literal agraphia consists mainly in the fact that the reproduction of this form is impossible for the patient, and the case whose history was given in the beginning of the article is a marked example. In literal alexia the actual defect consists in a non-recognition of letters, and in consequence of this of words also. A special form of alexia and agraphia is produced, as we shall soon see, by a damage of the tracts which connect the word conception with the psychical elements represented by letters.

It would lead us too far from our theme and would not be in consonance with its importance here to trace diagrammatically all the true disturbances of written language, as was done for those of speech. But in regard to the form which such a diagram must take, if it is to correspond with the majority of the facts, the most important points may be indicated. It generally corresponds to that developed for speech (Fig. 138) if, in place of the conception center, *B*, we use the word conception, *c*, in place of the motor speech center, we use the conception of writing movements, β, and in place of the sound pictures we substitute the optical memory pictures of letters, a. While, however, in the diagram of speech processes we must adhere to the fact that a direct tract, *B*, *b*, exists, it is very unlikely that a corresponding tract, $c\ \beta$, comes into question in written language. The diagram thus becomes more simple than for speech. It appears that in writing a path over the optical field of memory, a, is absolutely necessary, probably because of the method most often used in learning to write. I do not doubt that a change in this method might influence the clinical importance of this tract. Thus, the older among us, without exception, first learned to read letters and then to write them. Now these arts appear to go hand in hand, so that children are first taught not only to read and then to write, but are taught to read while learning to write. Under this method the motor components in the production of a letter gain an increase in dignity.

On the other hand the difference between printed and written letters teaches us that the motor components of the conception of writing movements, at least in recognizing a letter, are indispensable, so that the conception of writing movements of the letters cannot be regarded as an essential factor of this.

In employing the expression, "conceptions of writing movements," I desire not to be misunderstood. The term signifies only that conceptions of movements, in Meynert's sense, produce writing movements just as, in quite a different muscular area, they produce speech movements. A certain recollection of the process to be followed in writing is therefore admissible and cannot be denied; neither can we deny a certain localization of this special function of memory in the left arm region, provided we write with the right hand. But it will be the same locality in which originate all the finer movements of the right hand which are not specially localized, but serve this purpose, as in conceptions of speech movement. "L'écriture n'est qu'une des formes de motilité de la main" (Déjérine). When writing is done with the

left hand, the same, although less common conception of movements, is localized in the right arm region, and we localize these in the right leg region when we write in the sand with the left foot. These are the conceptions of movement described by Storch, which in writing, as in every motor act, dictate the movements of a certain portion of the body, because we have in the optical fields for memory of letters a special depôt and a complete diagram of the conception of those movements of direction involved in writing. There are persons who can produce writing movements with their tongues. Exner's theory of a special motor center for writing at the base of the second frontal convolution therefore seems scarcely plausible, to say nothing of the fact that it was based on an uncritical utilization of doubtful casuistic material, and has not since been supported by pathologico-anatomical findings. An absence of these conceptions of movement, analogous to Broca's aphasia, must therefore be assumed in all cases of paralysis of the right hand from a left-sided cortical focus. The general incapacity of the right hand includes actual inability to write, while the retained motility of the left hand permits writing, and proves the continuance of its necessary conceptions of direction. Such a unilateral agraphia can under no circumstances be synonymous with Broca's aphasia. This was probably present in Pitres' case in which there was a cortical lesion, and, in spite of recovery of the finer motility of the right hand, an exclusively right-sided agraphia was permanent. I shall later discuss the explanation of this extraordinary and instructive case.

There is no unanimity of opinion among authorities as to the existence of a so-called optical word center, actually an optical letter center. This mooted question from a purely practical and clinical standpoint, might well be left out of consideration, and we might be content with recognizing the insignificance of a circumscribed unilateral lesion in the posterior lower area of the lower parietal lobes in the sense of localization, an area which is important as the seat of origin of what Naunyn, upon the basis of statistics, called indefinite aphasia. Here the decisive point which belongs among the most positive facts of cerebral localization, is that a unilateral focus deeply situated in the medullary structure of the parietal lobe produces a combination of right-sided hemianopsia and isolated writing blindness or alexia as persistent symptoms. If the affection is disseminated and reaches the cortex of this portion of the brain, agraphia is added to alexia (Déjérine).

If these facts are grouped and explained like those of spoken language in our diagram, the cortex of this portion of the brain would be considered the point of optical "alphabet memory pictures," and pure writing blindness as subcortical alexia analogous to subcortical sensory aphasia. It is self-evident that experience alone can determine whether or not we will accept this conclusion, and theoretic considerations must be held in abeyance. It may even be admitted that there are theoretic grounds for the assumption of such a center. The principle of conservation of energy appears to favor the unilateral condition, the especial peculiarity, which characterizes the memory pictures of letters of the alphabet above all other optical memory pictures as a corresponding special localization; I shall revert to this later. Finally, it must be observed that this is the standpoint of such competent authorities as Bastian, Déjérine and A. Pick. Nevertheless, I cannot agree with them without sacrificing the theory of the internal connection and the fundamental

principles for the understanding of the structure of the brain which were taught with such conspicuous success by Meynert. This is also v. Monakow's view; he doubts the existence of a true optical word center, and maintains that optical memory pictures of letters of the alphabet are double. I had previously expressed this opinion and had declared myself opposed to the localization of the so-called optical pictures of word memory. Since a positive theory of localization, if erroneously taught, may imperil the advance of the law of localization, as we have seen in the case of Broca's aphemia, I feel forced to explain somewhat more minutely the untenability of the assumption of an optical word center in the cortex of the gyrus angularis.

Theoretically all that we know of the nature of optical memory pictures is opposed to such a narrowly limited localization of alphabet memory pictures, particularly to a unilateral one. More recent investigations have clearly shown that the memory pictures of the special senses have motor components which from their nature are inseparable. Storch has designated optical memory pictures by the appropriate term, direction conceptions. Such conceptions of direction also apply to all motor projection fields, but nowhere so clearly as in the optico-oculomotor projection field which, in contrast to the true field of light (H. Sachs), includes the convex surface of the occipital lobe and the gyrus angularis of the lower parietal lobe. For example, such conceptions of direction, in so far as they relate to the arm and hand, are localized in the so-called middle third of the central convolution. As shown above, they coincide with conceptions of writing movements. Now the optical memory pictures of letters, like those of figures or any other objects which have but two dimensions, differ from concrete things by the fact that to the sense of sight they each have but one form (Storch [1]) while all other concrete objects possess innumerable ones. Their limited number, and the exceedingly frequent use which we make of them, will cause them (according to the principle of well grounded tracts) to appear as a particularly firm, and consequently easily utilized, possession in pictures of memory. Finally, optical memory pictures of letters are characterized, as I previously attempted to explain, by the fact that they bear no direct relation to concrete conceptions. Except a single tract which is the most marked, hence the most easily understood tract, they have no communication with a unilateral speech region nor, we may presume, primarily with the acoustic sound center. These three properties which they possess in a greater degree than any other optical memory pictures, give us no reason to assume a specially circumscribed localization, except that the point of most distinct sight and of the finest differential oculomotor conceptions of direction has a special localization in the cortex which has not yet been proven. Only one of these properties, namely, exclusive association with the left temporal lobe, is a factor important for the unilateral nature of the alphabet memory pictures. But to this I shall refer later. If we consider the functional acquirement of memory pictures of letters of the alphabet, it does not favor the predominance of the left hemisphere. In typical hemianopsia the form of the field of vision permits us to conclude that the immediate surroundings of the point of fixation are almost always bilat-

[1] Details in an article by Storch: "Versuch einer psycho-physiologischen Darstellung des Bewusstseins." Berlin, 1902.

eral, and supplied by each optic tract. Usually the picture of a letter is formed exclusively within this central area of sight so that its memory picture is formed in each hemisphere. If the letter is large, the glance will wander as with every large object, but central sight will always be exclusively implicated.

We might believe that the possible unilateral nature of optical memory pictures of letters might be directly determined by ascertaining whether there is a difference in the peripheral portion of the field of vision, and whether the patient reads with the right or the left half of the visual field. This theory, however, is shattered by the fact that even the largest letters can be read only in the immediate vicinity of the point of fixation, while beyond this it is impossible to say with certainty whether or not there are any letters. It is usually maintained that central acuity of vision rapidly declines outwardly from the point of fixation. How rapidly this becomes apparent on an attempt to recognize letters, and there is no difference between the right and left halves of the field of vision. But certain facts in the pathology of the brain are much more important than these academic considerations. Above all, we can form no conception of the combination of isolated word blindness with right-sided hemianopsia in the sense that hemianopsia, i. e., the functional interruption of the subcortical tracts which substitute for the left cortical tract, is to be regarded as the cause of word blindness, as the acoustic tract which terminates in the left temporal lobe is the cause of subcortical sensory aphasia. On the contrary, the majority of cases of typical right-sided hemiplegia show no trace of word blindness or alexia; at most, reading is made difficult by the hemiopic defect itself, from the fact that the patient cannot see the letters or words immediately following, but every letter or syllable is recognized unless there is an impairment of memory as in a case of Redlich's. It is noteworthy that with the sudden appearance of hemianopsia this defect is soon recognized, therefore reading need not be even temporarily prevented by the hemiopic defect. In all of these cases we must assume that the patients depend exclusively upon the right hemisphere in reading. In occlusion of the tractus opticus the left hemisphere must to a certain extent be regarded as blind. If, in spite of this, the patient recognizes letters and their sequence it must be because of the termination of the optic tract in the cortex of the right hemisphere. This conclusion is so obvious, and so convincingly disproves the theory of the unilateral nature of the so-called optical word center in the left hemisphere, that its exponents, particularly Déjérine and Bastian, were forced to consider it. They attempted to nullify its importance by assuming that fibers of the corpus callosum, which pass from the optical projection field of the cortex of the right hemisphere to the symmetrical cortical fields of the left hemisphere, were still able to reach the optical word center upon the left side. For this purpose they differentiated between a general visual center and a special optical word center: The former is bilateral, the latter unilateral, and present only upon the left side. In the same way they differentiated a general center of hearing in both temporal lobes, and only a left unilateral and special center for the understanding of word sounds. I believe this entire reasoning to be erroneous, as it controverts the views of the pioneer Meynert, and ascribes functions to the fibers of the corpus callosum, which are merely a continuation of the fibers

of the tract of the optic nerve, or of the auditory nerve, beyond the cortical projection field into the opposite hemisphere. Were these the true conditions, the hemiopic defect in a unilateral lesion of the optic tract would eventually be compensated for by the other hemisphere, and no longer be noticeable. Finally, I must refer to the clinical condition known as soul blindness, which frequently accompanies word blindness or literal alexia.

If pathology thus disproves the unilateral and narrowly circumscribed localization of an optical word center, all the more necessary is it specially to review the circumstances which produce this unilateral and circumscribed condition. Apparently it is the relation brought about by association fibers between the unilateral speech region and the optical memory pictures of letters, and chiefly of their acoustic components which are calculated to simulate this unilateral condition. Here two possibilities become obvious, but further investigation by necropsies will alone clear the situation. One view, accepted by Monakow, is that foci in the gyrus angularis, besides interrupting the optic radiation of Gratiolet, have also implicated the crossed optic-acoustic commissure (Bastian), i. e., the association fibers between the left temporal lobe and the right optical oculomotor projection field. The optical memory pictures of the left hemisphere are then obliterated by the interruption of the optic radiation of Gratiolet (hemianopsia). The optic memory pictures in the right hemisphere might yet be stimulated by means of the right optic tract, but could not be utilized in reading because they could no longer evoke by means of the previously mentioned commissures the absolutely necessary acoustic or word sound constituents of the letters. In other words, the power to form words from the individual letters would be lacking, and reading by spelling the words would be quite impossible. As an exception to the rule of reading by spelling, reading would be limited to the few words which the patient had previously learned to recognize. The majority of patients experience great difficulty in naming objects shown them, and this is usually observed in cases of word blindness; it is ascribed to a lesion of the communicating tract which leads to the left temporal lobe, but incorrectly so, as we shall soon see. Moreover, this does not explain the fact that letters, especially, are seen but not identified, all other objects being clearly recognized. We have seen that other relations than those of letters to the acoustic projection fields do not exist. It is a question, too, how in word blindness thus produced the form of the letters themselves can be recognized, copying being done correctly, and also how it is that the same letters in different arrangement appear identical, being transferred from one arrangement to the other, the purely optical components remaining undamaged. For some of the cases so far observed this expectation has not been realized; on the contrary, a most conspicuous feature of these cases is that the patients do not actually write the letter of the alphabet while anxiously following the copy provided, but are only able to draw it. If we have ever noted how these patients laboriously draw each letter, and how they find this equally difficult whether the copy be written or printed, we will scarcely search for the cause of this defect in the interruption of the decussated optico-auditory commissure.

Of course we must bear in mind that the optical memory pictures of letters represent complexities of form conceptions, and these attain definite importance only from the fact that they are associated with the word conceptions

of the letters belonging thereto. Articulate speech is composed of word conceptions, i. e., it represents the combination of a sound picture and a definite conception of speech movement, and these bear to the optical memory pictures the relation above described, and when this relation ceases the latter at once deteriorate into meaningless signs. This may explain why the identity of a printed and a written letter, or of a Latin and a German word, is no longer recognized if the sound which produces the relation can no longer be internally produced. It is even questionable whether the pure optical components of letters, detached from their sounds, will permit us to recognize that we are dealing with the letters of the alphabet.

If we transfer this first possibility to the diagram it would correspond to a combination of subcortical and transcortical symptoms. The unilateral affection of the tract would be subcortical, and the interrupted continuity of the crossed optico-acoustic commissure, transcortical. Alexia would arise because only one of the tracts, *c* and *a*, the crossed and the uncrossed, would be interrupted, but the other, in consequence of the interruption of its subcortical tract, would be useless. On the other hand the unimpaired, uncrossed tract *c a* would be amply sufficient for the power to write as before.

The second possibility is that which simultaneously assigns to the left optico-oculomotor projection field a predominant and indispensable rôle by permitting an ability to read; this is quite in contrast to the first theory, according to which the left-sided projection field appears to be especially excluded. According to the principle of the conservation of energy it permits the view that only the optical memory pictures of letters in the left side are united to the acoustic projection field by a well beaten tract; that, therefore, there exists only one uncrossed acoustico-optico commissure or tract *c a*. This would lead to the further assumption that the connecting link, *c* and *a*, necessary for reading, must always follow a circuitous route over the left optico-oculomotor projection field even when, in consequence of right-sided hemianopsia, reading is accomplished solely by means of the right hemisphere. The interruption of communication which alexia causes must be sought in the commissure between the two optico-oculomotor projection fields, particularly between the oculomotor projection fields, a view which in the main coincides with that of Bastian and Déjérine. The only difference would be that the memory pictures, not the point of perception, are united by fibers of the corpus callosum. I have elsewhere expressed myself concerning this possibility.[1] Transferred to the diagram this interruption would not affect the tract, *c a*; therefore in regard to *a*, it would be of double subcortical importance; first, on account of the tract fibers of the same side, and second, on account of its detachment from the optical memory pictures of the right side which are still susceptible to light. From this point of view the power to write which emanates chiefly from the left center, *a*, would, because of the integrity of the tract, *c a β*, show no disturbance.

The question of the unilateral origin of an optical word or letter center is evidently of great importance for the entire law of localization. If we deny it, as in my opinion we must, there is still some hope of a pathology of the fibers of the corpus callosum; for it would be comparatively easy to

[1] *L. c.*

decide which part of the corpus callosum must be implicated in order to produce the symptoms of subcortical alexia or pure word blindness.

I must admit also that our case of apparently isolated and literal agraphia described at the beginning of this article cannot be better explained than by adopting the view of the unilateral location of the center, *a*. The tract, *a β*, would then be interrupted at a point transcortically beyond *a*; the assumption of two tracts, namely, one upon the same side and one upon the crossed arm region, makes this the more plausible because they have the same point of origin in *a*. Nevertheless, as we shall soon see, the apparently natural and obvious explanation is in absolute opposition to other experiences. In contrast to the view of a special position for the letter center among the optical pictures of memory, this case exhibits a singular feature since the patient, besides having lost the faculty of writing, had also lost the power of drawing the simplest figure.

The clinical picture of pure word blindness, more correctly letter blindness, or, in accordance with my suggestion, subcortical alexia, is based upon quite a number of cases; Redlich[1] has collected from literature no less than twenty-seven pure cases and, as has already been stated, there is no lack of anatomical foundation for this view. The chief symptom of the clinical picture may be delineated by a case which is quite typical.

L., a plasterer, aged 66, understood questions I addressed to him, and answered them all correctly. He was intelligent and showed no defect in speech. When asked to write something at dictation he did it correctly and uninterruptedly, and also wrote down any figure required of him. At my request he wrote a letter to his married daughter; although there were errors in spelling and the alignment of the writing was poor, and although there were breaks and repetitions as from uncertainty of motive, the letter was upon the whole correct and comprehensive; had he been a man of education it would probably have been perfectly correct. The man was then asked to read what he had written, and the surprising fact was revealed that he could not read a word, not even a letter or a figure.

The same condition is found in all analogous cases, provided they are as well marked as that of my patient. The cases differ only in that figures are often clearly recognized. We learn from this case that the difficulty is not due to visual disturbance, for the patient said his sight was good, which was also proven by the fact that he could copy letters and words which he did not know, of course in a mechanical way, copying character for character like entirely unfamiliar figures. Hence he did not understand what he had written, and it has been repeatedly observed in analogous cases that by this circuitous method the patients are able to decipher what has been written, although slowly and with great difficulty. The investigation of the acuteness of vision proves that this is quite sufficient for the recognition of letters. Nevertheless, this patient's sight was defective; this was evident from the peculiar position of his head while writing, from the running together of the words, and the defective alignment. That is, he had a typical right-sided hemianopsia, with vertical separation of the halves of the field of vision into the familiar form in which a narrow portion, passing over to the right, still

[1] Redlich, "Ueber die sog. subcorticale Alexie. *Jahrb. f. Psych.*, Bd. XIII, 2 und 3 Heft.

retains the faculty of sight. Bilaterally the acuity of vision was at least one-half of the normal; the eye-ground was absolutely normal. This defect manifested itself by making fluent reading impossible, because there was no view of the following letters, and in our case the only letter seen in totality was not recognized. By showing objects we convinced ourselves that the patient's sight was sufficient; he was able to recognize any object. But a phenomenon is frequently observed in analogous cases, and is sometimes more highly developed than in this case: Difficulty in properly naming an object. In our patient this occurred only now and then. Expressions of embarrassment or descriptive terms were first uttered; finally, on being prompted, the correct word was eagerly accepted. In other cases not a single object shown can be correctly designated, and this was the case with a patient that I saw about 25 years ago in the Charité. It was the only symptom which indicated aphasia. In spontaneous speech, words are not lacking, not even those for visible objects. When, on the other hand, this symptom is more conspicuous, as, for example, in my earlier case, there is more or less difficulty in finding words for concrete objects, even in spontaneous speech, but, as a rule, this is not so difficult as naming objects which are shown.

During the first period of observation another phenomenon appeared which is apparently of much more serious import than the mere difficulty of finding words. The patient for a time was absolutely unable to recognize objects that he saw, although he perceived them distinctly; he therefore presented the well known symptom of mind blindness. This condition is not the result of accident, but is frequently observed in cases of so-called pure word blindness. Deducting conclusions from the course of our case, we recognize an increase of this defect in alexia, and in our case an apparently secondary effect depending upon an indirect focal symptom of the lesion producing alexia. In regard to the simultaneous occurrence of these two phenomena, experience teaches that mind blindness usually leads also to word blindness, but isolated writing or word blindness frequently occurs without mind blindness. In the first case I observed there was no indication of mind blindness. In the case just considered it was remarkable that during the time of soul blindness the patient was unable to recognize objects even by touch. Mind blindness, therefore, existed even to the extent of asymbolia, the most severe sensory defect which can be referred to focal disease. Concerning the history of our case, its course so far and experience in similar cases permit the conclusion that we are dealing with a stationary condition in which a possible improvement may be hoped for. While there is no present sign of hemiplegia there were symptoms of this six months ago, but these completely disappeared within two to three weeks, a fact which indicates that embolic processes were present. The symptom-complex now existing attracted attention because the patient was no longer able to read his newspaper as usual. We are justified in attributing this to embolism and to resulting softening.[1] The probable locality of the pathologic focus will be subsequently pointed out.

Besides the typical pathologic picture which our case presents, no doubt there are other isolated disturbances of the faculty of reading which are of

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[1] E. Storch, "Zwei Fälle von reiner Alexie." *Monatschr. f. Psych. u. Neurol.*, XIII, Ergänzungsheft.

different nature; for example, the dyslexia first described by Berlin,[1] a partial inability to read which manifested itself subjectively, and soon increased to complete inability to read; after a little rest the patient was able to go on reading but it was with recurring signs of exhaustion which rapidly increased. Here the feeling of disinclination was remarkable ("fear of reading," Bruns). We cannot refrain from assuming that this is a functional affection of the same or similar origin as is pure word deafness, and the suspicion of an organic foundation, no matter of what nature, must be borne in mind all the more as Berlin's experience led him to emphasize the unfavorable prognostic significance of this symptom which usually appears suddenly. Hemiopia may be present but it is not a requisite as in alexia. Most of the cases succumbed in a few years from serious affections of the brain. Atheromatous and syphilitic disease of the cerebral arteries were repeatedly found.

As we have seen, isolated word blindness is a combination of various conditions, usually of the interruption of a subcortical tract and the integrity of a transcortical tract. From the well known location of the pathologic focus which corresponds to these requirements, deep in the medulla of the gyrus angularis, we may conclude that the tract of the cortical surface of the gyrus angularis, which has remained intact, must lie nearer than the destroyed one. Therefore, the fact mentioned above (which was utilized by Déjérine in the consideration of a center of optic word memory pictures), that widespread destruction in this region produces literal agraphia as well as literal alexia, entirely destroys the perception of written language, also the fact that if a disease at the cortex be added to a disease of the deep medullary substance, agraphia is added to the previously isolated word blindness. According to our diagram, agraphia and alexia are produced by the interruption of the tract, *c a*, that tract which forms the necessary power for all perception of written language. As I have reiterated, this power most likely exists in nature in two entirely different tracts whose anatomical relations we have yet to discuss. This may explain why their common division causes an exclusive arrest of the function of written language without any implication of speech, a condition so far very rarely observed; we shall see that the two cases of Rieger and Sommer are the only examples which approach this. It would, however, be a somewhat common occurrence if the cortex of the gyrus angularis were actually an optic word center, as Déjérine maintains. It is evident from the diagram, which illustrates Déjérine's views of the extension of the speech region, that this author ascribes an influence to spoken language, to the so-called optical word center, similar to that of the acoustic word center. This opinion we cannot accept; it is opposed to our theory developed above that in persons mentally sound letters, not words, form optical memory pictures.

As already indicated the coincidence of literal alexia and agraphia without essential affection of speech has been observed only in the two cases of Rieger and Sommer. They are of such great importance that I must relate some of their details. Rieger's report, which is a remarkable example of impartial description in a difficult realm, shows that the patient had lost all conception of three of the small letters of the German alphabet, p, x, and y,

[1] *Arch. f. Psych.*, 15. Bd., p. 276, "Wanderversammlung südwestdeutsch. Irrenärzte u. Neurologen," 1883.

and of the small Roman letters these three as well as d, h, k, and v. Of the capital letters he had lost the conception for fourteen of the Roman as well as the German alphabet; namely, all those previously mentioned with the exception of D, as well as B, E, F, M, N, R, T, and W, so that he retained the power to use only eleven letters. This defect included both written and printed letters. When the patient was shown these letters he behaved as though they were absolutely foreign things, and when he was told "This is a B" or "This is an E" he shook his head and said "I do not know." But he at once recognized the letters he knew, being able both to name and to write them. He responded in the same manner to words composed of letters with which he was familiar. He was absolutely unable to write spontaneously the missing letters; he could laboriously draw them from a copy as if they were unfamiliar arabesques. So far as his perception of letters extended, the patient could write from dictation or from copy, but at first without understanding what he wrote. Spontaneous expression by writing was absolutely impossible, and in general he read without any comprehension of the subject. In his spontaneous speech there was nothing conspicuous. Now and then he was disturbed by being at a loss for a word, usually a substantive. Of figures he knew only 0, 1, 2, and 3, all others and all combinations of those he knew being absolutely incomprehensible to him. Besides this more or less localized defect the patient exhibited another which usually belongs to the realm of speech; that is, an inability to name objects shown him. He handled these, and in a general way had some perception of them, as well as of their properties. There was also an impairment of memory and of all the realms of special sense; this was no less marked than in Grashey's celebrated case which was subsequently investigated by Sommer and Wolff. As showing other defects, we must also mention the loss of the art of drawing in this man who was previously a skilful sculptor. All appreciation of curves was gone. In a restricted, practical sense there was no diminution of intelligence. For our purposes it is interesting to report that this patient could always find words for objects shown him, even though after an abnormally long time, and that among the objects presented to him he seemed to prefer letters inasmuch as he could name these in one-half the time consumed in naming other objects. According to Rieger's explicit report, this condition remained unchanged for two and a half years. The patient was a sculptor and teacher of drawing, aged 32, who had sustained a severe fracture of the skull in a railway accident. The defect did not appear at once, at least not to its full extent, but developed six months after the accident and finally reached the degree which has been described. In Sommer's case [1] there was a similar and persistent lack of perception of certain letters, and a partial literal alexia and consequent agraphia, the result of an attack of apoplexy. In this case the recognition of a number of other letters varied and was uncertain. Moreover, the patient was unable to combine the letters he knew into words, and thus to read, so that the condition was practically a complete alexia. Still more complicated was the perception of written language which was somewhat better retained; hence the case reminds us of

[1] Sommer, "Zur Theorie der cerebralen Schreib- u. Lesestörungen." *Ztschr. f. Psych. u. Phys. der Sinnesorgane*, 5. Bd., p. 305.

subcortical alexia. For all practical purposes there was also agraphia. It is interesting to know that immediately after the apoplexy, and for about a year and a half, the patient was unable to read or write. At first he had little command of words. After fourteen days the right-sided hemiplegia improved, and while under observation right-sided hemianopsia was demonstrated. Aside from this disturbance of the power to read and write and his inability to find words with which to express himself, the patient was intelligent and sensible, hence we must assume that this disturbance of speech was not so marked as in Rieger's case. No autopsy was held in either of these cases.

In explanation of the peculiar partiality of this isolated impairment of the perception of written language it has been pointed out that in all disturbances of reading the more infrequently used letters, such as x and y, are more apt to be implicated than those in frequent use. However, in these patients it was not the letters most seldom used which were lacking but the quite common ones, and the persistency of the defect appeared to exclude a purely functional nature. In all our experience in speech disturbances in focal diseases of the brain we know of nothing analogous except one case of mental disease after recovery from which a partial motor sensory aphasia and a defective use of words persisted.[1] In fact, as in mental diseases which show a preference for certain association tracts and functions, we must assume in the first case a similar degenerative process as a consequence of shock to the brain, and in the second case an apoplectic attack. Obviously the affected tract is the tract *c a*.

As an illustration of pure isolated agraphia the case might answer which I mentioned at the beginning of this article (unusual and rare as this case is) because it presents the postulated defect in a comparatively pure form, and careful investigation demonstrates that the patient also suffered from a disturbance of internal speech and word conception. Besides we would necessarily assume that there was formerly a decided disturbance of speech which had greatly improved. The reduction in the power of spontaneous speech which was conspicuous during the time of observation and even two years later, was in contrast to the otherwise active intelligence of the patient, and this justifies the differentiation of the disturbance as one of transcortical motor aphasia, although not of high grade. In this connection I may refer to the published report of my own case.[2] Therefore, it will be seen that the same complication was present in the few comparatively pure cases of agraphia which I compiled from literature, and I reached the same conclusion as Déjérine that there is no pure literal agraphia, or motor agraphia, as Pitres called it. Notwithstanding this it is certain that the peculiar symptoms of the case cannot be explained solely by the disturbance in word conception, but that aside from this, the exquisitely literal or, better, the motor, character of the agraphia forms the predominant feature in the pathologic picture. This is also true of the few analogous cases which I was able to find. My patient, as is proven by the comparatively well retained faculty of reading, undoubtedly received optical memory impressions of letters, but, unlike a

[1] Heilbronner, "Aphasie und Geisteskrankheit." Psychiatrische Abhandlungen, herausgegeben von Wernicke und *Zeitschr. f. Psych. u. Physiol. d. Sinnesorgane.* XXIV, p. 83.

[2] *L. c.*

normal person, these could not be transmitted to the motility at his command. Comparing this with our diagram of written language there appears to be an interruption of the tract $\alpha\ \beta$. From my previous presentation it is evident that this tract is bilateral, and the especial peculiarity of my case, as well as of the few analogous ones, is the bilateral absence of function.

In this case symmetrically situated foci in both hemispheres, but less marked in the right, might possibly be thought of; the periodic appearance of left-sided symptoms might be regarded as favoring this view, and such a possibility must be borne in mind. But the analogous cases presented signs of left-sided focal disease exclusively, and cannot be explained by the assumption of symmetrical foci. We are therefore forced to the conclusion that the disturbance of word conception and also a general damage to the functions of the brain might under some circumstances have such an effect that besides directly damaging the unilateral tract it might render the other also incapable of function. In our case the transcortical disturbance of speech, still evident, is probably insufficient to permit the utilization of a tract on one side for a function which is too difficult and, therefore, impossible to perform. As a rule, when these conditions exist we must consider the unilateral interruption of the supposed tract. We actually find an excellent example of this in the case which Pitres, in spite of the unilateral nature of the disease, designated as "agraphie motrice pure." In this case it appears that some time previously an agraphia similar to that of my case had existed. This, however, improved, persisting only in the right hand, although the motility of the hand was otherwise restored. Right-sided hemiopia with good vision was permanent. There was originally a right-sided hemiplegia with severe general symptoms. In these cases of unilateral disturbance of the power to write, the use of the term agraphia might very properly be questioned. But no doubt this is such, and scarcely any other explanation is permissible than that it is a unilateral interruption of the tract $\alpha\ \beta$ of our diagram. Liepmann's case of unilateral apraxia showed on minute investigation the same phenomenal motor agraphia limited to the right hand, only this formed a part of the total picture of unilateral apraxia. The patient was able to write with the left hand, but he performed mirror writing. This example, it appears to me, throws light upon the function of the right-sided tract, $\alpha\ \beta$.

According to Liepmann mirror writing or, more appropriately, abduction writing, is a striking proof of the purely mechanical way in which memory pictures of letters are transferred to motility. While learning to write most persons practise this process by chiefly using the left-sided tract, $\alpha\ \beta$; which, we may at once explain, is in accordance with the principle of the conservation of energy. But we must also take into account the general experience that in such practice symmetrical movements of the other half of the body are unconsciously practised, movements which we attribute not only to the functions of the motor anterior horn cells of the spinal cord but also to complicated cerebral functions. The so-called conclusion by analogy depends upon this. If, however, the same innervation of the right arm center takes place by means of right-sided memory pictures of letters in the right-sided tract, $\alpha\ \beta$, as in ordinary writing, and the left arm center by means of the left-sided tract. $\alpha\ \beta$, mirror writing or abduction writing must result. Now it is also the function of this tract voluntarily to conduct writing move-

ments of the left hand in such a way that they correspond to the identical direction conception instead of to the symmetrically opposed ones, as in writing with the right hand. But this is primarily drawing, which is done without practice, and even in opposition to the involuntary innervation passing in an opposite direction. It therefore represents a much more difficult cerebral activity than mirror writing, which is produced by a tract which is somewhat well marked. Subjectively this necessitates a special exercise of the will. Thus we comprehend that a transcortical motor aphasia, even though only feebly indicated, renders correct writing by means of this tract impossible; the necessary stream of innervation is too weak at its source. What, however, is not so easily understood is the fact that our patient with agraphia never showed the slightest inclination to mirror writing; our suspicion of a very circumscribed, symmetrical focal affection of the right hemisphere must be thereby increased. Moreover, the conditions which produce mirror writing are still imperfectly understood. Accompanying right-sided hemiplegia, either with or without disturbance of speech, it is apparently sometimes observed under the same conditions, and sometimes not. It is chiefly noted in persons whose minds are somewhat impaired, with or without signs of unilateral atrophy of the brain, and especially in idiotic children (Soltmann). If normal children who have had the same elementary instruction are forced to write with their left hands, some produce mirror writing, others do not. When mirror writing is noted it is usually quite correct, and proves that there is no literal agraphia.

I shall return to the presumable position of the tract $a\ \beta$. We see that a certain point in this tract may be determined with some degree of likelihood, and thus isolated agraphia as a focal symptom becomes as important in local diagnosis as is isolated alexia as a focal symptom of the gyrus angularis.

One of Rieger's cases which was reported by Wolff[1] appears to be an example of pure cortical agraphia. Here total central blindness due to bilateral destruction of the optical projection fields of course also resulted in cortical alexia.

For the rare cases of isolated literal agraphia which are analogous to our first case, it is practically unnecessary to search our diagram for a designation. Nevertheless it is instructive to consider this analogy and the corresponding disturbances of speech; it then appears to be the much disputed conduction aphasia which forms the prototype for the assured possession of this form, so that a lesion of the tract $a\ \beta$ would justify the conception of conduction agraphia. As I have repeatedly emphasized, its nature is evident from the fact that in writing there is no conception of the form of the letters, while the power to produce them is still intact; hence those cases of agraphia in which the conception of the form of the letters is well retained, no matter how incorrect the writing, do not belong to the pure disturbances of written language, but to the sequels of disordered speech or disturbed communication of word conceptions. Such cases are therefore differentiated from the previously described literal agraphia as verbal agraphia. That this verbal agraphia may possibly result from a lesion of the tract $c\ a$ is shown by a case which

[1] G. Wolff, "Ueber krankhafte Dissociation der Vorstellungen." *Habilitationschrift*, Leipzig, 1897, pp. 43 *et seq.*

I mentioned in my previous report; certainly it is an extremely rare occurrence, for I have never seen anything similar. It is quite conceivable that cases of sensory aphasia which have recovered, and these are not infrequent, may leave such a circumscribed defect because restitution if not perfect may perhaps be incomplete in only one respect; namely, that the most difficult function is impaired. Without doubt the transmission of the internal word conception to its written expression is the most difficult function which is here called into exercise. This view bears out the general experience that among all the symptoms of aphasia the disturbance of written expression is the most intense and tenacious. This explains why paraphasic symptoms which are not noticeable in speaking are observed in writing. It often happens that paraphasia which is only slightly evident in speaking becomes most obvious on spontaneous writing, or the existing paraphasia may be greatly aggravated. This condition is frequently noted in paralytics with aphasia of varying degree.

Paragraphia in a restricted sense, or literal paragraphia, in which the form of letters is distorted, is of practical importance, being the disturbance of the power to write noted in paralytics. A tentative diagnosis of progressive paralysis may not infrequently be made by observing a few words which contain distorted letters. A general impairment of the memory in senile or other organic atrophy of the brain, in presbyophrenia and polyneuritic psychosis, produces a similar paragraphia, apparently because the patient forgets the exact formation of the letters.

A review of the disturbances of written language shows that I was justified in claiming that we are dealing with essentially transcortical symptoms, and it is especially interesting to note that these transcortical symptoms may follow lesions of quite definite areas of the brain, just like cases of pure word blindness and so-called pure literal agraphia.

It is not a mere accident, therefore, that the study of written language has up to the present time enabled us to arrive at most valuable conclusions concerning the purely transcortical process which, according to prominent authors, we may most aptly designate as "word finding." Word finding, particularly for individual letters, is a prerequisite for ordinary reading by means of spelling and, for practical purposes, the letters which must be found are like all other word conceptions. Therefore, reading is a part of the expressive portion of the speech process, in which the finding of words for different conceptions is the first and most necessary object. According to Rieger and G. Wolff, word finding for letters that are seen occurs normally and unisensually by optical conceptions, like those of color. Word finding for letters is, however, the most necessary and most common part of expressive language; therefore, in comparison with all other visible objects, it may be preferable or easiest. In Rieger's case word finding for visible objects was about twice as rapid as for others; in Wolff's case (identical with Grashey's) it occurred instantly, and apparently even without the otherwise necessary writing movements, for reading was immediately possible. For word finding the optical memory pictures of the letters equals the different memory pictures of concrete substances which form the conception, *B*. If we cast a glance at Fig. 137 it at once becomes apparent that the direct communication, *B b*, which we must maintain for clinical purposes, is probably unnecessary for letters,

hence we postulate merely the association tract, *B a*. This tract which, like *B*, here equals the optical memory picture, is contained in the optico-acoustic commissure (Bastian) of the same and of the crossed side. Its anatomical foundation for the same side is perhaps the lower longitudinal bundle; for the crossed side, the forceps tapetum tract, as it was called by H. Sachs.

C. S. Freund and H. Sachs also assume that the same association tract produces word finding for objects shown, and its injury is held responsible if, under some circumstances, an object which when first seen cannot be correctly named, although it is clearly recognized, is correctly designated after examining it by the sense of touch.

For this symptom, if restricted as above, the name optical aphasia (S. Freund) is not inappropriate. It occasionally appears in the form just described, and then belongs to the interesting group of conditions known as "dissociation of conceptions." It cannot, however, be referred to the previously mentioned tract, since all experience shows that for visible objects a recognition of their meaning is indispensable to word finding, and no case has yet been observed (for instance, of mind blindness) in which objects not recognizable by the sense of sight were nevertheless correctly named. For the recognition of visible concrete things their secondary identification (in this case the association of their optical fields of memory) is absolutely necessary for the tactile conceptions belonging thereto. As we have seen, the conditions are different with letters.

Although most conspicuously lacking for visible objects, the symptom of impaired or entirely arrested power of word findings may usually be determined more quickly than any other impression of the general senses. In Rieger's case all of the special senses responded to the same stimulus which proved so successful in word finding, but they invariably required a certain time which was about uniform.

The preponderating power of the sight in comparison with the other senses was noticeable in the Wolff-Grashey case; the object seen, not merely the one felt or heard, enabled the patient to find the word (in writing). In the unisensual conceptions of the tactile realm some exceptions were noted, such as wind, heat, cold, and in the auditory thunder, polka, waltz. That word finding for such paramount tactile conceptions as smooth, rough, pointed, sharp, and the like, requires the conjoint action of optical perception, also proves the special influence in this case of optical memory pictures.

From the valuable work of Wolff it appears that various conceptions, according to their meaning, cannot be composed of partial conceptions or memory pictures of equal value, but that a main determining area of special sense, and secondarily of memory pictures, may be differentiated. He presumes by analogy that the sense of sight is the chief and determining one for word finding, as in his case; but this is quite unlikely, and is disproven by Rieger's case. If, however, in Wolff's case, we ascribe to the optical tract a comparatively better retained function than to the other projection fields, we can readily understand its exceptional action, otherwise never observed, which shows that the patient found his words by the roundabout method of writing pictures.

When, as in the cases of Rieger and Sommer, the power to find words

for letters is so disturbed that only the perception of certain letters is missing, others being perfectly unimpaired, we may quite properly maintain that the patient had lost all conception of the letters in question. Much more common is a condition in which there exists merely an uncertainty in word finding, especially for certain letters, without this symptom being constant. In spite of this, patients can occasionally read without effort words containing letters of which previously there had been no conception. An example of this was furnished while our case of agraphia was under observation. Inability to read because of the loss of individual letters was much more marked in a case of Bastian's in which, in consequence of extreme paraphasia, not a single letter could be correctly named nor could words be read aloud; nevertheless the patient understood what he read. I mention these cases because, more than any others, they expose to probable criticism our view that reading is the result of spelling. They are explained, however, by two generally recognized experiences. The one is this, that every virtuosity in the sequence of individual acts, which is acquired by practice, suffers as soon as close attention is directed to this individual act. The simplest example of this is running down stairs, with or without observing the individual steps. Attention drawn to the individual act, in this case manifested for the individual letters, is therefore a deleterious factor for the complete performance of an act, and we therefore understand that the complete act would much more readily follow than the individual performance. A similar experience is found in the more frequently observed cases of pure word blindness, in which ordinarily there is no impediment of speech nor paraphasia, but in which the name for objects shown cannot be recalled or only with the greatest difficulty. Here also the attention bestowed upon the object may prevent the word finding. It appears to me that Bleuler maintains the same in differentiating the more automatic and fully conscious association processes. A second general fact, in which spelling appears to compensate for some defects in reading, is the familiar one that all impression processes are facilitated by secondary identification. The most marked example of this is the recognition (in spite of demonstrable and severe sensory disturbances) of objects that are only felt. That this process, closely akin to guesswork, normally plays a great rôle, is known to us all (as in the reading of handwriting).

The celebrated case of Grashey was first explained by the supposition that the aphasic disturbance and inability to find words were due to a purely functional disturbance, namely, the loss of memory for recent impressions or, more accurately, a decrease in the power of memory. I formerly agreed with the opinion that in Grashey's case it was necessary to recognize a special form of amnesic aphasia. In the meantime, Sommer showed the untenability of this view, and G. Wolff's subsequent careful investigation of the case led to the same result. The peculiarity of Grashey's case must not be sought in the nature of the aphasic disturbance, but in the artifice dependent upon the individual activity which the patient resorted to in order to conceal the actual aphasic defect of word finding for visible objects. We have noted how this artifice favors the fact that our written language is essentially a language of letters. The difficulty of word finding has, however, always been regarded as an important aphasic factor; very early Trousseau, for example, contrasted it with Broca's aphasia, and it actually corresponds to the practical require-

ments according to which Pitres[1] lately included under the definite term of amnesic aphasia all cases in which this symptom is either observed alone or is prominent in the clinical picture.

We cannot go beyond the recognition of this practical requirement, for, on the contrary, we must emphasize the other variations in the cases which in this one point agree. The majority of cases of isolated word blindness belong in this group, also Rieger's case, and the very peculiar case of Grashey. Both of these have the common symptom of an almost complete loss of memory, but this phenomenon alone could not have been the cause of the inability to find words, for this is absent in some cases of pure word blindness marked by difficulty in word finding. Besides, the majority of cases are of different origin, for they are usually cases of aphasia in which there is a certain compensation as regards speech.

Pitres collected 10 cases in 8 of which he found that the lower parietal lobe was implicated; this is not mere coincidence, but agrees with Naunyn's statistical report that in the cases which he called "indefinite aphasia" there was the predominance of a lesion at the boundary of the lower parietal and occipital lobes. Of all the disturbances of speech, motor aphasia appears to be the one in which, when recovery follows, a real disturbance of word finding is most rarely found; we are therefore forced to agree with Pitres, inasmuch as he also fails to include the implication of Broca's convolution. For the rest, however, the great dissimilarity of the cases proves either that very different localities are implicated, or that functional disturbances of different localization must act together. Pitres, who recognized the majority or the generally acknowledged disturbances of speech and written language, emphasizes the great rarity of pure cases which would fulfil all of his requirements. He considers it essential that they show absolutely no disturbance of the understanding, either in reading or in writing, but only a difficulty in word finding.

The meaning in such cases of a severe impairment of the memory has not escaped him. He points to the universal experience that a patient with difficulty in word finding, notwithstanding repeated instruction, has the same difficulty in finding definite words (which he gladly accepts if prompted) as when only the beginning of the word is given him. Moreover, Pitres attempts to explain the want of understanding in reading by the theory of a disturbance of the memory, but this is opposed by Grashey's case of markedly impaired memory with intact ability to read. It is very interesting, however, to know that Pitres does not absolutely accept the view that the lower parietal lobe is the seat of disturbance; on the contrary, he recognizes that in word finding the stimulus of the true speech center is distributed by neurons over the entire cerebral cortex, and these are to be regarded as the carriers of psychical operations; the lesions therefore have no fixed topography, but act by the interruption of the commissure between the different centers of the word pictures and the cortical areas in which the higher psychical acts are carried out. "Amnesic aphasia consists solely and alone in a lack of connection between the conception and the word which serves to express it." In a note we even find the statement that amnesic aphasia should properly be designated "transcortical aphasia."

[1] A. Pitres, "L'Aphasie amnésique et ses variétés cliniques." Paris, 1898.

For the practical recognition of amnesic aphasia it might be advantageous to differentiate between those cases which show only a difficulty in word finding and those which disclose, in addition, the symptoms of paraphasia. The latter, by far the most common, are almost always the residua of past disturbances in speech, and therefore need not be considered. Aside from these, difficulty in word finding shows itself almost exclusively or most markedly when objects are presented to view (and also probably when listening, touching, etc.), and sometimes by the absence of definite expressions, especially substantives, in the otherwise fluent and correct spontaneous speech. A defect scarcely noticeable at first becomes more prominent when we ask the patients to name objects that have been shown. To cover their confusion the patients frequently use roundabout expressions which denote embarrassment, such as "thingumbob." The choice of incorrect or unsuitable expressions, however, belongs to the realm of paraphasic symptoms.

The difficulty in finding words may, as is well known, be met with under normal circumstances. Rieger very aptly remarks that this occurs in normal persons only when using words of limited importance; for example, the word "magnolia" cannot be recalled. "When, however, a person can no longer say 'This is a tree,' he must unquestionably be regarded as having a disease of the brain." It is also true that the extent of these terms of limited importance may vary greatly according to the occasion, therefore according to the mental condition in which the person happens to be. Rieger, so far as I know, was also the first to call attention to the fact that all persons whose command of a foreign language is imperfect show a very marked inability to express themselves in words, many of which they may understand perfectly. Were this equally true of their mother tongue, it would correspond to our conception of amnesic aphasia. Under the same conditions an increase of this defect may be apparent when they attempt to name objects shown them. This is generally more difficult in the course of conversation than at other times, and probably is due to the closer attention devoted to the object.

Pitres shows that the "systematic aphasia" of polyglots presents a stage in which word finding in the foreign tongue is impossible, while the power to understand has been retained; therefore a stage of amnesic aphasia.

After this description it is obvious that difficulty in word finding is nothing more than a special form of transcortical motor aphasia; in other words, a disturbance of the function of the tract *B b* of our diagram. In Rieger's case, as well as in that of Wolff-Grashey, this disturbance consisted of a slowing in the function which, however, was finally performed correctly. In other cases, word finding is either impossible or to a certain degree is accidental, and this is prone to be the case in the individual whose brain is not actually diseased.

Nothing is more obvious than the assumption that a disturbance within the complicated tract *B* is the cause of the difficulty in word finding. Aside from the already mentioned defect in the conception of letters, the fact that in Rieger's case there was also a decided and demonstrable impairment of conception favors this view; all conception of playing cards, coins, paper money, stamps and similar things had been completely obliterated, and it was impossible to awaken any conception of these objects, as well as of any figures higher than figure 3, of algebraic signs, of musical notes, of the mean-

ing of piano keys, or of punctuation marks. Something analogous, a decrease in the number of conceptions, appears only in consequence of mental disease, and the condition Rieger reported reminds us most forcibly of a case of aphasia following mental disease which I described, as did also Heilbronner in its later stages;[1] in this instance partial motor and sensory aphasia were manifested by the fact that there was only a number of words for which conception was lacking. For the retained power of conception we assumed a damage of slighter extent, namely, a decreased stimulation of the conceptions themselves as well as of the partial conceptions of which they were composed (for instance, memory pictures), which change we were forced to assume by the clinical findings, since the slowing of the process for the recognition of objects could be physiologically explained only as a slowing of the stimulation of some special sense. The same slowing could be demonstrated in regard to the understanding of words which express the conceptions. The contrast between word understanding and word finding, which might be urged in opposition to the theory of an affection of the conceptions, is therefore not absolute; both processes were slowed, but one much more so than the other. When objects were shown or handled, etc., the period of time required for the recognition of the conception could be determined, and it was at the same time evident that the chief delay was caused by the act of word finding.

At the same time it was demonstrated that the conception itself, not its partial memory pictures, always led to the knowledge of the name. In Wolff-Grashey's case the conception *in toto,* not a partial conception, produced the word. To the above emphasized unisensual conceptions there were naturally some exceptions. The contrast between sensory and motor functions, between the apparently retained understanding of a word and the difficulty in word finding, is also evident here, but the intactness of the conception itself is far from proven. If, for example, in answer to the question "Is the leaf blue?" Wolff's patient had answered "It may be" or "I do not know," this would indicate that his understanding of the sense of the words "leaf" and "blue" was also affected; therefore, for the receptive portion of the process of speech, a defective conception was demonstrated. If the condition is such as to necessitate a certain stimulation of the entire association complex which we term conception in order to innervate upon the tract *B b* the correct word conception, or the correct movements of word conception, we may understand the influence which perception by the senses may under some circumstances have upon word finding: The corresponding conception thereby gains in intensity. This influence is greater the closer the relation of distinct individual ideas which together form the conception. In referring again to the diagram, Fig. 137, the importance of the foregoing conclusion is obvious. No less than four equally important innervation streams must unite in *b* in order to produce the correct word conception.

We now understand why disturbance in word finding is so common after focal diseases of the brain which run their course with sensory defects. Of the many tracts which unite in *b*, only one or the other will be damaged, only the innervation of a partial conception need be decreased, to render

[1] *L. c.*

word finding difficult. That conceptions are also less intense under relaxation is readily understood.

It may perhaps not be deemed superfluous when I remark that neither in Grashey-Wolff's case nor in Rieger's case had the understanding of speech suffered, for both patients were able to repeat words without any slowing of the speech.

Usually we refer to transcortical motor aphasia only when the impairment of the power to find words has increased to an entire absence of spontaneous speech or to a state which almost equals this defect. If, however, the disturbance is confined within the limits of amnesic aphasia (Pitres), the phenomenon of a focal disease is apparently no longer present, but we suspect a disturbance of conception or intellect which, as we have seen, finds its analogy chiefly in mental diseases. Since Rieger has given his excellent directions for the investigation of such cases I do not doubt that in most instances some such intellectual defect will be found. Any focal symptoms which are also found will be only of secondary importance as indicating the area which is the seat of general decrease of cerebral activity; as, for instance, in cases of post-apoplectic dementia. That the symptoms of transcortical motor and sensory aphasia due to senile atrophy of the brain may frequently be recognized even without traces of a focal disease has been shown by Heilbronner. In such cases there are usually signs of mental blindness, or asymbolia, which, when there is mental disease, depends upon a disturbance of the power of secondary identification. To enter more minutely into the study of these phenomena is impossible, since the richness of their number and their importance demand special treatment. But I must point out one of the most recent developments in this borderland between mental disease and focal affection of the brain, because it is most intimately connected with the explicitly treated theme of word finding. This is the pathologic picture of unilateral apraxia, constructed and masterfully described by Liepmann. As an unusually favorable coincidence is necessary in order to produce the typical pathologic picture of transcortical motor aphasia, we also owe it to such a coincidence that in Liepmann's case a focal disease of the brain was so situated, and of such nature, that it severed the innervation of the transcortical tracts for the motility of one-half of the body. Moreover, as the patient in consequence of subcortical motor aphasia was deprived of the power of speech, the intelligence could be tested only by means of his motor reactions. The reaction of the members on the right side of the body indicated that the most extreme psychical defect, absolute dementia, existed. However, when the left members of the body were exclusively used in carrying out the reactions, a comparatively high degree of intelligence could be demonstrated. The autopsy of this case, which has not yet been published, confirmed the diagnosis of focal disease of the brain.

I shall not give explicit instructions for the examination of aphasics; it seems unnecessary, for this article embodies the main points. But two phenomena require special mention and careful consideration. One of these is the extreme *exhaustion* which these patients often manifest and which soon compels the physician to interrupt the examination, which can be only resumed after the patient recovers from the fatigue. If no attention is paid to this point, the different examinations will lead to absolutely contradictory results.

The second source of error is *perseveration* (Neisser); it consists in the inclination of the patients to repeat a reaction they have once found, even though they know or observe that they are doing wrong or are speaking incorrectly. The patients often express their annoyance at this, and show plainly that the second reaction was not intended. They are therefore subjected to a certain constraint. In the case of aphasics this very common symptom is extremely annoying, for the physician is often forced to pause, or to divert the attention of the patient into another channel, in order to obtain a new and correct reaction. As A. Pick has demonstrated, this disturbance may be so increased as to form a variety of pseudo-apraxia.

I cannot conclude these essential, clinical, symptomatologic considerations without discussing more explicitly the question of the unilateral position of the centers of speech, as set forth in the preceding. My entire description is based upon the fact, learned by experience, of the unilateral position of the so-called centers, and this fact must have been particularly impressive, for one of the earliest investigators in attempting to localize these centers maintained that the left hemisphere of the brain was exclusively the seat of the function of speech (Marc Dax).[1] In my presentation of the subject I have referred only to the left hemisphere, my purpose being to speak of the exceptions subsequently. Here two series of facts, abstractly connected, must be chiefly considered. One series of exceptions relates to speech disturbances in left-handed persons: These are almost exclusively due to disease of the right hemisphere, the seat of the focus and the symptomatology bearing the same relation to each other as the corresponding focal diseases of the left hemisphere in the right-handed. We do not know how to explain the fact that the conspicuous right-handedness observed in most persons, i. e., the choice of the right hand for all movements which require great strength or dexterity, causes the functional development of the speech center situated in the left half of the cerebrum, and only this one principle is absolutely plain, that the unilateral location in itself appears justified because it indicates a conservation of energy. A step forward is noted in Oppenheim's report of a case in which marked sensory aphasia due to a tumor appeared simultaneously with left-sided hemiplegia; the history revealed the fact that the patient was not left-handed from birth, but that in her seventeenth year, after sustaining an injury to her right hand, she was compelled to use the left, and had since become left-handed. Autopsy revealed the suspected tumor in the right hemisphere. This observation leads to two conclusions: first, that the right hemisphere may assume the function of speech even in adult life in place of the left hemisphere whose function it is normally; second, that the left hemisphere may completely lose the previously acquired function of speech if the person becomes left-handed. I know of no other observation which so strongly confirms our view that the localization of speech centers is the functional acquirement of each individual.

The other exceptions are pathological experiences in childhood. In focal diseases of the left hemisphere in childhood, disturbances of speech are observed which vary in degree according to the development of the child; these

[1] The first investigator was Bouillaud, who taught that the anterior lobes of the brain were the seat of the faculty of speech.

are proportionally and rapidly compensated for, even when subsequent findings reveal the complete destruction of the well known left-sided speech centers. Apparently, especially in childhood, the preexisting left-sided cerebral function is readily transferred to the right hemisphere. Under these circumstances left-handedness is not necessarily at the same time produced; if the case comes to autopsy at a more advanced age there may be no symptom referable to the left hemisphere, and the lesions may simulate those in right-handed persons; or, fresh lesions of the right-sided centers may cause aphasia in persons who are not really left-handed. As is evident, the faculty of purely individual acquirements belonging to the centers in question leads to a number of possibilities which, under some circumstances, especially when the history is obscure, may form an incomprehensible exception to the otherwise regular localization of the speech centers. In fact we cannot deny a greater individual latitude than heretofore to the presumption, according to which at one time the left hemisphere, at another time the right is exposed, or perhaps both in the same case, with or without the predominant assumption of the function of speech by one hemisphere.

This point of view becomes especially important when we consider the greatly discussed question whether in the fully developed brain, and in how far, a substitution for destroyed speech centers takes place by means of tracts in the same localities of the other hemisphere. The explanation of the symptoms during convalescence from aphasic disturbance, in the widest sense of the term, depends chiefly upon this prior question, but, concerning it, we are still in ignorance. It is generally conceded that slowly growing pathologic foci which act exclusively by displacement, at least to the same extent as is otherwise the case, produce no symptoms, not even of speech disturbance, although the autopsy findings may indicate that the center or the tracts under consideration have been severely affected. But this experience is by no means general. There are cases which appear to prove the contrary, as for example, Oppenheim's case, in which during the growth of the tumor the transmission of the sensory speech function to the left hemisphere would have been much more likely, as this merely involved the resumption of a function previously exercised. The explanation of this contradiction need not be based entirely on individual differences. It may readily happen that the number of general symptoms accompanying the focal symptoms will guide us to a decision. The two focal diseases which here come chiefly into consideration are *brain tumor* and *brain abscess*. These diseases may sometimes present but few accompanying general phenomena, at other times the general symptoms are most pronounced. The activity of the other hemisphere is to the same extent preserved or damaged; and it is obvious that its injury does not favor the reestablishment of function. According to general experience, the sensory speech function shows a greater resistance to injury than the motor. In this connection it is more strictly correct to differentiate between the impressive and the expressive parts of any speech process. The conditions are clearer when we consider the substitution by the other hemisphere in pure diseases of the brain, or in those in which there is a predominant destructive action, such as cerebral hemorrhage or cerebral softening. Here the possibility of restitution by a new arrangement may be materially influenced by the general symptoms of the apoplectic insult, for we have often noted that a

severe attack, besides leaving unilateral focal symptoms, produces general injury to the brain as shown by the ready exhaustion, the impaired memory, and the emotional weakness which become chronic. Since experience teaches that the accompanying insult in motor aphasia is usually decided, and in sensory aphasia and pure word blindness is usually only slight or absent, we also conclude that this favors restitution of the sensory speech function, which is the rule, and heightens the importance of pure alexia as a focal symptom.

Concerning the unilateral position of the speech centers there is another condition which, in my opinior can only be explained by the theory that the

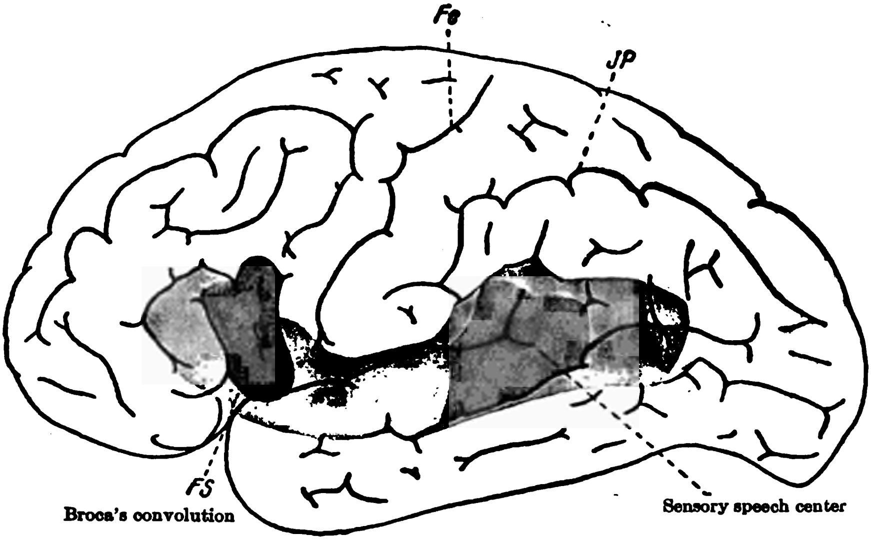


FIG. 139.

motor or, more correctly, the expressive, portion of the speech process is not always, and is not exclusively, unilateral. As is well known, the indirect focal symptoms of acute focal disease of the brain after well marked apoplectic attacks in general are limited to the affected hemisphere. To this, however, there are often exceptions; for instance there may be a more or less well developed motor aphasia and a resulting left-sided hemiplegia after severe apoplectic attacks. This condition usually lasts for only a few days, a proof that we are not dealing with left-handed persons in whom aphasia, even as an indirect focal symptom, must persist for a longer time. It can be explained only by the assumption that in the affected individual, prior to the attack, the right hemisphere with a certain portion of the process of innervation was implicated in active speech; when, therefore, the apoplectic insult originating from the right hemisphere causes any injury whatever to the left hemisphere, obviously that function which was previously exercised by the conjoint action of the right hemisphere is most easily affected by the disease. Whether we are here dealing with a permanent damage to the left-sided Broca's convolution by the trauma of apoplexy, or only with a temporary func-

tional disturbance, will be evident in the further course. There is a symptom which is positive proof that the apoplectic insult has damaged Broca's convolution. This is the condition designated as *syllable stumbling*, usually regarded as a reliable indication of progressive paralysis, and therefore a so-called *paralytic speech disturbance.* Exactly the same speech disturbance, syllable stumbling, is observed in the majority of patients who have recovered from motor aphasia, even though it has been merely an indirect or transitory focal symptom of the affection of the left hemisphere. Little as this fact is appreciated, it is nevertheless proven by many experiences, and I call special

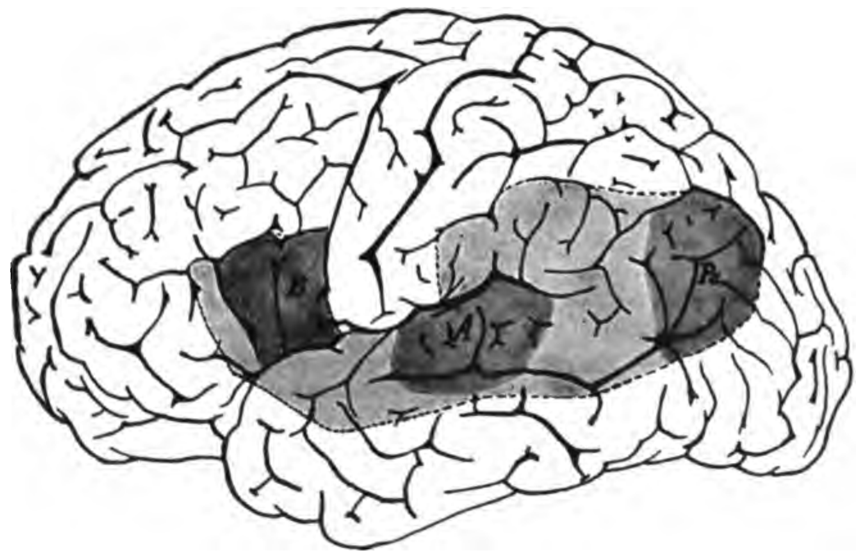


FIG. 140.

attention to it because of its importance in differentiating this affection from progressive paralysis.

We must now discuss the *anatomical region of speech in the brain*, or, as Déjérine briefly called it, on the basis of the preceding description, the "zone du langage." I believe this cannot be better done than by presenting the illustrations (Figs. 139 and 140) taken from the works respectively of Monakow and Déjérine; the darkly shaded portions in both illustrations are the centers which have been positively recognized, and the shaded areas represent the speech regions. On comparison these two illustrations show a very satisfactory agreement, especially when we consider that the two investigators worked independently. It is evident that, with the exception of the anterior processes and the lower connection of the central convolutions, the entire first embryonic convolution, i. e., the convolution limited by the Sylvian fissure, may be included within the speech region. The portion situated in the frontal lobe corresponds to Broca's convolution, therefore to the motor speech center; that situated in the temporal lobe, therefore below the Sylvian fissure, is the sensory speech center; that above the termination of the posterior Sylvian fissure, the so-called marginal convolution which is also known as the

supra-marginal convolution, both authors have included within the regions of speech, without having assigned to it a definite and specific function. These authors are also unanimous in no longer considering that the part above Broca's convolution, the so-called foot of the second frontal convolution, belongs to the speech region, although Exner, Charcot and Pitres, and recently Ziehen, locate the motor writing center in this region. Moreover, these authors concur in believing that the part of the convolution situated in the lower parietal lobe and adjoining the first embryonic convolution is still included with the speech region, inasmuch as it is prolonged posteriorly by means of an anastomosing and communicating bridge to the second embryonal convolution. This area extending horizontally and posteriorly is the angular convolution, the "pli courbe" of French authors. Déjérine also includes in his speech zone a neighboring part of the second temporal convolution, but this is not depicted in Monakow's illustration; in my opinion it corresponds with the majority of the findings.

Within the shaded speech region of Monakow's illustration there are two, and in Déjérine's three, darker areas which, in the latter, are sharply demarcated; two of these almost absolutely agree, and they correspond to the more sharply defined sensory and motor speech centers. Déjérine alone indicates in a restricted sense a third center, which occupies the region of the angular convolution, and is designated by him as the center for optical memory pictures of words. I have explained above in what sense we are to understand this; v. Monakow does not recognize such a special center. The two illustrations show distinctly that the speech region not only consists of the centers previously differentiated but also embraces the neighboring parts of the convolutions of the convexity. Nevertheless, they represent only a part of the speech region, for, according to the two previously mentioned authors and almost all other authorities, the entire island covered by the first embryonic convolution and situated in the depths of the Sylvian fossa should also be included. In order to understand this, the Sylvian fossa must be deeply penetrated and the adjoining convolutions—those belonging to the embryonic convolution—should be separated. The island is then exposed to view as a deeply situated, triangular, cortical area covered with compartment-like convolutions which are separated from the cortical substance, according to Burdach, by three deep spaces, an anterior, an upper, and a lower space. Where the lower and upper spaces meet and form an acute angle, a deep furrow covered by the marginal and first temporal convolutions passes to some extent in the direction of the upper space and backward, so that in the brain it almost reaches the transverse plane of the angular convolution. The adjoining illustration (Fig. 141), which is taken from Henle's "Anatomy," gives us an idea of these relations.

The importance of the island for the function of speech, everywhere recognized, permits us to conclude that it is the objective point for the association tracts, and the area where the two centers of speech, and presumably also the different areas of the speech region, unite with each other. In fact the finer anatomy of the region of the island permits no other explanation than that we are dealing with an actual association center. This is obvious, not merely because the three deep spaces which bound the island show everywhere at their bases the well known U-shaped, so-called, fibriæ propriæ or laminæ

arcuatæ Arnoldi to be richly developed, but also because of the typical structure of the cortex of the island. As is well known, the extension of the cortex of the island reaches the external border of the largest ganglion of the brain, the lenticular nucleus. Between the lenticular nucleus and the cortex of the island are found two layers of white substance, of which that adjoining the lenticular nucleus shows a very delicate internal structure known as the capsula externa, that belonging to the cortex of the island a much coarser one following its outline, and designated as capsula extrema. Between the capsula externa and capsula extrema we note a structure of gray substance, the claustrum, which is found nowhere else in the brain. This compartment-shaped ganglion, formed like the cortex of the island, is broader from above down-

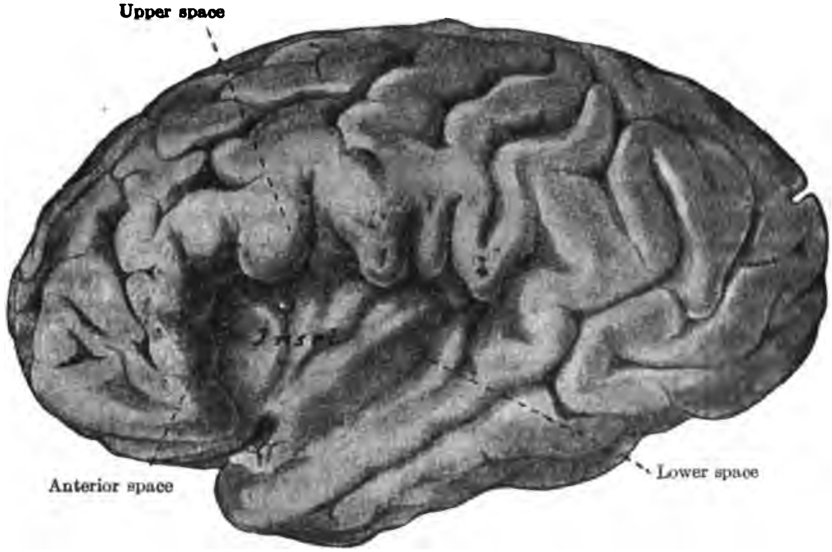


Fig. 141.

ward, while it narrows longitudinally; as Meynert has said, with its processes pointing toward the periphery it wholly disappears in the first embryonic convolution; he tells us, too, that it consists exclusively of spindle or association cells, and is nothing more than a cortical layer of unusual development and deeply penetrating the medullary substance which everywhere consists of spindle or association cells. In fact it disappears at the so-called foot of the island, i. e., at the lower point of the junction of the radially situated convolution of the island with some of the cortex of the island as well as with the substance of the amygdalate nucleus. Besides the cortex of the island and its claustrum, there are still two important association bundles which must be considered in the anatomy of the speech regions. One of these is the fasciculus uncinatus, penetrated by the widened base of the claustrum which divides it into several layers, and this suggests the thought of a connection between the cells and fibers. If the cortex is detached, the chief mass of the association bundle is found at the foot of the island and further toward the interior, and here is best seen its hook-shape which reminds us of the composition of

the laminæ arcuatæ. From this mixed piece the bundle splits and bifurcates into the two cerebral lobes (whose shortest combination it represents), the frontal and the temporal lobes. Some portions of it certainly extend to the third frontal convolution, including Broca's convolution and the speech region of the first temporal convolution.

While the fasciculus uncinatus appears to unite by the shortest connection with parts of the frontal and temporal lobes, the second bundle, or fasciculus arcuatus, is not actually a special bundle, but a general system of association fibers of the convex surface of the brain having a sagittal direction. Nowhere are the ganglion cells included in this. The anatomical conditions of the brain are such that a special bundle of association fibers is most readily produced where the foot of the corona radiata passes from the internal capsule, therefore above and outwardly from the lateral edge of the lateral ventricle to the upper border of the lenticular nucleus. As it is also contained in the substance which bounds the Sylvian fossa, it is perceptible in the hardened brain if the cortex of the upper space with its fibriæ propriæ is detached. This will then convince us that a special bundle penetrates the deep medullary substance of the marginal convolution as far as the first temporal convolution, encircling the posterior process of the Sylvian fissure in an arch. The portion of the arch extending sagittally in the former direction lies medially from the just described path, and the interparietal furrow penetrating deeply into the convexity as well as the posterior processes of Burdach's upper space narrowly constricts this portion. In this area fibers which originate from the marginal convolution and the posterior third of the temporal convolutions no doubt join the arch bundle. The medullary substance of the lower parietal lobe consists largely of these fibers, which are closely pressed together in an extremely narrow space. When we consider that a part of the anterior portion of the fasciculus arcuatus extends to the top of the lateral ventricles above the lenticular nucleus, and that its sagittal continuation for the parietal and occipital lobes is first narrowed by the previously described furrows, the usually very deep posterior end of the parietal or first temporal furrow at once makes it obvious that the medullary substance of the lower parietal lobe in a comparatively small space interrupts a large part of the association fibers contained in the fasciculus arcuatus.

I will limit myself to these anatomical remarks which mainly show the peculiarity of the lower parietal lobe (that is, the angular convolution) which must contain a very important passage for the region of speech. Besides this passage to the interior, i. e., toward the ventricle, we find anatomically three sharply demarcated layers, namely, the most external one, that of the lower longitudinal bundle, then the so-called optic radiation of Gratiolet, and, finally, the tapetum of the corpus callosum, which together form the sagittal, medullary layer of the occipital lobe; in this restricted space lie tracts of such varying importance that the appearance of a center may readily be simulated, and in my opinion this is the origin of Déjérine's erroneous conception. To those more deeply interested in these anatomical relations I recommend Déjérine's "Anatomy of the Central Nervous System" and my "Atlas of Sections of the Brain." Fig. 142 is taken from the work of v. Monakow, and shows a horizontal section through the left hemisphere of the brain at the height of the first temporal convolution, the third frontal convolution, and the

splenium of the corpus callosum. The three obliquely shaded areas upon the convexity show the seat of lesion in motor and sensory aphasia and in pure

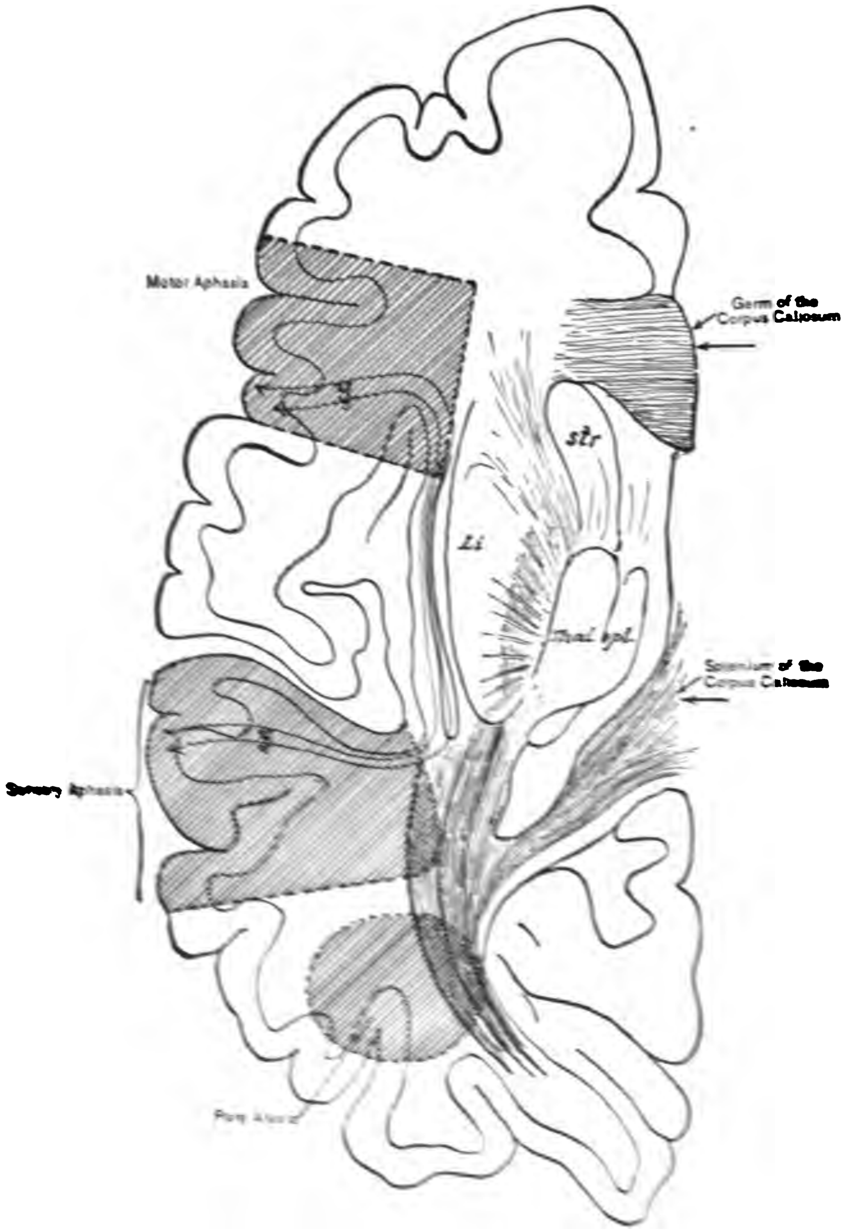


Fig. 142.

alexia. In contrast to the oblique shading, we observe in the illustration a long-stranded fiber mass composed of three bundles. It shows diagrammat-

ically the above mentioned sagittal medullary portion of the occipital lobes which in the hardened preparation is likewise sharply demarcated from the medullary layers of the temporal and lower parietal lobes, as in the illustration. The circular shaded area which is noted in the lower parietal lobe, v. Monakow considers to be the interruption of these three fiber layers which is a prerequisite for the occurrence of pure alexia, and which, according to his conception, arises from the destruction of the connecting link between the sensory speech center and the two occipital lobes.

As already indicated, I concur in v. Monakow's opinion that Déjérine's assumption that the cortex of the angular convolution is the seat of a unilateral writing picture center is not at all proven. Déjérine's observations do not permit this conclusion, but only prove that the destruction of this region invariably and severely impairs the power to write, and the degree of this action varies according to whether the deeper layers alone or simultaneously the superficial layers of the angular convolution are implicated. In the first case pure alexia results, in the second, alexia and agraphia. But in either case the cortex of the angular convolution may not be at all involved.

The anatomical relations which have been outlined merely permit us to conclude that the medullary substance of the lower parietal lobe, to a comparatively slight extent, severs all of the connections between the centers of speech and the two occipital lobes. In the near future autopsy findings must be utilized for the study of these points, and the following anatomical tracts, which I shall briefly enumerate, then come under consideration: First, the sagittal medullary substance of the occipital lobe with its three individual layers. The middle layer is the so-called "optic radiation of Gratiolet," the one which presents the most marked clinical symptoms, namely, the accompanying hemianopsia. The internal layer, the so-called tapetum, originates in the splenium of the corpus callosum, and contains at least two known tracts, namely, that which connects the two temporal lobes with each other, and that which unites the temporal lobe of one side with the occipital lobe of the opposite side, the forceps tapetum tract of H. Sachs. It also appears certain that the latter tract, which corresponds to the crossed optico-acoustic commissure of Bastian, passes the splenium of the corpus callosum. The external layer, as the fasciculus longitudinalis inferior, permits a free communication between each of the temporal lobes and the occipital lobe of the same side; it may, therefore, perhaps contain Bastian's optico-acoustic commissure of the same side. According to Probst's recent investigations it consists exclusively of corona radiata fibers which pass to the optic thalamus; this, however, I cannot confirm. It may be admitted for the upper half of its extension, but for the rest we acknowledge only its importance as an association tract between the temporal and occipital lobes. The sagittal medullary layer with its three component parts is situated not only within the lower parietal lobe but also within the sensory speech center in the posterior third of the first temporal convolution. Secondly, the true medullary substance of the angular convolution lies not only external to, but also above, the sagittal medullary layer. In a diffuse medullary mass, which cannot be further analyzed, it contains at least two principal constituents, namely, the fibers of the corpus callosum originating in the splenium of the corpus callosum which comes from the forceps major, bundles of which permeate the sagittal medul-

lary layer, and the above described component parts of the fasciculus arcuatus. This is composed of all the tracts which unite the motor cerebral regions with the occipital or lower parietal lobes of the same side, and is the shortest means of communication between the sensory speech center and the lower parietal or occipital lobes. The above considerations make it seem to a high degree probable that, besides destruction of the sagittal medullary layer, the intactness of the latter tracts contained in the medullary substance of the angular convolution is a prerequisite for the production of pure alexia.

Autopsy findings in pure word blindness combined with hemianopsia do not always reveal merely one focus localized as in v. Monakow's illustration, but often several foci interrupt the optic radiation, that is, their point of origin in the occipital lobe, as well as portions of the fibers of the corpus callosum. The splenium of the corpus callosum is especially involved, and it is pointed out that the same artery—the posterior cerebral—supplies the occipital lobe and the splenium of the corpus callosum, the regions belonging to the area of softening. Which portion of the corpus callosum is interrupted, whether the forceps major which unites symmetrical areas of the convexity or the forceps tapetum tract of H. Sachs, is still doubtful.

The tapetum of the temporal lobes can no longer be supposed to exist in the area designated by v. Monakow (identical with that of Déjérine), for it lies too near the occipital region; hence my thorough description, previously given. This, however, does not preclude the consideration in the clinical picture of both tracts with their variations; for example, the ability to copy may be due to this.

In the relatively rare form of pure, isolated, literal agraphia, the approximately definite seat of the focal lesion may be determined by the comparison of a few positive cases. This corresponds to the point where the sensory corona radiata of the extremities decussate certain deeply situated portions of the fasciculus arcuatus, its frontal plane lying anterior to the angular convolution in the region of the marginal convolution, its horizontal plane being higher than the lesion of the medulla of the angular convolution found in pure word blindness. Explicit details will be found in the report of my case.

The complexity of the anatomical relations of the regions of speech renders it obvious that objections may often be made to the meager description of the pathologic findings in those infrequent cases which have been thoroughly investigated clinically and reported by reliable observers. A purely casuistic standpoint does not affect the utilization of autopsy findings, for, on the contrary, we then find that almost all such experiences as I considered of general application are opposed by other observations. The unbiased study of C. H. Bastian's valuable report, noted for its rich material, and the most exhaustive compilation on the subject since the time of Kussmaul, forms the best proof of this statement. In spite of this, it is a great satisfaction to me to maintain the operation of a fixed law in this realm, which is also the standpoint of A. Pick, v. Monakow, and Déjérine, investigators who, in addition to their undoubted clinical ability, have at the same time shown themselves to be masters of the structure of the brain. The following remarks are based partly upon the observations of these authors, partly upon my own experiences and conclusions.

As to the exact limits of the motor and sensory speech center, I shall

confine myself to the description given above. The anterior obliquely shaded area in Fig. 142 shows the usual extent of the destruction in cases of pure motor aphasia; this, according to our nomenclature, at the same time includes verbal agraphia. Most cases of motor aphasia without agraphia have proven to be subcortical motor aphasia. According to the findings at hand, we must also reckon with a partial destruction of Broca's convolution, a damage to the lower part of which toward the Sylvian fossa causes word mutism; the integrity of the remaining parts, according to v. Monakow, permits the power

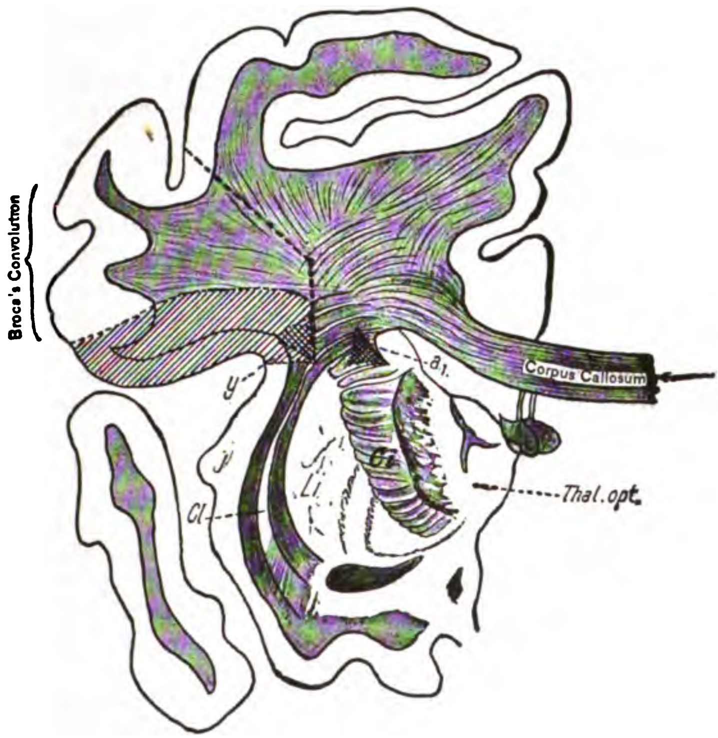


FIG. 143.—*Cl*, Claustrum; *Ci*, internal capsule; *Li*, lenticular nucleus; *y*, fasciculus arcuatus; *a*, secondary degeneration after cortical motor aphasia. (After v. Monakow.)

to write. Fig. 143 shows the seat of the lesion in cases of cortical motor aphasia which, exceptionally, are not combined with agraphia.

The secondary degeneration which persists after cortical motor aphasia has its definite seat in the internal capsule, indicated by a_1 in the accompanying figure. The accurate seat of the slightest destruction which causes subcortical sensory aphasia or pure word deafness can unfortunately be proven in only a single case, but in this, in my opinion, absolutely beyond doubt. It is the case of Liepmann, previously mentioned, the autopsy report of which was recently published. On account of accompanying conditions this proof was merely by exclusion; the destruction of the corona radiata of the temporal lobes at the point where it unites with the island of Reil was evident, but this proof was convincing because at the same time a secondary degenera-

tion of the tapetum of the temporal lobe of the opposite side, the right hemisphere, was found which could be looked upon only as an old lesion in the medulla of the temporal lobe of the left side, since the cortex of the temporal lobe and the entire occipital lobe upon the left side were well preserved, the right hemisphere alone showing recent destruction. In explaining his case Liepmann quite properly refers to H. Sachs who, on account of the narrow compressed course of the corona radiata of the temporal lobe between the posterior and lower processes of the third link of the lenticular nucleus and the tail-piece situated upon the roof of the lower horn of the caudate nucleus, stated that subcortical sensory aphasia is most readily produced by an injury of this area.

Many years ago I quoted experiences[1] which show that within the posterior processes of Burdach's upper space must lie fibers whose injury produces motor aphasia. I believed at that time that these fibers extended to the internal capsule and formed a special motor speech tract, that they therefore contained the fibers of Broca's convolution which projected to the nuclei of the bulbar nerves. After much experience had taught me that sensory aphasia, if combined with motor aphasia, is usually overlooked, and that lesions of the internal capsule do not produce subcortical motor aphasia, I finally came to the conclusion that these experiences refer to cases of transcortical motor sensory aphasia. v. Monakow, who still adheres to the opinion that the impulse for spontaneous speech must be transmitted over the sensory speech center, positively declares that except the cortex of the island the fasciculus arcuatus is the only association tract between the motor and sensory speech centers. Aside from this view, Heilbronner has recently and quite properly emphasized that the richness of the fiber communications between the temporal lobes and the third frontal convolution forces us to consider the temporal lobe as the chief point of origin of the transcortical fibers for Broca's center. In deciding this question I attach great weight to a case reported by Heubner which admits of no other explanation than that the principal constituents of the fasciculus arcuatus were separated; certainly the layer intended for the medullary substance of the first temporal convolution must have ruptured. Clinically, however, this case unquestionably presents the characteristics of sensory as well as of motor transcortical aphasia. In this case there was an area of so-called cortical softening which took the form of a furrow, and the marginal convolution and the posterior third of the first temporal convolution almost appear to be sequestered from the surrounding cortical substance; this proves that foci of softening may be of remarkable form and extent, and that the conditions produced by nature in pathologic cases may present the most manifold combinations, and admit of the most instructive applications.

In regard to diagnosis, I refer to the leading facts in the general pathology of the brain, according to which, when focal symptoms are present (and to these the phenomena of aphasia in the widest sense belong) we must determine whether we are dealing with direct, indirect, or transitory focal symptoms. I shall not here describe these, as I take it for granted that they are known. But it appears to me that the transitory focal symptoms are not

[1] "Lehrbuch der Gehirnkrankheiten," 2. Bd., 173, 176, 179.

always sufficiently appreciated. I must therefore state that these phenomena are caused by the occlusion of an artery with succeeding compensation. The affected cortical area is then periodically in a state of ischemia, but does not soften because the circulation is re-established. Substantive and acute focal symptoms which disappear in the course of one to three weeks must always awaken a suspicion of this sort of development. In an individual case, preceding transitory focal symptoms may enable us to make a positive differentiation between embolism and cerebral hemorrhage, which in the majority of cases is otherwise impossible. The diagnosis of subacute and chronic focal diseases and also of aphasia is based upon the same common principle, i. e., the utilization of the accompanying general symptoms. The same is true of the diagnosis of aphasic disturbances occurring in meningitis and progressive paralysis; here I must emphasize that motor as well as sensory aphasia is frequently observed after paralytic attacks, and these usually share the favorable prognosis of the focal symptoms. In all cases, the preliminary question of the right-handedness or left-handedness of aphasic disturbances in childhood from which the patient recovered, the full possession, the absence, or an impairment of the ability to write prior to the attack, must be decided. Thanks to the researches of A. Pick, the processes of a chronic and progressive disease of the brain, namely, of simple cerebral atrophy usually of senile or alcoholic etiology, have recently been made susceptible to diagnosis. A. Pick has shown that, when general cerebral atrophy occurs, localized atrophies of more decided character may betray themselves as focal symptoms; one or both temporal lobes, and combined with this or even by itself the first temporal convolution, may be the seat of extreme focal atrophy. This shows itself by a diminution in the size of all the constituents of the convolution, of the cortex as well as of the medullary substance. The resulting pathologic picture is that of transcortical sensory aphasia, the occurrence of which is unilateral focal atrophy, although indicative of a slowly increasing process, is comprehensible because the generalized atrophy, although only slight, makes compensation by the other hemisphere difficult. A. Pick and Déjérine have given reliable descriptions of cases which, on account of accompanying extreme deafness, were considered to be subcortical sensory aphasia or pure word deafness. Liepmann, however, has proven that the clinical picture was that of transcortical sensory aphasia, but the conspicuous bilateral deafness rendered the diagnosis of the cases difficult. A more extreme bilateral deafness, especially if the atrophy is chiefly seated in the comparatively small sensory speech center at either side, may really lead to absolute pure word deafness, which of course prevents us from recognizing a simultaneously existing transcortical sensory aphasia. We must therefore reckon with the occurrence of such transitional cases. That the cortical function from the periphery can still be determined when a process almost uniformly implicates all parts of the convolution which suffers damage, although one and the same medullary layer of the convolution contains subcortical as well as transcortical fibers, is readily explained by the well known persistence of sensory memory pictures once acquired, and their faculty of complete reproduction even when very deficient impulses are transmitted in a distorted way from the periphery. The most familiar example of this is the fact that the tactile property of one hand may be retained even when there are serious sensory disturbances of both

nerve tracts, the median and ulnar, so that only very imperfect tactile impressions are transmitted from the periphery to the cortex of the brain. Monakow takes the opposite standpoint, and believes that this specific function of the cortex of the sensory centers may suffer damage by a slight disturbance in function; upon the basis of much experience I must deny this. The preceding explanation embodies the important diagnostic law that slowly developing sensory aphasia of a transcortical nature (the recognition of which may be rendered difficult by extreme deafness), always permits the assumption of a localized atrophic process in the first temporal convolution upon the left side, and we here have a rare example of a definite focal symptom permitting the recognition not only of the area but also of the nature of the pathologic process. Sensory aphasia due to embolism or thrombosis always occurs suddenly, and often with more or less conspicuous symptoms of apoplexy.

In the later stages of motor aphasia it will be noted that syllable stumbling, and in sensory aphasia a difficulty in finding words for objects shown, usually persist, and if there are deeper lesions of the affected centers further compensation is not to be expected. As a rule we cannot utilize the symptom of difficulty in word finding for a focal diagnosis; on the contrary, it is often merely the expression of a general impairment of function, which may at different times develop to a different degree. A. Pick has shown that improvement of sensory aphasia occasionally occurs by a stage of *agrammatism*, i. e., by a disturbance of speech in which merely the essential parts of a sentence are used, without connectives or conjunctions, and without any regard to the tense of verbs, as is common in sending a telegram, or with a very rudimentary knowledge of a foreign language. This fact also may be of value in the diagnosis.

In by far the great majority of well developed cases we must remember that of the two opposite types of arrested or still fluent speech, only the former can be designated as motor aphasia. With a well retained power of speech, paraphasia of varying grades is the most conspicuous symptom, inability to understand what has been said is usually not appreciated, even by the most expert observers, and in every case is a symptom that must be sought for and determined with the utmost care. Apparently the prevalent opinion in regard to the present situation, and its power to readjust itself, is the reason why such a gross defect is so frequently overlooked. This may very readily occur if partial compensation has taken place for ordinary questions or commands. If motor aphasia at the same time exists, naturally an opinion as to whether spoken sounds are understood is even more difficult, therefore we must be the more cautious in examining for this symptom. In regard to disturbances in writing, I refer to my above descriptions. Any one desiring additional instruction for the diagnosis of finer speech disturbances and their importance as focal symptoms will find these in A. Pick's book[1] which indicates a decided advance in this branch of disease.

[1] "Beiträge zur Pathologie und pathologischen Anatomie des Centralnervensystems." Berlin, 1898.

TREATMENT

In the therapy of aphasia I may be brief, as this can only come into question when the focal symptom exists in an otherwise healthy brain.

The best treatment is exercise, which in sensory aphasia will of itself produce a gradual compensation of the defect. This spontaneous improvement is lacking when there are special circumstances, for example, an accompanying general atrophy of the brain or a bilateral disease of the temporal lobe. In motor aphasia, as has already been observed, even systematically conducted exercises are usually of little use. But the successes in the treatment of motor aphasia which Déjérine and Thomas report indicate that, under some circumstances, and after systematic practice, speech movements may be understood by the sense of sight, therefore all persons who understand this method should be advised to employ it. The few physicians who have devoted themselves to this specialty and teachers in deaf-mute institutions will be the most suitable instructors. In pure word blindness we usually succeed in teaching the patients to trace writing with their fingers, and by this method they learn to write themselves, and also learn to read. The patient whose history I related at the beginning of this article has in this way learned to recognize most letters and to read some words. But the method presupposes such attention, energy and endurance that compensation of the defect is only attainable in very exceptional cases.

My description will have given the impression that the aphasic symptom-complex, while to-day incomplete, forms a full, exceedingly rich, and interesting chapter in cerebral pathology; not in the remotest degree do I claim that it is complete, but I limit myself in the present state of our knowledge to presenting its principal characteristics, so that to some extent we may learn to recognize the outlines of this large and complicated structure. A few historic notes I have used for the purpose of elucidating certain points of view, which have so far aided in rearing this structure.

As in every investigation not yet concluded, we have here found that our field of activity appears to widen more and more, that even now the mind can hardly grasp all the facts that have come to light, and new questions constantly arise and demand an answer. If we penetrate beyond the true aphasic symptom-complex, we meet with ever expanding fields which are actually the offspring of aphasia; I need only mention *mental blindness, asymbolia* and *apraxia*. I am not claiming too much when I maintain that the pathologic picture presented by these affections would not have been correctly observed, described, nor understood if the points of view which I have delineated as the true kernel of the teaching of aphasia had not facilitated their understanding. Everywhere we meet with complicated psychical phenomena which are not infrequently also observed as partial symptoms of true mental disease. These form, in fact, a borderland between focal diseases of the brain and mental diseases. But even the restricted realm of aphasia furnishes in itself such transitional cases, which lead us to the conclusion that so-called mutacism, the strange muteness of patients with mental disease, is often only a peculiar form of transcortical motor aphasia. A minuteness of clinical observation and analysis, of which we may be justly proud, is begin-

ning to form a special pathology for localized psychical disturbances. I need mention only the researches of Lissauer into mental blindness, and of Liepmann into the pathologic picture of unilateral apraxia, both of which may be termed classic. A purely descriptive presentation of mental diseases which would include all of this recently acquired knowledge might be attempted. If the value of a scientific hypothesis depends upon the question in how far it renders research possible and leads to new questions, we must admit that the much disputed and actually the most disputable question, the transcortical form of aphasia, owes its origin to a valuable and at present indispensable hypothesis, which justifies and maintains its position. Moreover it gives me satisfaction to state that the best text-books, such as Oppenheim's, v. Monakow's and Goldscheider's, practically recognize the transcortical forms of aphasia, i. e., as indispensable for the symptomatologic differentiation of certain pathologic pictures.

THE NATURE AND THE TREATMENT OF DISTURBANCES OF SPEECH

By H. GUTZMANN, Berlin

Disturbances of speech are divided by a natural method into three principal groups: (1) Peripheral impressive; (2) Central; (3) Peripheral disturbances of expression. As the faculty of speech includes not only the expression of thought but the prior understanding of what has been spoken, we designate as peripheral impressive those disturbances of speech in which the means by which we learn to speak (upon which is based the entire apparatus of speech the individual parts of which are so difficult to recognize) are limited, are difficult of apprehension, or are even entirely absent.

PERIPHERAL IMPRESSIVE DISTURBANCES

In *peripheral impressive* disturbances of speech we ordinarily consider only the method by hearing and, as a matter of fact, this is the most common and the best way of learning to articulate. Children who from birth or from infancy have defective hearing or are deaf do not learn to speak at all; they are mute: *Deaf-mutism, surdo-mutitas.* In these cases, therefore, the areas of the brain which we assume to be the seat of sensory and motor speech are entirely unused, and do not develop. It is not always easy to determine what sense of hearing is possessed by a speechless child. While in older children the hearing may be accurately tested with tuning-forks, in younger children, under six or even seven years, this is not the case. I have frequently observed gross errors in testing the hearing of little children, and therefore I advise the utmost care in making this test, and that we base our conclusions upon what the parents themselves have noticed in regard to the hearing of the child rather than upon the results of our own examination.

I once examined a little girl aged 4½ years. The parents desired my opinion as to whether or not the child was a deaf-mute. The little girl, when looking at pictures or other objects as well as when at play, was often so engrossed that it was difficult to divert her attenion to any other channel. Her gait was steady, not dragging as is so frequently the case in deaf-mute children, nor did the child otherwise give the impression of being a deaf-mute. What we are to understand by giving the impression of being a deaf-mute cannot be described. There is no doubt, however, that congenital affections stamp a general impress upon the patient which cannot fail to be recognized by one who has observed many such persons. I was born in a deaf-mute institution and grew up among deaf-mutes, and therefore I am familiar with the general appearance of deaf-mute children without being able to define accurately its individual characteristics. This child *did not* impress me as being a deaf-mute, and when I stated this to the parents they declared that neither did they believe the child to be deaf. They

related to me a number of facts they had observed in the child: She could hear the tinkle of a bell and the ringing of a bell; she sometimes heard what was spoken and proved that she understood by some corresponding action, without any motion or indication on the part of the speaker which might aid the understanding. The child also made sounds of a senseless character while playing with her doll, a sequence of sounds which clearly imitated the speech melody of those about her; that is, sometimes high, sometimes low, sometimes loud, sometimes soft. At other times her reaction to impressions of hearing and also sometimes to impressions of feeling was very slight.

In the meantime the child was occupied with a picture-book which I had handed to her, and was so busy looking at the pictures that when from behind her I suddenly blew at her with my mouth she showed no reaction to this wave of air. Now we know that even adults, if greatly preoccupied, react but slightly to external impressions, and we say of them that they are entirely oblivious of what goes on about them. Yet in these cases there is no disturbance of sight or hearing. From these observations, therefore, I came to the conclusion that the child could hear, and that her deafness was not *deaf-mutism, surdo-mutitas,* but *idiopathic dumbness, audi-mutitas* (without deafness).

Only after the examination did the parents tell me that they had previously been to an ear specialist, and that after a long and thorough examination he had declared the child to be perfectly deaf. During the examination by the ear specialist, while the child standing at the window was completely engrossed with something going on in the street, the physician suddenly made a very loud noise with some instrument behind her. She did not turn around at this, and the ear specialist therefore assumed that she did not hear. A similar result was obtained by a test with the tuning-fork. In spite of this opinion I believed my diagnosis to be correct, and I was not a little pleased when six months later the little girl was brought to me again, and I found that she spoke as well as any child five years of age. *The power of speech appeared spontaneously soon after the consultation, and had developed very rapidly.*

I have detailed this case minutely because it is a classical case—idiopathic dumbness (without deafness)—and because it clearly demonstrates that in children of this age even the most careful examination by a prominent ear specialist may lead to erroneous conclusions. I must remark briefly that the child was quite intelligent. The only conspicuous feature in the case was the child's great preoccupation when at play or engrossed with anything which had attracted her attention.

If children are really deaf, other errors may arise; it may be supposed that they hear. As a proof of hearing it is often stated that such children hear a bell. We know, however, that extremely loud and shrill bell sounds are frequently noticed by deaf-mute children. It is also declared that noises in an adjoining room are heard. This, however, does not necessarily indicate that they hear; the knowledge may be conveyed by sensation, and it is by no means a rare occurrence for children who are very deaf to notice the moving of a chair in the next room more readily than those of normal hearing. A trustworthy sign of deafness in children is an immediate reaction to impressions of sensation while there is no response to impressions of sound. A congenital deaf-mute will always turn when the foot is stamped upon the floor, because this has been to him a signal from his earliest years, and deaf-mute malingerers can readily be unmasked if, after a number of sound tests have been made, they do not turn when this experiment is tried, but believe that they must feign not to hear the sound. Therefore the observations of parents must be very carefully tested if upon these we must base our opinion as to the faculty of hearing or the deafness of children. A valuable proof of the sense of hearing in a child is that in a lalling play it attempts to imitate the speech melody of those around it.

Let us assume that we are actually dealing with a deaf-mute child, and that the central areas for motor as well as perceptive speech are undeveloped, since these usually develop only by way of hearing. This peripheral impressive way is, however, not the only one which we control. *The second method for perception is by the eye.* This is therefore utilized to teach children the motions of speech. *The third method is by sensation,* and by this they are enabled to recognize the vibration of the vocal cords corresponding to sounds which are readily transmitted to the entire thorax, and may there be perceived by the hand (pectoral fremitus). Feeling and sight are the methods of perception which remain to the deaf-mute child, and the German method of educating deaf-mutes by these means not only promotes the complete structure of speech, but by reading the movements of speech from the mouth an almost complete perception of spoken language is attained. *Therefore the sensory speech center in the deaf-mute must be sought in the optic cortical fields of the occipital brain, while the motor speech center is most likely situated in the same area as in a person who hears.*

An illustration will show how speech is taught in deaf-mute institutions. The teacher takes one hand of the deaf-mute child and places it upon his own larynx while the other hand of the child is placed upon its own larynx. Then the teacher opens his mouth and articulates *a*. The child *sees* the opening of the mouth, it *feels* the vibration of the larynx, and, as is readily noted in deaf-mute children, its speech producing organs not being paralyzed and its vocal organs being capable of function, it imitates as nearly as possible what it sees and feels. Perhaps the vowel which first results is not a pure and resonant *a*, but by modulating the height and depth, the strength and weakness of the tone, which may be also felt, it gradually becomes purer and clearer. Now the teacher closes his lips and places the hand of the child upon the roots of his nose; he enunciates a prolonged *m*, the child feels the vibration of the walls of the nose and attempts to imitate this by placing its other hand upon its own nose. This attempt also is soon successful, and now the teacher combines the consonant with the vowel and says "*ma.*" If he repeats this syllable twice the child has learned its first word, *of course only in the form of a purely mechanical articulation*: "*Mama.*" Now the *conception* alone is wanting. This is conveyed by showing pictures which represent a mother in the family circle, for example, waiting upon the children, distributing food, etc. The intelligent deaf-mute child understands immediately what is meant by the sequence of sounds, and at once makes use of it.

It is evident that this method of building up the power of speech is difficult and circuitous but absolutely certain, and *psychologically it is the only natural way.* It has always been incomprehensible to me that this German method of educating deaf-mutes could be designated an artificial one. Certainly it is the most natural and progressive way which can be imagined.

It must be mentioned that in deaf-mute children some power of hearing may still be frequently found, particularly in congenital deaf-mutes; and it can scarcely be doubted that the utilization of this power facilitates the development of speech, so increasing the volume of sound and the purity of tone that under some circumstances the perception of speech also may be promoted. On the whole, in deaf-mute children perception will be chiefly by sight, in the

form of reading from the lips, since only in rare cases is the sense of hearing so acute that perception by the ear is practicable.

When impairment of the hearing and deafness occur earlier than the fourteenth year, the faculty of speech may be completely lost. Ordinarily it is absolutely lost if deafness occurs before the seventh or eighth year of life. If deafness or partial loss of hearing occur later, the power of speech once developed may remain more or less perfectly. If impairment of hearing or deafness appear in the adult his perception of spoken language and also his own power of speech suffers, but by no means in proportion to the loss of hearing which has developed. Persons who have become wholly deaf may, nevertheless, have the faculty of almost normal speech, and others who reveal but a moderate loss of hearing may articulate so imperfectly and so indistinctly that conversation with them is almost impossible. Their difficulty in speech does not depend upon the seat of the pathologic process. If the impairment of hearing is so extreme that in ordinary intercourse perception by the ear is insufficient, such persons must learn to read from the lips. With sufficient practice all articulate sounds which form words and sentences may be read from the movements of the lips, the cheeks, the lower jaw, and the floor of the mouth. In these persons a mere *displacement of the sensory speech center into the central cortical field of the opticus* has taken place. Whenever necessary in such cases speech production is improved *by employing the sensory tracts.* In teaching such persons by sensation the difference between height and depth, strength and weakness of tone, they are taught to exercise a control over their own speech which had previously depended almost absolutely on hearing alone.

This finishes our discussion of peripheral impressive speech disturbances. It might be supposed that loss of the sense of sight would result in impairment of speech, and in fact it has been maintained that children who are born blind learn to speak later than those in otherwise similar circumstances who see. It is also possible that a congenital or acquired deficiency of tactile and muscular sense in the organs of speech may interfere with the development of speech; for a coördinated movement of the parts is only conceivable when sensation is undisturbed, and we know that the ataxic gait of the tabetic is the consequence of disordered sensation. As has been stated these are possibilities, but I must mention for completeness' sake that they practically come into consideration as disturbances of speech only because this derangement is a secondary condition.

Among the central disturbances of speech is aphasia, the nature and forms of which will be discussed in another chapter (Wernicke). But I should like to call attention to its nomenclature, and to state that *aphasia*—that is, *speech which has been lost*—can occur only in those who formerly possessed this faculty. The inability of the deaf-mute child to speak cannot be called aphasia because it has never possessed the power of speech, and the same is true of inability to speak in a child in whom hearing has been inhibited. In these cases I shall constantly refer to mutitas, and it must be borne in mind that I previously differentiated between surdo-mutitas and audi-mutitas. There is no possibility of such a condition as congenital aphasia.

The aphasias which develop in consequence of apoplexy, embolism, etc., may be divided into two great groups, *sensory* and *motor.* In both forms

good results may be expected from exercises in speech persistently practised. What has been lost may again be acquired provided that the general intelligence of the patient has not suffered too severely from the attack, that the destruction of the center is incomplete, and that the patient is not too old.

We have frequently been content with instituting practice in speech at any time after the attack, and of course it soon became evident that success was to be expected only when the aphasia had existed for a short time. Apparently, the spontaneous improvement noted in by far the majority of cases of aphasia has either been left entirely out of consideration or not accurately estimated. If we desire to gauge correctly the value of practice in aphasia, we must try it in aphasia which has continued unchanged for a long time. From professional policy I advise that only this method of practice be instituted in such aphasias. The patients themselves will then correctly appreciate the results. Moreover, it is wise not to subject to practice too early a brain that has only recently recovered from a severe disease, or one in which the pathologic process itself has perhaps not yet been arrested.

For lip-reading in such aphasias we must necessarily begin with the elements of speech, and must first systematically practise elementary sounds. We presuppose that the patient with motor aphasia is absolutely unable to speak. We naturally begin with the simplest sounds, and, according to experience, these are the sounds in the first realm of articulation, those made with the lips and the upper teeth. We first practise on the vowels, beginning with *a* for the production of which the mouth is wide open and the voice is intonated. For the vowel *o* the lips are contracted in an oval shape; and this contraction is still more marked with the vowel *u*. For *e* the mouth is opened more widely, and in *i* still more widely. We invariably observe that the vowel *a* is the easiest for the patient with motor aphasia to enunciate, that the vowels *o* and *e* necessitate a moderate muscular contraction of the lips which is soon learned, that, on the other hand, the extreme vowels *u* and *i* require long practice. *In these exercises the patient must carefully observe the mouth of the physician who teaches him to speak.* By this means he receives *optical impressions of memory* which aid him in producing these sounds spontaneously. To heighten this effect, I urge the patients to use a mirror by means of which each may observe the phases of his own speech motions and compare them with those of his physician. It is interesting to note how the patient who has just attempted unsuccessfully to reproduce the sound of *o* which the physician has made for him is often enabled to do so immediately if he takes the mirror in his hand and imitates the position and movements he has just seen. After learning some of the vowels we proceed to the labial sounds, and here the toneless *p* which is most easily formed is the one we practise first. The lips are closed, then by a sharp expiration are parted, after which by closing the nose we sometimes see that the column of air actually passes through the mouth. The consonant which has been learned is then combined with the vowels, at first in the meaningless syllables of *pa, po, pe.* Memory for the newly acquired sounds is at first very weak, so that even two successive syllables can scarcely be repeated; frequently either the last or the first is forgotten, according to the attention which the patient has given to the movements of the vocal organs. Sometimes his ability to remember and reproduce the syllable depends upon its emphasis. By patience and perseverance we soon

teach him to speak correctly the three syllables in varying sequence. This *practice of meaningless syllables* is of the greatest advantage in strengthening the memory for speech processes. Then we take up *b*. The difficulties increase to such an extent as to try severely the patience of the learner. If the consonant is not soon clearly differentiated from *p*, it is well to postpone its practice to some subsequent period, and take up *m* instead. Here the use of the mirror alone is insufficient; besides the optical stimulation of the picture by the mouth movement, the *tactile control of the resonant column of air* which comes through the nose must be utilized. For this purpose, just as previously in the case of the deaf-mute child, the patient places his finger upon the physician's nose so that he feels the resonance of the air which comes through the nose. Even when practising the vowels, it is well to allow him also to practise this sensation control, as well as to place his hand upon his larynx.

As is obvious from the illustrations given, the teaching of the various vocal sounds presupposes on the part of the physician a thorough knowledge of the physiology of speech. Without this the results attained by his endeavors will be merely accidental and clumsy. If the physician desires to treat aphasiacs, the first prerequisite is that he thoroughly learn the physiology of speech. It is not my purpose here to describe the individual processes in the development of speech in motor aphasia from the point of view of the physiology of speech, nevertheless we must devote some attention to a few measures which assist this process.

With the exercises in articulation which have been described I combine *systematic writing exercises with the left hand*. As is well known, Broca refers the right-handedness of most persons to the action of the left brain. The congenital preponderance of the left brain results in the early use of the right hand for skilful movements. Accordingly the left brain rather than the right brain is also the central seat for the finer movements of speech. In aphasiacs who were left-handed an anatomical defect was found in the right half of the brain. Therefore, without fear of challenge, we may draw the conclusion that by practising the left hand for finer movements, for example, for writing, the movements for speech are better set in motion in the right brain. Such considerations have been utilized by numerous authors; I shall only mention Clarus, Berkhan, and especially Bernhard who points out that even with total destruction of the left speech tract compensation in the faculty of speech may result if the right half of the brain influenced either spontaneously or by exercise substitutes for the left-sided region in which the function has been lost. Consequently Bernhard believes it to be the duty of parents, even from the infancy of their children and even in healthy children, to see that they learn to use their left upper extremity as well as the right, so as to evolve from the preponderating property of the left brain a double brain condition. In all the cases of aphasia brought to me for treatment, I have instituted the systematic exercise of the left hand for writing with the exception of those cases in which, from long continued loss of power or permanent paralysis of the right hand, a greater facility with the left hand has been acquired. I was invariably able to show a distinct parallelism between the improvement in the power of articulation and improvement in writing with the left hand. A further advantage of the writing exercises is that

they assist the memory by a sequence of sounds. It may happen that, even after long practice, a word cannot be produced in front of the mirror, yet success is readily attained after a few preceding writing exercises. Therefore each process assists the other. I combine the reading exercises with those in articulation just as soon as possible by writing the individual letters upon small slates which are sought out from a large number of slates, the letters being therefore actually read, and are then placed in combination with one another. In acquiring the power of speech, writing and reading are of course to be regarded only as aids, for, otherwise, patients would regain their power of speech only by a very circuitous route.

It is remarkable that, in almost all cases of ***motor aphasia, the combination of perception and word formation*** needs particularly to be practised. The patient repeats certain words distinctly, and understands their meaning perfectly. If, however, the object is shown him he cannot name it. Therefore he is able to repeat the word, but he cannot innervate the motor speech conception from the optical picture. If the word "slate" is spoken he repeats it, and also shows that he understands it. If, however, after some time, we point to the slate and ask him to name it, he finds it impossible to do so. This peculiar psychologic condition may be explained by quoting what Störring has said in his excellent "Lectures upon Psychopathology": "In normal psychical life the perception of an object is very rarely followed by the reproduction of the corresponding name, but the hearing of a name is followed by the conception of the object designated." Störring mentions as an example that when we hear the word "horse" we much more frequently form a conception of the object—therefore reproduce mentally the picture of a horse—than we reproduce the name "horse" on seeing the object. We note that in the development of speech the first named method is the one earliest utilized by all persons. For this reason it is longest practised, and in all aphasias it is the last to be lost. From this it also follows that "in reviewing the various stages of diminished ability to develop correlations of reproduced conceptions in the centers for object pictures and sound pictures with a definite and uniform decrease of the activity of these centers, it must be that sound pictures bring to mind the object pictures, while the object pictures are powerless to recall the sound pictures" (Störring). We must, therefore, employ ***pictures*** systematically in motor aphasia. I bisect the leaves of a small book, pasting on one side one-half of the picture of an object, and writing the name of the object upon the other side. The patient must learn to combine the name with the picture just as we learn the vocabulary of a foreign language.

All that I have thus far stated relates to motor aphasia. In ***sensory aphasia*** in which perception is chiefly disturbed, we must attempt to substitute for this another mode. Exercises in hearing such as were practised by Westphal, Oppenheim and Nemann were unsuccessful. I have therefore tried, as in the case of deaf-mutes, to substitute vision as the method of perception, and have taught the patient with sensory aphasia to read from the lips. After sufficient time for practice, a control test showed in how far the patient with sensory aphasia had practised perception by the eye. I spoke to the patient by merely making the motions of speech, so that he could hear no sounds but only see the movement. This succeeded as well as could be expected, the results depending on the amount of practice in reading which the patient had.

If I turned away from the patient and spoke aloud, so that he could hear my words but could not see the movement of my lips, perception was almost as poor as at the beginning of the exercises; if I turned toward him and spoke aloud so that *he saw the movements as well as heard them,* perception was absolutely perfect. It follows from this that training of the eye enables us to utilize to better advantage the acoustic method.

French authors (Féré and Danjou) have pointed out that in motor aphasia gymnastics of the organs of speech should precede the practice of individual articulations. In fact, practice in the movements of the lips and tongue, perhaps by means of a glossodynamometer and in the form of resistance gymnastics, most effectively facilitates the production of individual sounds in the special speech exercises which are later undertaken. If this preliminary practice is instituted the desired results are more speedily attained.

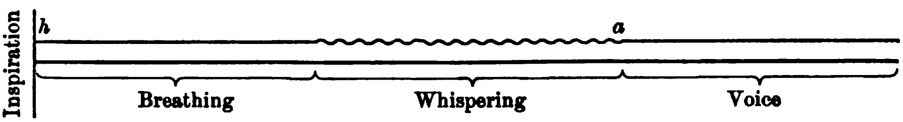
The results of this method of treatment of aphasia of course vary greatly according to the length of time the practice is continued and the patience of the sufferer. In absolute motor aphasia, as a rule, five or six months of practice is necessary before the patient can spontaneously use the words and sentences which he requires in daily life.

I must reiterate that the treatment by exercise which I have here described is not to be instituted until a spontaneous cessation of the cerebral process has been observed, or until (for example, in syphilitic cerebral processes) this has been thoroughly treated. In aphasia due to syphilis we frequently note astonishing improvement after thorough inunctions and the use of potassium iodid. Naturally this aphasia is characterized by relapses; nevertheless the treatment is simple. Exercises should be resorted to only when there is great destruction of the cortical substance or the conduction tracts. By exercises I taught several patients, in whom aphasia was unquestionably due to syphilis, and in whom no improvement followed the use of mercury and potassium iodid, to speak even after the aphasia had existed for years.

Aphasia, too, which is not merely functional, especially that of a hysterical nature, is more amenable to treatment by this method than by hypnotism or suggestion. *It is questionable whether we are justified in differentiating between "organic" and "functional" aphasias,* for we may generally assume that with a disturbance of function an organic disturbance also is present although we cannot invariably discern it. If, at the autopsy, no lesions are found in that part of the brain which controls these functions, this should never be regarded as proof of the absence of disturbance. We recognize many so-called functional disturbances of the center which we are unable to demonstrate. Apparently the functional disturbances are of milder nature than the organic, certainly this is true of the phenomena of aphasia. But this by no means disproves that they may exist unchanged for years. We recognize a voluntary mutism not only in certain forms of mental disease but also as an unpleasant symptom of psychical depression in mild, functional, disturbances of speech. Finally, we must enumerate in this category the hysterical aphasias in which treatment by exercises is decidedly valuable. In cases in which the action of the electric current is absolutely negative, patients are sometimes enabled by this purely physiologic method to regain not only the voice but also speech in a surprisingly short time.

It is especially difficult to regain the voice in hysterical aphasia, and here

the process must naturally be the same as that which we employ in *hysterical aphonias* when we desire merely to regain the voice. It is well known that electricity (feeble galvanic currents applied to the larynx) sometimes gives remarkable results; we also know that there may be a relapse of the aphonia, and that subsequently the galvanic current often loses its efficacy. In these cases I have almost always secured a permanent effect by the physiologic development of the voice. If, after an oral inspiration, expiration is practised so that we pass from breathing to whispering, and from whispering to the spoken voice, we innervate in a sequence first, in breathing, the vocal muscles or the internal thyreoarytenoids, then in whispering the lateral crico-arytenoid muscles, after which merely the action of the oblique and transverse muscles is necessary for the production of voice. The exercise is graphically portrayed in the following diagram:



In hysterical aphonia we must at first be satisfied if the patients pass from breathing to whispering, and then very cautiously attempt to use the voice. This is true not only of hysteria which produces ordinary flaccid aphonia but also of spastic aphonia. That aphonia spastica resists all external remedies is well known, and this affection has even been looked upon as incurable. Decided irritation at once produces spasm, and is therefore prohibited. Painting the larynx, the application of the electric current, and many other curative measures are unsuitable in these cases. The treatment by exercises here described produces the least possible irritation, but it may be mentioned that, if we proceed too rapidly, even this practice may cause severe spasms. I once attained a permanently good result in aphonia spastica that had existed for years. It was in the case of a soldier who, frightened from his sleep by an attack made by a comrade, took to flight, and for days wandered aimlessly hither and thither. He was absent from his regiment for fourteen days, and on returning was charged with desertion. Besides numerous other psychical symptoms of depression, *complete mutism* now gradually appeared. Every attempt at speech caused severe respiratory and vocal spasms which subsequently became general. Careful examination revealed that this was not a case of malingering, but that, probably owing to the nocturnal attack of his comrade, a traumatic psychosis had developed. The symptoms on the part of the voice were most intractable. After the disease had lasted for a year and a half, the Minister of War sent the soldier to me for treatment, and a few weeks later he was discharged, his speech being perfectly normal.

CENTRAL DISTURBANCES: STAMMERING

Unquestionably the most familiar and most prevalent central disturbance of speech is stammering. Viewed from a purely external standpoint, it represents a spastic neurosis of coördination. The spasms inhibit, hinder or interrupt speech, they occur in the three chief muscular regions which are

called into action by coördinated speech, in respiration, in voice, and in articulation. We designate it as a neurosis because we do not know its anatomical seat; that is, its pathology. That it is a central affection is obvious from the symptomatology, and whether we refer it to the accepted seat of motor speech, the third left frontal convolution, as Ssikorski does, to the cortex of the cerebrum in general, as was previously done by Schrank, or to a *congenital irritable weakness of the entire coördination of articulation* (Kussmaul), none of these theories may be regarded as absolutely acceptable. The last mentioned view, that of Kussmaul, appears to me to be the most likely, as will appear when we consider the etiology of the affection.

Etiology.—I have treated in my Clinic 569 cases of stuttering in which the etiology was carefully studied (doubtful and incomplete cases have been excluded from this review). Almost all of these cases occurred in children, and the individual data were obtained from the mothers themselves; where investigation in the family was necessary, the desired data were obtained by my assistants. In adult stutterers we are very apt to arrive at erroneous conclusions as to the etiology. An example of this is reported by Ssikorski who concluded "fright" to be the cause of stuttering in over 70 per cent. of his cases. In my cases 422 were boys, 147 were girls. The affection was found running in families in 162 cases, therefore in 28.6 per cent. Direct heredity was proven in 47 of these cases, therefore in 8.3 per cent. In this reference to direct heredity I wish it clearly understood that the stammering child did not hear its stuttering ancestor (a fact which Epstein also emphasizes); otherwise, imitation would have great significance in the etiology. We shall presently discuss this point more minutely. In 57 cases it was ascertained that the father stuttered, and in 20 of these cases the child did not know that his parent stuttered. In 16 instances the mother stuttered, but in 11 of these cases only in her earliest youth; the paternal brother stammered in 13 cases, the maternal brother in 9, an uncle of the mother in one case, a great-uncle in 2, aunts in 3, the grandfather in 11, and cousins in ten cases, brothers and sisters 115 times. Therefore, in these 162 cases, 237 people who were related stuttered. Peculiar hereditary leaps in the etiology of stuttering are best illustrated by a few genealogical trees which I here reproduce.[1]

In the first ancestral tree we note that among the grandparents the grandmother stuttered from earliest youth. She had two sons, the elder of whom stuttered in youth but not the younger. The son that stuttered married (long after he had ceased to stutter) a woman who did not stammer, and they had three sons, of whom the eldest did not stutter, but the two younger ones did. In the second case the father had stuttered in youth, and his two children, a boy and a girl, both stammered throughout life. The girl married a man who did not stutter, and from this union four non-stuttering boys were born. In the third case, the ancestral tree starts with non-stuttering parents, but the brother of the husband was a stutterer. From these parents a stuttering daughter was born who married a non-stuttering man, and they had five children, four boys and a girl, all of whom stuttered. The fourth and fifth cases may be easily understood from the diagram.

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[1] The black circles indicate "stutterers." If these are within parentheses, it indicates that stammering had previously appeared in the family.

From these illustrations it is evident that, besides heredity, there is an individual congenital predisposition to stuttering; for, in the first case, the

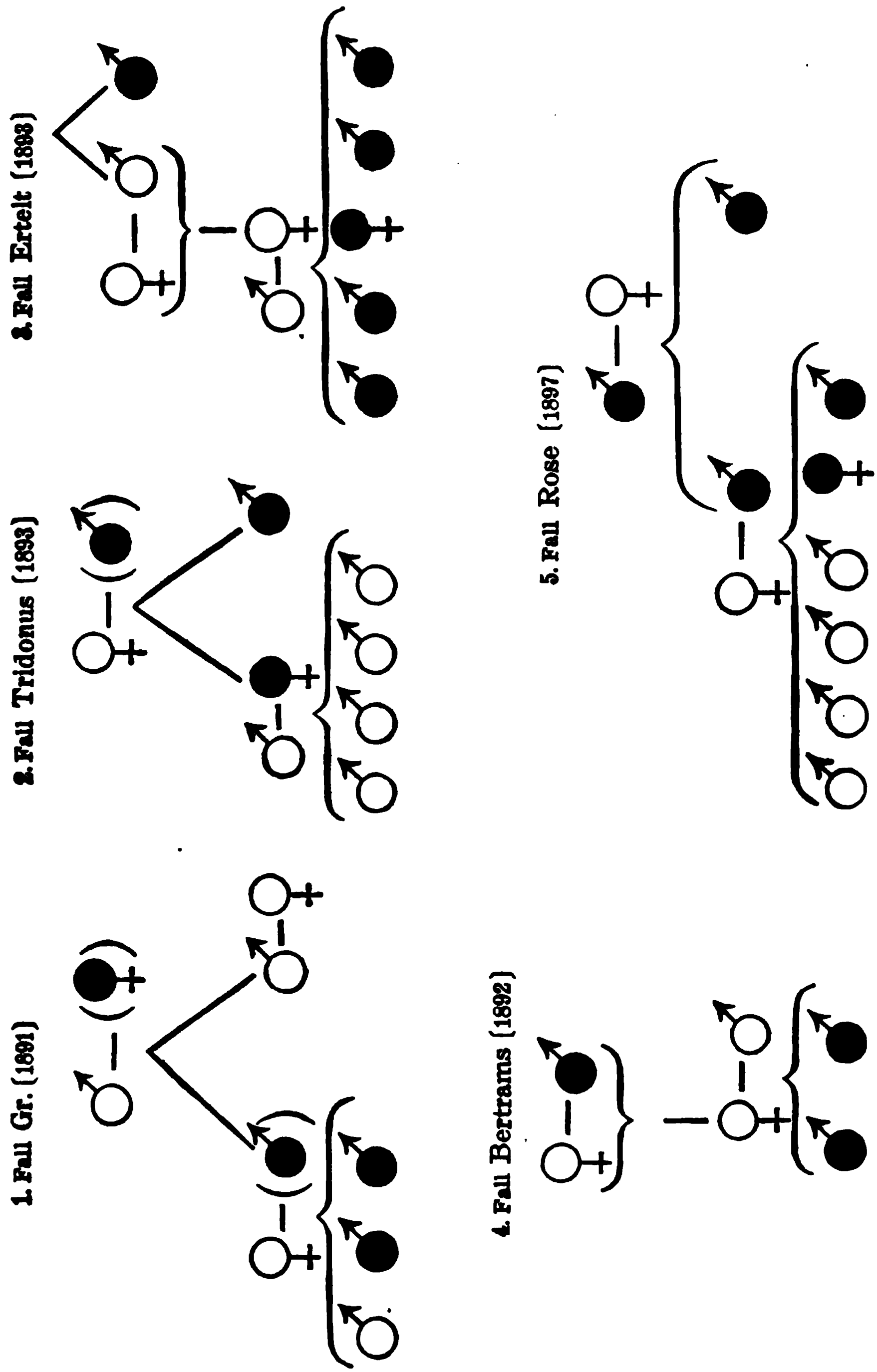
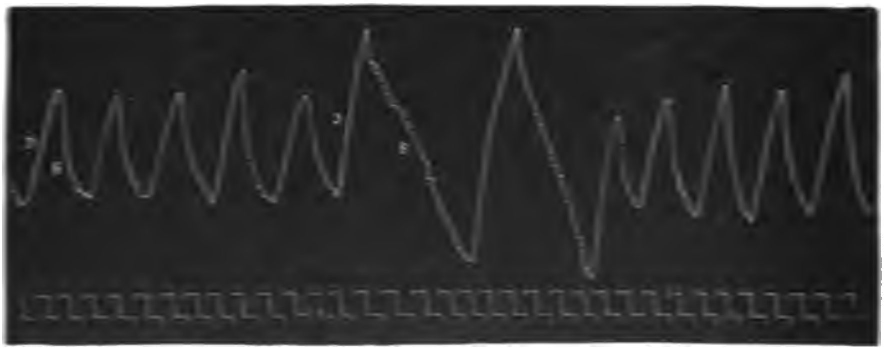


Fig. 144.

eldest son did not stutter, but the two younger did; in the second case none of the children stuttered, although the hereditary predisposition was great; in the third case the hereditary predisposition was relatively slight as only a brother of the grandfather stuttered, nevertheless all of the grandchildren stammered; in the fifth case the hereditary predisposition was marked, yet the four elder boys did not stutter, while, on the other hand, the two younger children, a girl and a boy, did. Only an individual congenital predisposition will explain this irregularity in the appearance of the affection, and the assumption of a congenital, irritable weakness (Kussmaul) affords the most plausible theory. We may the more readily adopt this view since, as we shall soon see, we find in stuttering children many symptoms which indicate a neuropathic congenital taint.

In the same category apparently belong the 26.7 per cent. of cases in which, notwithstanding closest investigation, we were unable to find the cause of the affection. Stuttering developed in earliest youth, gradually increased, and became very embarrassing, especially in the first years at school. I have pointed out in another article that in the development of the power of speech in the child there are several factors which conduce to stammering. In 10.2 per cent. these are infectious diseases, in 14 per cent. a fall or fright, and in 11 per cent. other diseases are named as causes. There can be no doubt that disturbances of speech may follow infectious diseases and brain shock; in fact, any psychical shock. Nevertheless the number of cases to be attributed to these occasional causes is relatively small, and I fear that in spite of the utmost care in the investigations which have been mentioned this is over-estimated. Imitation of a stuttering comrade or schoolfellow was expressly mentioned as the cause of stuttering in 9.5 per cent. of all cases.

Among external abnormalities which may bear some relation to stammering, I must particularly mention *adenoid vegetations*, those hyperplastic structures of the pharyngeal tonsils which have, in part, a purely mechanical



Quiet Breathing. Speech Respiration. Quiet Breathing.

FIG. 145.—Diagram showing the normal process of speech in man, taken with the Gutzmann-Oehmecke girdle pneumograph. I. inspiration. E. expiration.

and inhibitive, in part a reflex effect upon speech. We found these proliferations—and I have included only those cases in which they were large enough to protrude above the upper choanal border and thereby visibly hindered respiration—in 30.3 per cent. of the cases, that is about three times as frequently

as among normal school-children. I do not intend by this statement to maintain that adenoid vegetations are the cause of stammering, for if this were actually the case there would be more stammerers. But they secondarily cause an impediment in speech acting on a neuropathic predisposition. Such impediments occur only in those persons who are very susceptible to irritations which in normal persons would produce no reaction! We frequently observe,

Quiet Respiration. Marked Stuttering. Quiet Respiration.

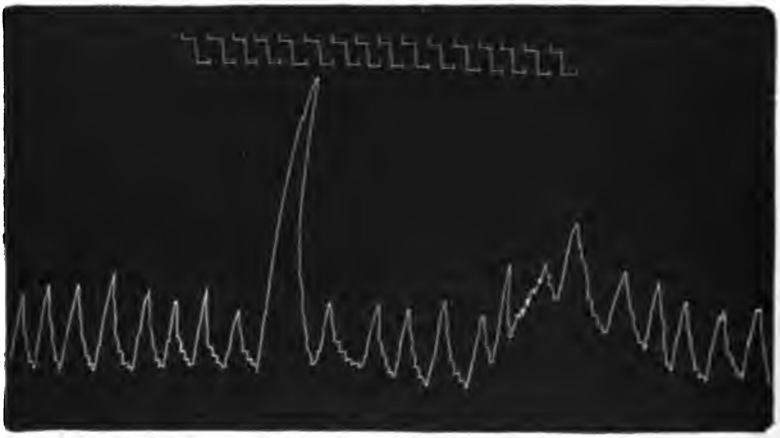
FIG. 146.—Stuttering in a girl, aged 21. Taken with the Gutzmann-Oehmecke girdle pneumograph.

and this may be done by any one who has practical experience in disturbances of speech, that an acute coryza during the course of treatment will cause a severe relapse in a stammerer. That this relapse is a reflex phenomenon is obvious from the fact that in some cases it may at once be relieved by cocainizing the nose. It is impossible for me to give a better illustration of the fact that in persons who stutter the wave of irritation is more deeply situated than in normal persons, that, accordingly, they must be regarded as nervous or neurasthenic with a neuropathic predisposition. By plugging the nose with cotton, I have in some cases succeeded in reproducing the stuttering in a few minutes. We may here observe what is *almost* a parallel between stuttering and asthma as reflex phenomena.

Symptoms.—The symptomatology of the condition may be briefly described. It consists in spasms of respiration, of the voice, and of articulation. These spasms are occasionally of such a nature that they are susceptible to and recognizable by inspection and palpation, and are at other times so slight and so transient that careful instrumental examination is necessary to demonstrate them objectively.

As the scientific study of stuttering and other spastic disturbances of speech is essentially of most recent date, I shall permit myself in this article to discuss this investigation somewhat minutely. In normal speech, inhalation through the open mouth is brief and inaudible; expiration takes place in the same way, but it is the vehicle of speech, and therefore much slower. Speech respiration differs decidedly from normal rest respiration, which, as is well known, takes places through the nose, and during which the duration of inspiration about equals the duration of expiration (I—$\frac{3}{4}$ E). These relations are best shown by employing Marey's pneumograph or similar apparatus.

In all such apparatus the principle of transmission depends upon the fact that an air capsule pressed over the epigastrium, and thus dilating the circumference, transmits this pressure by means of a rubber tube to an ordinary Marey or Engelmann sphygmograph. If it is directly transmitted, inspiration



Respiration during rest. Sighing. Rest. Stuttering. Rest.

Fig. 147.—Stuttering in a girl, aged 21. The period of time, two seconds, is indicated over the curve. The respiratory curve was taken with the Gutzmann-Oehmecke girdle pneumograph.

causes a movement of the writing lever upward, if indirectly transmitted it produces a downward motion. The curve here reproduced has been taken by a girdle pneumograph which I invented, and shows the interchange of inspiration and expiration in rest, subsequently the very distinct differentiation of this process of respiration in speech. It will be noted that speech expiration

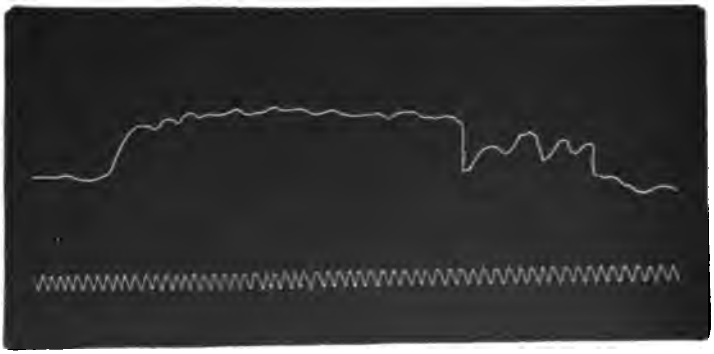


Fig. 148.—Stuttering during the enunciation of "l" and "p" in the word "lamp." The spastic movements of the floor of the mouth were taken with Marey's cardiograph. The limit of time was one-tenth of a second.

is extraordinarily full, and, compared with respiration in rest, consumes a decidedly longer time. It may also be noted that otherwise it runs quite uniformly; the slight excursions are only indications of the greater or less air pressure required to enunciate the individual sounds.

The curve of respiration in stammering is quite different. Here there are various types of faulty respiration, of which I shall mention and describe the three which are most frequent. The first form is that in which the stammerer, when he wishes to speak, expels most of the air in a single rapid expiration, and then attempts to speak with what remains. The second type is seen when speech becomes impossible because of an inspiratory spasm of the diaphragm. Very distinct clonic spasms are shown in the curve. The third type is marked by the fact that the curve at times remains upon a monotonous line, an expression of the fact that a tonic contracture of the

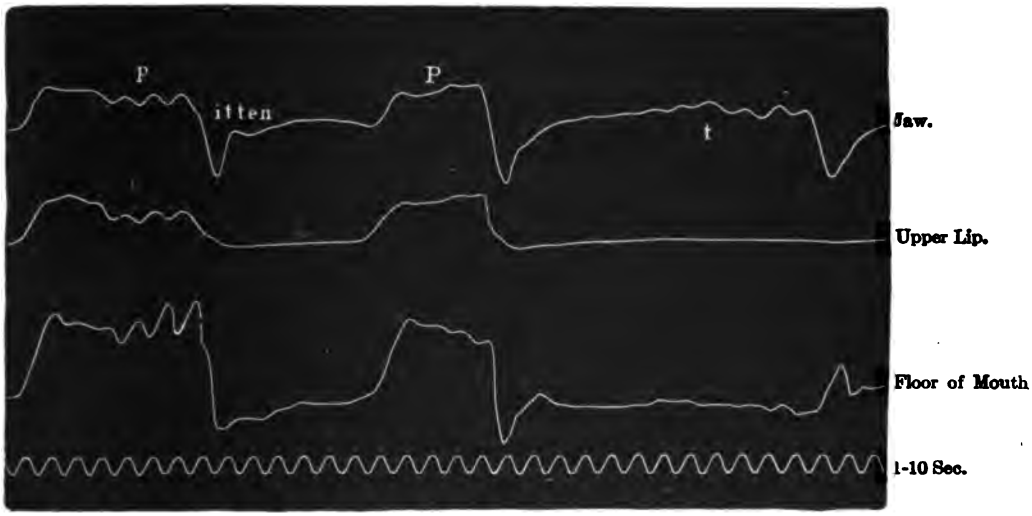


Fig. 149.—Diagram of stuttering, made with Zwaardemaker's apparatus. This curve was finished by Professor Zwaardemaker (Utrecht). The clonic spasm at "p" and again at "t" in pronouncing the word "pitten" is distinctly visible.

diaphragm results, and prevents the further course of the expiration. Between these three types we find every possible transitional stage, and not infrequently all may be combined in one and the same person.

Spasm of the organs of speech may be readily diagnosticated in the great majority of cases by auscultation, particularly by means of the phonendoscope, the rod of which may be shifted to various parts of the thyreoid cartilage and the incisura thyreoidea superior. It is rarely possible to view a spasm of the larynx by the laryngoscope. Yet some authors, and I myself, have succeeded in a few cases. Clonic or tonic spasms are then seen, in most cases spasm of the constrictors of the larynx, in a few instances of the muscles which open the larynx, and this enables us to see the position of the vocal cords which form a large triangle; the vocal cords tremble spasmodically in the vain effort to produce voice. I regard this as a distinct spasm of the posterior cricoarytenoid muscle, and I observed such a case with Th. S. Flatau. Objective pictures of these vocal spasms may be obtained by a phonautograph, although even with the most complete instruments the reproduction leaves much to be desired.

All spasms of articulation may be reproduced most readily and with great

exactness by means of Marey's cardiograph. The palette of this instrument is placed lightly upon the muscular area in question, and its motions are transmitted as usual by means of the rubber tube and the sphygmograph. The tracing shows how exactly even unusually rapid spasms and spasmodic movements are reproduced in the curves. Zwaardemaker has applied a number of these or similar capsules to the head by means of a forehead band so that the normal processes of speech may simultaneously be written in a lower

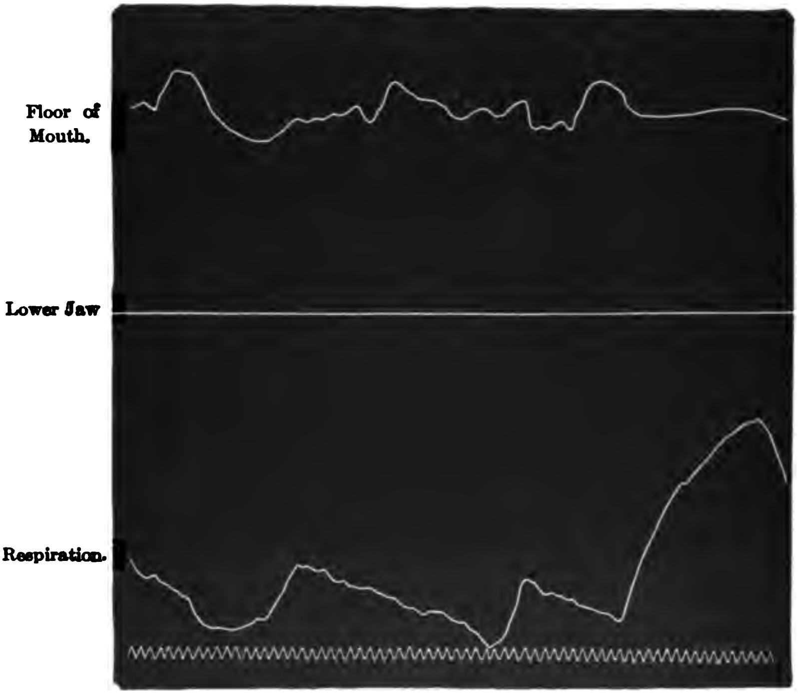


FIG. 150.—Diagram of stuttering in a patient, aged 24, taken with Zwaardemaker's apparatus. On the first attempt to pronounce the word "kakao," the patient halts, and repeats the "k" three times. On the second attempt, he does not stutter. The respiration was taken with the girdle pneumograph. While speaking the lower jaw was held in a spasm; hence the almost straight line.

jaw curve, upper lip curve, lower lip curve, and the curve of the floor of the mouth. This instrument may also be employed in the case of the stammerer, and the first curve taken by Zwaardemaker shows very characteristically the trembling spasm of the various parts. The use of Zwaardemaker's apparatus for the stammerer is somewhat difficult because its application distinctly irritates him, children and even nervous adults being afraid of the apparatus. It is therefore much more convenient, as I have found, to employ Marey's cardiograph which can be easily and correctly applied to the various areas, and just as readily be removed.

The *psychical symptoms* manifested by the stutterer may be accurately investigated objectively. It is characteristic of a neuropathic individual soon to become fatigued. This ready exhaustion, particularly after attempts to

speak, may be demonstrated with exactness in the stutterer by chronoscopic investigation such as Ziehen has employed in neurasthenics, as well as by Mosso's *ergograph.* The psychical disturbances which are revealed in the respiration of rest after stuttering should be used as objective tests for the ready psychical irritability, only it must be borne in mind that these investigations easily produce psychical irritation even in the normal person, and they must therefore be repeated if we would avoid the possibility of error.

From this brief review it is evident that we are not content to-day with the conclusion, "We are dealing with a stutterer," but we must determine to the minutest detail *the nature and course of the spastic processes* so that a special diagnosis may be made in the individual case; this was formerly never attempted but is nevertheless of immense importance in treatment. The old treatment of stuttering was a mere groping around in the dark and experimentation, while at present, by a comprehensive and careful diagnosis, we follow definite methods which lead directly to the goal.

The spasms of the stutterer previously mentioned need by no means occur in all cases and in all of the three muscle groups called into action by articulation; it may be prominent in one stutterer while in another there are only spasms of respiration, in another the action of the voice may be specially implicated, and in still another only the organs of articulation. This fact alone furnishes definite indications for treatment. The general symptoms of the stutterer and the nervousness which is almost invariably present require treatment, and experience soon shows that upon the basis of these considerations stutterers may be practically divided into three groups:

(1) Those stutterers in whom the spasms are principally due to absent-mindedness and inattention; among children these form the majority, and they may be easily discriminated by requiring the child to repeat immediately the sentence which was just stuttered, when, almost invariably, the repetition is less stuttering or it may even be fluent. In these cases the absent-mindedness and inattention to speech may almost always be referred to nervousness in the child, and this is often exaggerated and increased by adenoid vegetations or other abnormalities and disturbances of the upper air passages.

(2) The stutterers in whom a repetition of the stuttering sentence shows no amelioration but even an aggravation of the spasms. This increase of spasm upon repetition may be so great that no sound can be enunciated. In such patients attention to the speech only increases the inhibition. *The intention of the will becomes an irritant which intensifies the spasm.* Here, quite as frequently as in the first group, abnormalities and disturbances of the upper air passages augment the difficulty.

(3) The stutterers in whom stuttering has lasted so long as to produce psychical depression, a dread of speaking, a sense of inferiority in comparison with their fellows, idiosyncrasies as to definite sounds in which erroneous conceptions are such marked secondary phenomena of stuttering as to be the most prominent of all the symptoms. *In the main, I regard these psychical phenomena as secondary symptoms,* since they are usually absent in children and present in adults. That they are not a necessary result of stuttering is evident from the fact that a number of adult stutterers do not manifest them, and also from the fact that they may be associated with other disturbances of speech.

It is evident that this classification is of great therapeutic importance; for, in the first group, everything will depend upon cultivating the habit of attention in the child and removing all factors which interfere with this. In the second group, careful speech movements, beginning with the most elementary, form the only remedy with which to overcome the spasms. In the third group we must be guided by the conviction that upon concentrating the attention on normal processes of speech the spasm will under all circumstances be averted and the psychical auxiliary symptoms at once be relieved. In the third group an increase of the secondary phenomena is rarely so severe as to dominate the picture. Here we are dealing with markedly neurasthenic persons who should receive general treatment before they have any specially directed to the difficulty in speech. I once treated a young lady who, even with her first exercises in breathing, conceived the erroneous idea that I regarded everything she did as incorrect. This impression was so real that she burst into tears after the first exercise, and this crying prevented her from carrying on the breathing exercises. With every attempt there was a repetition of the tears. In such cases it is obvious that general treatment, perhaps in an institution for cold water cure, is indicated before the difficulty in speech can be especially treated.

In conclusion, it must be mentioned that in some stutterers associated movements follow these spastic symptoms, associated movements which are either reflected by the spasm to neighboring muscle areas, such as contortions of the face, closing the eyes, nodding of the head, and the like, or from the effort to overcome the difficulty they may occur in any part of the body, such as stamping, knocking with the fists, even jumping and the like. These constrained movements also must, of course, be regarded as secondary symptoms.

Treatment.—In passing to the treatment of stuttering I must remark from the onset that with the general disturbances almost invariably present, which primarily appear as nervous phenomena, a *constitutional treatment* of the stutterer, a careful regulation of his entire mode of life is an absolute prerequisite, provided we wish the cure to be permanent and not to be constantly surprised by relapses. Such a general treatment and regulation of the mode of life is naturally only possible in clinical treatment, and even then only when this clinical treatment is carried out in somewhat of a domestic manner. I have previously called attention to the extraordinary importance of proper diet in the treatment of disturbances of speech. General bodily exercise, abundance of fresh air, bathing of the entire body both morning and evening, sufficient sleep, plentiful but not too monotonous nutrition, are the important factors. That this mode of treatment fell into desuetude after the earlier clinicians had clearly recognized its utility can be due only to the fact that the treatment of disturbances of speech has recently been taken from the hands of the physician and entrusted to quacks and empirics. Any one who has made even a few clinical observations of stuttering children will surely come to the conclusion that the treatment of the stuttering child should be in professional hands, and whenever the family physician notes the development of this fault he should, above all things, regulate the child's general nutrition and mode of life. I have observed numerous instances in which such a difficulty in speech has been favorably influenced or even removed chiefly or solely by such general treatment.

We pass from these preliminary remarks to the *special treatment* of stuttering, which has been based on physiologic premises by no lesser authorities than Johannes Müller and Dubois-Reymond. Dubois-Reymond points out the fact that our bodily exercises are not only muscle, but also nerve, gymnastics. The normal muscle promptly obeys the nerve; the condition of contraction in the body present at any moment is determined by the state of stimulation of the nerves in the preceding moment. Since the nerves transmit only the impulses coming from the motor ganglion cells, it is obvious that the actual mechanism of coördinated movement has its seat in the central nervous system, and consequently practice in such movements is in the main merely an exercise of the central nervous system. This possesses the inestimable advantage that a series of movements which frequently conform to a certain law are readily repeated in the same order, increasing and decreasing and merging into each other as soon as an impulse of the will calls them forth (Dubois-Reymond's Lecture, "Ueber die Uebung"). The application of this law to our case is the following: If the normal respiratory movements, the normal movements of the voice, and the normal movements of articulation, carefully proceeding from the more simple to the more difficult, are systematically practised, and practised so long that they are made without attention to rules, therefore involuntarily, normal speech is the result, and stuttering is cured. Hence there can be but one rational treatment of stuttering, and this consists in practising the necessary movements of speech, by a physiologic method. By this mode we will observe that constrained movements are suppressed.

Opinions vary as to the adaptation and development of this treatment in the individual case. Consequently there are numerous methods, many of which may ultimately lead to satisfactory results, but all must be based upon this rational therapeutic principle. The best model for the corrective exercise in stuttering is the person who speaks perfectly. Respiration must be practised until the previously indicated respiration curves of normal speech are acquired; the voice must be trained by such elementary exercises as I have advised for aphonia spastica until spasms no longer appear, and articulation exercises for the most elementary movements, combined with the use of the eye and sensation, must be practised before a glass as long as any spasms occur.

It would lead us too far afield to explain in this article how the principles of the treatment are to be carried out. But I may refer the reader to the book published by Albert Gutzmann, to my lectures "Speech Disturbances" (1892), and to the monograph upon stuttering which I published in 1898.

PERIPHERAL EXPRESSIVE DISTURBANCES

We shall now consider briefly *peripheral expressive disorders of speech.* Here also we differentiate between functional and organic disorders, and then review the individual disturbances. I prefer, however, to discuss a few of these peripheral expressive disorders of speech, and at the same time to illustrate the entire group of these affections. It is clear that disturbances of respiration and of the voice which we find in diseases of these organs may also influence the speech, but in such cases the disease which affects the organs so

engrosses the professional interest that the disorder of the speech is regarded as merely secondary. I must, however, briefly mention an exception. This is the disturbance of speech which develops during *puberty,* and which may permanently impair the voice, and also the speech. It sometimes happens that the *falsetto voice* persists beyond the period of puberty, in which case we find grown persons with a ludicrously high voice who are mortified by their speech, and spare no pains to attain the normal male voice. Here electricity and local remedies are of little avail, but a satisfactory result may almost always be readily attained by the method, previously described, of transitions from breathing to whispering, and finally to the speaking voice. If, at the same time, mild lateral pressure is exerted upon the larynx, the patient, as a rule, is soon able to enunciate a deep wavy tone, and with subsequent practice a strong chest tone is developed. By holding the tone as long as possible, individual vowels and combinations of syllables are practised, and we soon pass to reading, after which it is only necessary for the patient to use this newly gained power of speech in his daily intercourse. Patients are best treated in an institution because here they most readily lose their diffidence with their newly acquired proficiency in speech.

Among peripheral expressive disorders of the organs of articulation, I must mention congenital and acquired paralysis, the cleft palate, inhibitions which result from a faulty formation of the tongue, and an abnormal position of the teeth and their influence upon speech. Defects of the tongue are rarely the cause of disturbances of speech, and it is remarkable that this fact so long failed to be recognized; it was apparently due to the synonymous use of the expressions, *tongue* and *speech.* From most remote antiquity the tongue has been regarded as the seat of speech. This view may be attributed to Aristotle, who not only expressed it in simple form, but referred all errors in speech to the tongue, either because it was too broad, too short, or not sufficiently motile. In at least many cases of disturbance of speech careful clinical observation would incline us to another view, and we note that even before Aristotle old Father Hippocrates held a much more correct theory of disturbances of speech. He says: "Indistinctness of speech is caused either by disease or by defective hearing, or because, before a thought is expressed, other thoughts arise, before words are spoken other words are formed, etc." How little speech is influenced by the loss of a large part of the tongue under operation is well known to the modern surgeon. In former times this would have been regarded as a miracle. Wonderful was considered the speech of the so-called African Disciple of Tipasa, who had his tongue cut out by the vandal Huneric in the year 484, yet, notwithstanding this deficiency, was able to proclaim aloud his belief in Christ. Twistleton in his little book, "The Tongue Not Essential to Speech," London, 1873, has shown that this phenomenon need by no means be regarded as a miracle. Nevertheless it is certain that an imperfect tongue cannot enunciate all sounds distinctly, and that a faulty expression, that is *stammering,* must be the result of such a radical operation. Even partial excisions of the tongue may result in faulty speech. To illustrate, I saw a patient who was successfully operated upon for cancer of the tongue, but after the operation a faulty articulation of the *s* sounds appeared, and was such an impediment that the patient thought of giving up his occupation. His family physician (Dr. Peltesohn) advised him to

practice exercises, and after much labor he succeeded in producing the *s* sounds perfectly. The faulty speech was due to the fact that the lateral excision of the tongue had drawn the tip of the tongue completely over toward the affected side, and had entirely changed the sensation for the position of the parts in expression. The patient articulated all the sounds of speech which are formed with the tip of the tongue or the areas in its vicinity with the side upon which excision had been performed. This produced, particularly on articulating *s*, a very disagreeable sound accompanied by the spurting of saliva, which in fact rendered conversation with him most unpleasant. The exercises undertaken were of such a nature that first of all by means of probes a new localization was produced for the points of articulation which, instead of originating at the tip of the tongue, were now formed upon the healthy side, at the point where the tip was formerly situated.

Among numerous *defects in expression* are the different forms of lisping (sigmatism) which are very prevalent. The difficulty is relieved by practice in sounds which correct the position of the tongue.[1] In one form of this sigmatism, the so-called lateral lisping, we almost invariably find an abnormal position of the teeth which probably causes the development of the disorder. If the alveolar border of the jaw is too small compared with the number of teeth, the teeth in their growth must overlap in such a manner that vertical or horizontal arches develop. Hence if the two jaws articulate upon one another at the point where the arch is situated an opening forms which apparently predisposes the person to direct a column of air to this point. As these dental arches are almost invariably found in lateral lisping—I found them in 90.3 per cent. of all cases—it is a mooted question whether these are not the actual causes of the difficulty. I shall briefly report a case which bears upon this question.

The four young daughters of a family were sent to me for examination on account of the same defect in speech, lateral lisping; I was told that the eldest child had acquired this from a wet-nurse, and although this wet-nurse was at once sent away she subsequently became the nurse of the younger daughters of the family who had contracted the faulty mode of speech from their elder sister. The parents as well as the grandparents spoke normally. In all four children I found lateral dental arches, and also found the same in the parents and grandparents, who apparently had no incentive to use the dental arches in this way.

This demonstrates that dental arches are not a *direct* cause of this form of faulty speech but only a *predisposing cause.* The same conditions which may be produced by a dental arch may be also developed by a tooth projecting posteriorly from a row of teeth, since it presses upon the tongue at this area, and causes a deviation of the column of air which normally passes along the middle of the lower row of teeth. We may see from casts of the jaw how variable are these dental arches. Where two dental arches exist, bilateral lisping may occur or the lisping may be only on the side where the larger of the two dental arches is found.

It might be supposed that in order to relieve the difficulty these dental arches should be remedied, but that the fault cannot be overcome in this way is evident from the causal relation which has been mentioned. A person

[1] Details will be found in my lectures upon "Disturbances of Speech," Berlin, 1893.

may become a lateral lisper without such a dental arch, and the dental arch itself is by no means the only cause of lateral lisping. I therefore believe the remedy proposed by Berkhan to be unnecessary; he advised that a tooth be drawn so as to give more space, and in this ray to widen the arch. If we train the tongue to a correct position by treatment with sounds, and in this way teach the patient to close the arch and direct the column of air to the middle of the lower row of teeth, we relieve lisping without a special change in the dental arch becoming necessary. [Good dental practice, everywhere available in the United States, readily corrects deflections of the dental arcades, and should always be enlisted in the management of these cases.—ED.]

I shall close my description with this brief review of peripheral expressive disturbances of speech. Of course it is impossible within the limits of a short article to consider the entire range of disturbances of speech. *From this review, however, it will be observed that these disturbances of speech have lately been restored to the domain of the physician, and the cultivation of this field constitutes for him a worthy and beneficent task which he may facilitate with all modern methods, and thus bring about a permanent and gratifying success which is impossible and will remain impracticable for the empiric and the quack.*

NEOPLASMS OF THE SPINAL CORD AND OF ITS MEMBRANES

By FR. SCHULTZE, Bonn

While tumors of the brain are much more common than those which affect the membranes of the brain, tumors of the spinal cord are less frequently observed than those of its membranes. On account of their practical importance we shall chiefly consider the latter, neoplasms of the spinal cord having merely an anatomical, although important, diagnostic interest.

VARIETIES OF NEOPLASMS

On briefly reviewing the *intramedullary* tumors, *tubercle* which may occur either singly or be multiple is comparatively frequent, with or without a simultaneous affection of the membranes, and preferably attacks the enlargements of the spinal cord. *Gummatous tumors* are more rare, and are usually combined with like changes in the meninges.

Gliomata and "*gliosarcomata*," which are either circumscribed or may extend throughout the entire spinal cord, even to the medulla oblongata, are frequently observed. *Sarcomata* also are numerous. Rarely do they attack the substance of the cord alone; their seat and extension vary greatly, and in size they range from that which is microscopic to an extent which involves the entire spinal cord. Often they are *metastatic*, solitary, or multiple, or they may proliferate into the medulla spinalis from without. Furthermore, there is a *multiple sarcomatosis* in which nodules simultaneously develop in the membranes of the central organs.

In isolated cases, *angiomata, cholesteatomata,* and *cysts* have also been noted in the spinal cord. Primary *carcinomata* of the spinal cord are unknown, and secondary ones with no direct proliferation from without are exceedingly rare.

External to the cord, therefore *extramedullary,* we must first mention those tumors which originate from the *nerve roots*; above all, the *multiple* fibromata or neurofibromata which are frequently merely a partial phenomenon of general neurofibromatosis of the nervous system, and are most often found in the nerves of the cauda equina. Although usually small, about the size of a hemp-seed, they may become so large as to produce pressure symptoms. *Sarcomata* may also grow externally from the plexus into the vertebral canal. *Gummatous tumors* are also observed.

Much more frequently the *meninges of the spinal cord* are attacked with *primary neoplasms* which develop within them.

We chiefly observe *circumscribed tumors* which originate either from the dura, or from the pia, or, now and then, even from the arachnoid. These are usually either *fibromata, fibrosarcomata* or *myxomata,* while lipomata, adenosarcomata, lymphangiomata, and teratomata are very rare. More common than the last named are *psammomata,* which are difficult to differentiate from *endotheliomata,* but which may also be multiple. *Sarcomata* of the meninges either press upon the spinal cord, or proliferate into it, or they develop simultaneously in the cord and in the membranes; they are rarely multiple. They may be primary or secondary; the latter in rare cases also in the form of multiple *melanosarcomatosis.* It is a significant fact that a quite *diffuse sarcomatosis* of the meninges may develop, which permeates their entire length, and decidedly thickens them. Tumor-like, circumscribed, tubercular foci and gummatous swellings may also be produced. Now and then *echinococci* are found, and, in about one-fifth as many cases (according to Schlesinger), *cysticerci.*

These tumors vary greatly in size. Isolated tumors are chiefly found opposite the dorsal cord. According to pathologico-anatomical statistics, they are more frequently intradural than extradural.

I shall not here enter into details, since we are mainly interested in the clinical phenomena of these tumors and their treatment, the paramount question for us being, How can we diagnosticate circumscribed extramedullary tumors?

SYMPTOMATOLOGY

The following is the history of a young man, aged 24, who reported that as a child he had frequently suffered from tonsillitis as well as the various acute exanthemata, but from the time of puberty he had been perfectly well. Syphilis, gonorrhea, and the abuse of alcohol were absolutely denied. There had been no trauma. About six months prior to his coming to the hospital he first felt a *slight pain* low down in the back and to the *left of the vertebral column.* At first this pain occurred upon bending his body, and on rising from bed in the morning. A month later he felt it while lying down, and subsequently even when sitting. Coughing and sneezing greatly increased it. About three or four months after it first appeared, he experienced an abnormal sensation in the *left leg* which he designated as "dull," later it was accompanied by formication. Soon *uncertainty* and a *feeling of exhaustion* appeared while walking, and the pains in the back decreased, but were still very severe upon sneezing and coughing. A few weeks after the appearance of these abnormal sensations in the left leg there was also paresthesia in the right; the *urine* could not be voided with the same force as formerly.

This history shows as the most characteristic factor a *progressive disturbance,* chiefly manifested by an increasing weakness of the legs after pain in the back of varying intensity, and of regressive nature. The examination of the powerful young man showed, briefly, a *spastic paresis* of the legs which was more marked on the left than on the right side, a somewhat *uncertain and ataxic gait,* and *pathologically increased tendon reflexes.* The abdominal reflex could be evoked only in the upper portion of the belly. *Tactile sensibility,* as well as sensitiveness to pain, was diminished in the legs, also up to a certain height upon the trunk, while the temperature sense was but little disturbed. There were no other symptoms. The functions of the brain and of the cranial nerves were intact. Above all, there was neither nystagmus, anomaly of the pupil, nor any ophthalmoscopic change. No symptoms were referable to the arms.

Since hysteria and other functional neuroses as well as polyneuritis may be excluded, a *spinal affection* will naturally be thought of, above all *myelitis* or *meningomyelitis dorsalis.* The onset of pain, the early sensory disturbances, the uniformly

progressive course of the disease, and the absence of ocular symptoms as well as increased reflexes in the arms, preclude multiple sclerosis; and since the ordinary causes or auxiliary factors of myelitis and meningomyelitis are absent, such as syphilis, infectious diseases, exposure to cold, and over-exertion, we must, as in similar cases, consider *compression of the spinal cord,* for progressive myelitis without multiple sclerosis or syphilis is exceedingly rare.

This compression could originate only from the *vertebral column,* and here, above all, tuberculous disease might be assumed. Upon accurate investigation, however, we find a somewhat marked lateral deviation in the dorsal portion, but neither Potts' kyphosis nor a lateral deviation of individual vertebræ. But little importance is to be attached to the moderate prominence of some of the vertebral processes, provided that simultaneously no motor disturbance of the vertebral column or demonstrable sensitiveness to pressure appears in these abnormally situated vertebræ. After investigating many normal vertebral columns, we shall be convinced that projection of the vertebræ posteriorly has no special significance, but *that the same or similar irregularity is by no means rare in normal persons.*

After numerous examinations of our patient, it was noticed that pressure upon a definite point in the vertebral column, somewhat to the side of the median line, invariably *produced intense pain,* although the vertebral column could be easily moved in all directions, but not without some pain. The sensibility of the different vertebræ to pressure may be tested in various ways. I have found it advisable to make pressure with the posterior surface of the index finger, or with the second finger much flexed at the first phalangeal joint. This leads to more accurate results than if we make pressure with the tip of the finger or with the fist.

When we recall the various pathologic processes in the vertebræ which may lead to compression of the nerve roots or of the spinal cord, everything is opposed to the view of tuberculosis: the absence of hereditary predisposition in this robust young man, the negative findings, the free movability of the vertebral column, the absence of fever and emaciation. Since a carcinoma of the vertebræ could not be assumed, we could consider only those rare tumors of the vertebral column which are occasionally observed as of primary origin; for secondary neoplasms there was no proof of a primary and pathologic focus. These rare tumors are *sarcoma* and *myeloma* (the latter, however, being usually multiple), even *osteoma, chondroma,* and *angioma.* Primary sarcoma, however, usually produces alterations in the form of the vertebral column before it causes symptoms of compression. *Myeloma* is multiple in other bones, and, aside from other pathologic symptoms, not rarely leads to albumosuria, which was absent in our patient. *Exostoses* and *osteoma* develop more slowly, and during their growth toward the spinal cord do not produce symptoms so rapidly as was the case in this instance.

Enchondromata, which also grow very slowly, are so rare that we should rather consider instead the presence of one of those extra- or intradural tumors of which I spoke in the introduction, particularly as the X-ray examination revealed nothing anomalous in the bones of the vertebræ. *Intramedullary* tumors, however, cannot be primarily diagnosticated because they do not set in with a neuralgic, prodromal stage, and because as a rule they early lead to partial sensory paralysis. Sometimes they may accompany chronic meningitis, and pain will be produced; in compression of the spinal cord from without, the various forms of sensation need not be uniformly implicated.

This much is certain,—that other authors as well as I have usually found an extramedullary tumor in cases similar to the one described. We should ascertain its exact position and size, since its relief is only possible by operation, and it is our duty to inform the surgeon as accurately as possible where it is located.

Besides pain on pressure, the localization of which must be accurately determined by frequent tests, we must also ascertain the upper boundary of the tumor by correctly outlining the upper limit of hypesthesia and hyperesthesia, no matter how slight.

We assume that the patient to be examined is capable of accurate responses, which is generally the case; we must not regard the limit to be that point above which there is no perceptible anesthesia, but that in which there is a

decreased sense of contact, in distinct contrast to the other normal cutaneous areas. Sometimes we find in this line another area in which there is hypersensitiveness to cutaneous irritation; upon minute investigation this was found in our case.

We should also endeavor to ascertain whether, under observation for weeks or months, this limit remains at about the same height, whether it rapidly rises, or whether it shows any variations whatever. It is manifest that, in tumors of slight extent which increase chiefly in thickness, the intensity of the hypesthesia will be chiefly increased, but its extension upward in only a slight degree. When tumors rapidly grow upward, the anesthesia they cause must also rapidly ascend; but these are much more rare. In our case the determination of this upper limit of hypesthesia indicated that it chiefly existed in those striated portions of the skin covering the trunk which belong to a segment of the spinal cord, and opposite to which the vertebral column was especially sensitive to external pressure. For instance, there was hypesthesia in the cutaneous area of the 10th dorsal segment, and hyperalgesia in that of the 9th. The 10th spinal cord segment is opposite the 9th thoracic vertebra, and this is opposite the 8th spinous process, and just between the 8th and 9th sensitiveness to pressure was most distinct. The upper border of the tumor probably extended somewhat higher than this, for there was also hyperalgesia in the area of the 9th segment.

It is more difficult to determine the lower boundary of the tumor. If the lumbar enlargement be exposed to pressure, the tendon and cutaneous reflexes of the legs are at least in part absent, and somewhat marked muscular atrophy, possibly with DeR, may be present. How many dorsal segments are implicated cannot be determined, because the abdominal reflexes aid but little in the investigation. The upper, the middle, and the lower may be absent even when the tumor lies above the dorsal segment; since in our patient some of the abdominal reflexes were present, particularly the upper ones, it seemed likely that the lower dorsal segments were implicated.

As the abdominal muscles reacted normally to electricity, were not atrophic, and were still innervated, a tumor projecting downward was excluded. Simple paralysis might, of course, exist from pressure upon the spinal cord high up.

Experience has shown that, in cases quite similar to ours, a circumscribed tumor existed which could be removed by operation. Unfortunately, we are at present unable to determine whether the tumor is extradural or intradural, or, above all, whether or not it can be removed; this depends upon the possibility of adhesions, upon the relation of the tumor to the spinal cord, and particularly upon the fact whether the tumor has extended anteriorly or posteriorly. We are forced to suggest operation to the patient because we know of no other curative measure, and the sequels of complete compression of the spinal cord would cause death. Prior to operation it is always advisable to try antisyphilitic treatment, as we cannot absolutely exclude syphilis occulta, particularly in the early stages of spinal disease.

DIFFERENTIAL DIAGNOSIS

The diagnosis of circumscribed **extramedullary tumors** of the spinal cord therefore essentially depends upon the existence of a gradually progressive

motor and sensory spinal paralysis, which, in spite of increasing intensity, extends upward very slightly, at most about one segment. This paralysis is usually preceded by a neuralgic prodromal stage of varying duration, in which the seat of pain generally corresponds to the uppermost limit of the paralytic phenomena. Not infrequently local sensitiveness to pressure in the vertebral column opposite the affected spinal cord segment is revealed by minute investigation. Occasionally, as is proved by my own experience, there is neither local pain on pressure nor radiating neuralgia; nevertheless, on account of the progressive paralysis, we must make a diagnosis of tumor.

In a differentio-diagnostic respect, as I mentioned in the history of my patient, we must first consider *meningomyelitis—more rarely myelitis.* With the former we must include *chronic pachymeningitis,* which, owing to the pressure of its exudate upon the spinal cord, as well as to the propagation of the inflammation, may produce the same symptoms.

Many years ago (1869) Charcot described *pachymeningitis hypertrophica cervicalis,* in which, as a rule, the same symptoms are observed as in circumscribed tumor of the membranes of the spinal cord. Thickening of the dura is a form of tumor which produces the same effects as that described at the beginning of this article. But pachymeningitis usually runs a much longer course than most tumors of the membranes of the spinal cord; it may last for two decades, and in these cases it is usually an accompaniment of syphilis. In hypertrophic pachymeningitis the cervical portion of the cord is commonly attacked, while most tumors are found in the dorsal cord. I have seen them in the meninges of the dorsal cord; and they have frequently been noted opposite the cauda equina and the lumbar portion of the spinal cord.

Ordinary meningomyelitis can readily be excluded, since, as a rule, it shows a progressive course only when it develops upon a syphilitic basis. Usually there are simultaneously other symptoms on the part of the cerebral nerves, of the meninges, and of the brain. Even when these are absent, the spinal symptoms are not so uniformly and gradually aggravated; above all, they are not so constantly limited in their upward course; finally they not infrequently respond to syphilitic treatment.

In *pure myelitis* there is usually no pain; this symptom, it is true, may now and then be lacking in tumor. After a more or less rapid onset, the intensity of the pain becomes stationary; in many cases its causes are obvious.

Multiple sclerosis, at least during a certain stage of the disease, may occasionally resemble transverse myelitis; but during its course the uniform sensory disturbances developing from compression, and distributed upon the skin of the trunk from below up to a certain height, are absent; in sclerosis the localization and periodicity of hypesthesia vary greatly. The ordinary picture of multiple sclerosis differs so greatly that no differentio-diagnostic confusion is possible.

The differentiation from **intramedullary tumors**, with or without syringomyelia, not to speak of the rare cases which are combined with intramedullary or extramedullary tumors, is more difficult and sometimes impossible.

In intramedullary tumors there is usually no preceding stage of radiating pain, particularly at the onset, while partial sensory paralyses are often observed, especially when a glioma, or gliomatosis, simultaneously causes central cavity formation.

But dissociated sensory paralysis may now and then be the result of extraspinal tumors—Brown-Séquard's semi-lesion may be noted with extramedullary and intramedullary tumors; in spinal tumors there may be radiating pain if chronic meningitis has simultaneously formed; or, for unknown reasons, even without this, probably because of intraspinal irritation of the sensory pain tracts before their destruction. Why there is not always such an irritation, and why inversely, and in exceptional cases, there is no pain in spite of the pressure of an extraspinal tumor upon the extraspinal portion of the sensory nerve roots, we do not know.

Although the differential diagnosis is thus rendered perplexing, it is somewhat facilitated by the fact that extramedullary circumscribed tumors are vastly more numerous than intramedullary ones. The most common form of the latter, tubercle, occurs in patients who reveal tuberculosis of other organs.

Syringomyelia may be differentiated from circumscribed tumors in that the latter, according to their position, produce symptoms referable to a central and elongated focus, that they more rapidly spread upward, perhaps to the medulla, and that much more circumscribed muscular atrophies are observed, even when large muscles are involved. After a long time, ordinary syringomyelia without gliomata will usually produce terminal symptoms of spinal paralysis just as severe as those of extramedullary tumor.

I have already referred to the differentiation from diseases *of the vertebræ* and from tumors. It may be added that in most of these affections there is generally greater rigidity of the vertebral column than in tumors of the spinal meninges. It was astonishing to note that one of our patients, the upper portion of whose medulla spinalis was compressed by a tumor the size of a walnut, complained of stiffness in the nape only at the beginning of the disease, while active and passive movability of the cervical vertebral column continued to be normal for a year afterward, and until violent spastic muscular spasms at last appeared. In tumors of the sacral and lowest lumbar regions we should always palpate the bones per rectum, especially since in this region X-rays do not aid us. In my case X-ray examination revealed nothing abnormal.

THE SEAT, EXTENSION AND COMPOSITION

Passing to our case, and to the diagnosis of the seat, extent, and composition of tumors of the spinal meninges, the localization of certain irritative phenomena may give light.

THE SEAT

In a certain number of cases [illegible] may be detected at [illegible] as a [illegible] that side [illegible] cases. [illegible]

[illegible] are sensi-

tive to pressure. This symptom has the same significance as local sensitiveness to pressure, which, if it appear on active movements of the vertebral column, is accompanied by pain in the same area.

If *extensive* sensitiveness to pressure is demonstrated, we cannot at once assume the existence of a tumor. In the first place there may be chronic meningitis above or below the tumor; secondly, increased pressure of the cerebrospinal fluid may perhaps render the sensory nerves more sensitive; finally, in addition to hyperesthesia of the skin there may be a purely functional hysterical disturbance. The *second* irritative phenomenon is *circumscribed neuralgia*. If combined with local sensitiveness of the vertebral column, neuralgic pain is of particular import when it is seated within the nerves belonging to those segments of the spinal cord which, according to our present knowledge, lie opposite the sensitive spinous processes and the vertebral bodies.

Very significant, too, are the *hyperesthetic* zones which may extend above the hypesthetic ones. Since these are often absent we must depend upon the *phenomena of absence of function, sensory* as well as *motor*. We must ascertain accurately by various methods of examination how far upward these phenomena extend; this will determine the upper border of the tumor.

The localization of the *motor* symptoms of absence of function aids us in locating the tumor in *enlargements* of the cord and of the *cauda equina* (of course when combined with sensory disturbances), while the sensory symptoms form a valuable indication if the tumor is situated in the *dorsal cord*. Among the motor symptoms we may note the possible disappearance or decrease of the various reflexes. Sometimes there are trophic disturbances. It must be admitted that at the present time the exact localization of tumor at the height of the lumbar enlargement and in the cauda equina occasions much perplexity.

By the combined researches of English, German, and American investigators, it has been quite accurately determined which individual segments of the spinal cord are connected with individual muscular and cutaneous regions, and the results of these investigations have been compiled in the form of tables, of which I shall mention particularly those of Sherrington, Head, Starr, Edinger, Kocher, Wichmann, and Seiffer.

THE EXTENSION

In these it was demonstrated that muscle nerves do not all come from one segment, nor are they all supplied by one root of the cord, but by several; in the same way the individual cutaneous areas are supplied by two, three, or even four segments situated one above the other, and by their nerve roots, so that the destruction of a single sensory nerve root causes but little disturbance. In the diagnosis we may therefore assume that the upper end of a tumor will be found opposite those segments of the spinal cord the cutaneous areas or muscles of which *show disease*; and, in regard to sensation, those segments in the area of which hyperesthesia can be detected.

To explain this segment innervation and its apparently remarkable relation to the muscles and skin, Edinger[1] compiled a diagram which is very

[1] "Neue Darstellung der Segmentinnervation des menschlichen Körpers." (*Zeitschr. f. klin. Medicin*, Bd. LIII.)

CERV. I.	II.	III.	IV.	V.	VI.	VII.	VIII.	DORS. I.	II.	VII.	VIII.	XII.	LUMB. I.	II.	III.	IV.	V.	SACR. I.	II.	III.	IV.	V.

Long and short muscles of the nape, muscles of the vertebral column. ......Long muscles of the vertebral column......

Splenius capitis. .....Splenius cervicis...

Longus capitis. Longus colli.

...Obliquus abdominis..........

All small muscles between the skull, diaphragm and vertebral column.

.... Scalenus medius. ....

Scalenus anterior.

Scalenus posterior.

. Scalenus minimus..

..Rectus abdominis..

...Transversus abdom...

Muscles of the chin and hyoid bone.

.....Pectoralis major......

...Pectoralis minor...

.....Psoas, Iliacus.....

..Quadratus lumborum..

Sphincters. Peri-neum.

INTERCOSTAL MUSCULATURE

Levator scapulæ.
Rhomboidei.
Subclavius.
Supra infraspinatus.
Subscapularis.
Teres major et minor.
Serratus anterior.
Deltoideus..
Brachialis.
Biceps brachii..
Triceps.
Coracobrachialis.
Supinator brevis et longus... .
Brachio radialis.
Pronator teres.
Long extensors and flexors of the hand and the finger.
Flexor carpi and ulnaris.
Muscles of the ball of the little finger.....
Lumbricales.
Interossei...

Small pelvic muscles.
Adductores fem.
Pectineus...
Sartorius..
Vasti fem..
Patella reflex.
Musc. rectus femoris...
Obturator.
Tensor fasciæ.
Musc. glutæi.
All posterior muscles of the thigh..........
Musc. of the calf.......
All anterior muscles of the lower leg......
Small muscles of the foot.

SEGMENT INNERVATION OF THE MUSCLES (after Edinger).
The reflexes are localized in the same segments as the muscles.

comprehensive, in which, however, as in all other diagrams, some defects are perceptible; but it must be remembered that there are variations in individual cases.

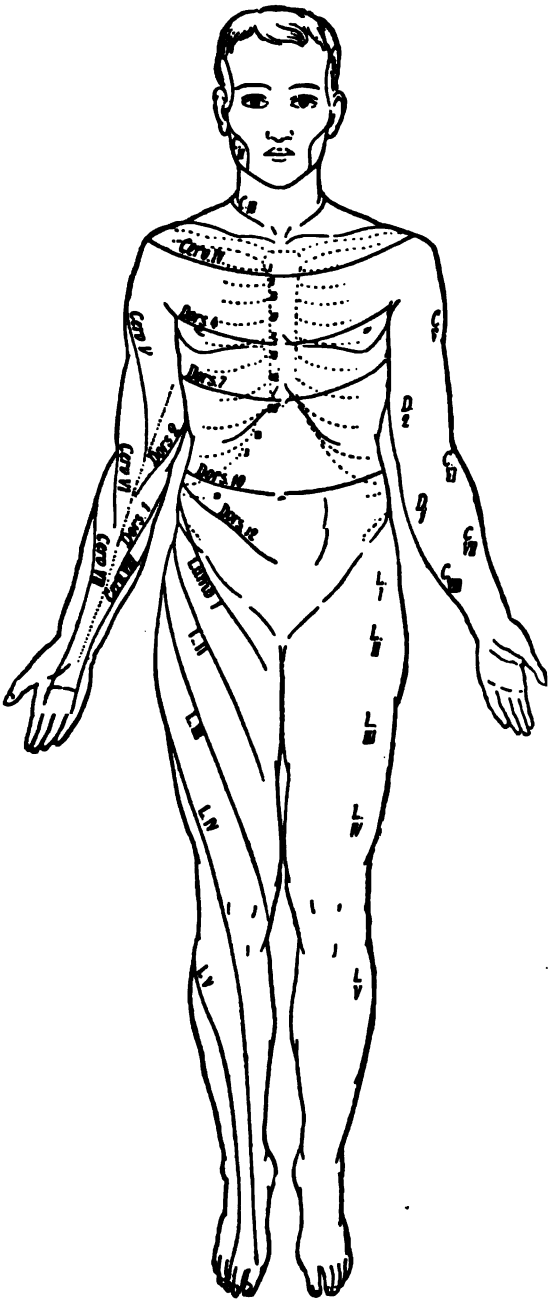


FIG. 151.—SEGMENT INNERVATION OF THE SKIN. (After Edinger.)

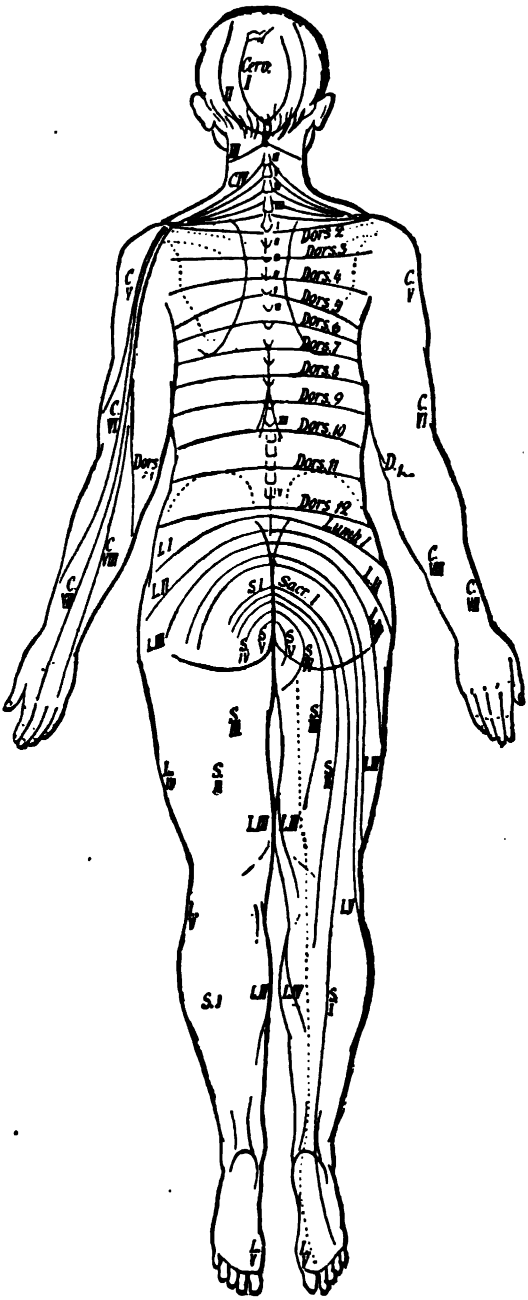


FIG. 152.—SEGMENT INNERVATION OF THE SKIN. (After Edinger.)

In this table the proximal muscles nearest the vertebral column are uppermost; the distal ones are lower down, according to their distance from the vertebral column. The segment number is marked above, and without follow-

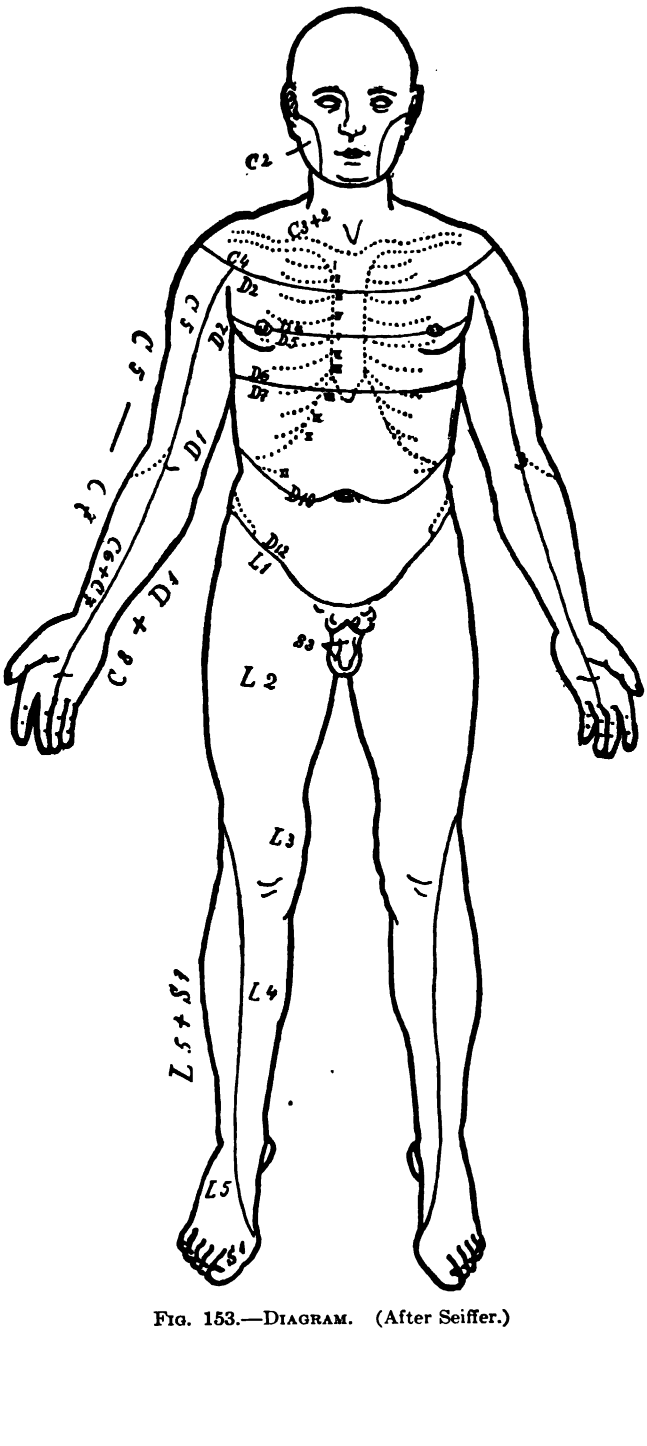


FIG. 153.—DIAGRAM. (After Seiffer.)

ing downward the lines between the individual segments we can plainly see which spinal cord segments supply the individual muscles.

The relation of cutaneous innervation to isolated spinal cord segments is

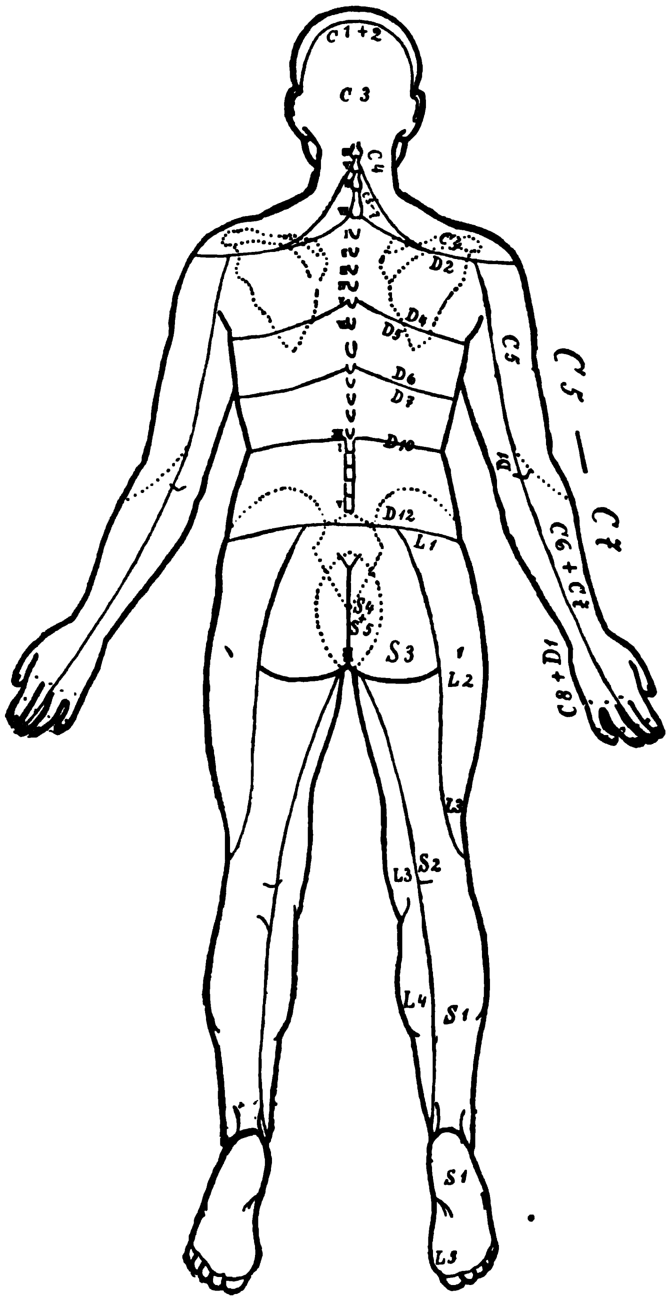


FIG. 154.—DIAGRAM. (After Seiffer.)

also clearly shown; in former diagrams the individual cutaneous areas were not sharply defined, this being impracticable because of the innervation of individual cutaneous areas by several different segments, and only the direction of the innervation was indicated by lines. But at either side of these lines lies the cutaneous area which is supplied by a definite spinal cord segment. At the same time it becomes evident, as Edinger explains, that the otherwise obscure distribution of cutaneous innervation in the extremities does not actually represent the case, because the diagram is based on embryology (Figs. 150 and 151). However, we may also utilize the diagrams of other authors. For the sake of the findings I have included those of Seiffer, as well as those of Head; the diagram of the latter author has enabled me exactly to localize many spinal cord tumors—at least their upper boundaries (Figs. 152, 153, 154, 155, and 156).

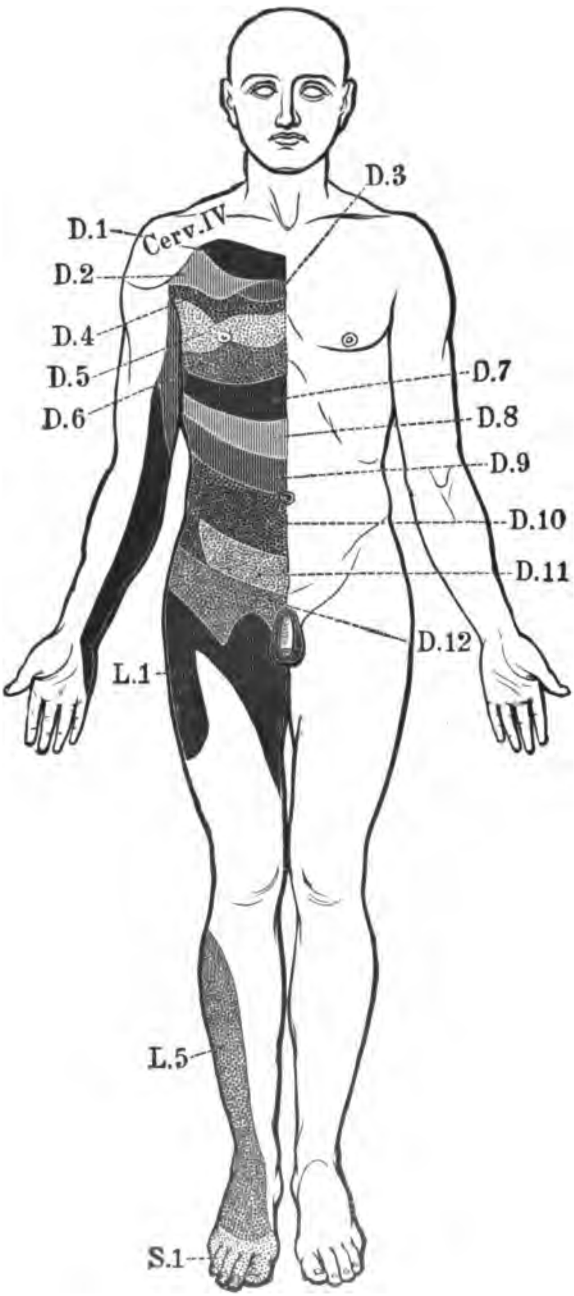


FIG. 155.—DIAGRAM. (After Head.)

By the aid of these diagrams we can usually ascertain exactly the upper boundaries of a tumor. It is only necessary to bear in mind the relation of the individual spinal cord segments to the vertebral bodies and the spinous processes belonging thereto. For this purpose Gowers' table is valuable; for it shows that the cauda equina begins opposite the first lumbar vertebral bodies and their spinous processes, and

that the greater portion of the lumbar vertebral column does not lie opposite the lumbar portion of the spinal cord (Fig. 158).

After determining by the aid of these diagrams the upper boundary of a tumor, we must ascertain its longitudinal extent and its lower boundary. This is often impossible, because the local phenomena due to absence of function which appear at the upper limit generally overshadow those produced by pressure of the tumor upon the total transverse section of the spinal cord. It is easier to ascertain the longitudinal extension of the tumor within the cervical enlargement, for when the muscles of the lower cervical segments remain uninvolved there is no strong pressure upon these. When the tumor is located opposite the lower portion of the dorsal cord, and when the muscles belonging to it continue to show no degenerative atrophy nor DeR, we may conclude that it does not press upon the upper portion of the lumbar enlargement. How many segments are compressed within the dorsal cord cannot be stated from our present knowledge. Examination of the abdominal reflexes permits no positive conclusions because these reflexes may be abolished even when a circumscribed tumor is in a high location. Their appearance, even if only partial, would contraindicate the extension of a tumor downward.

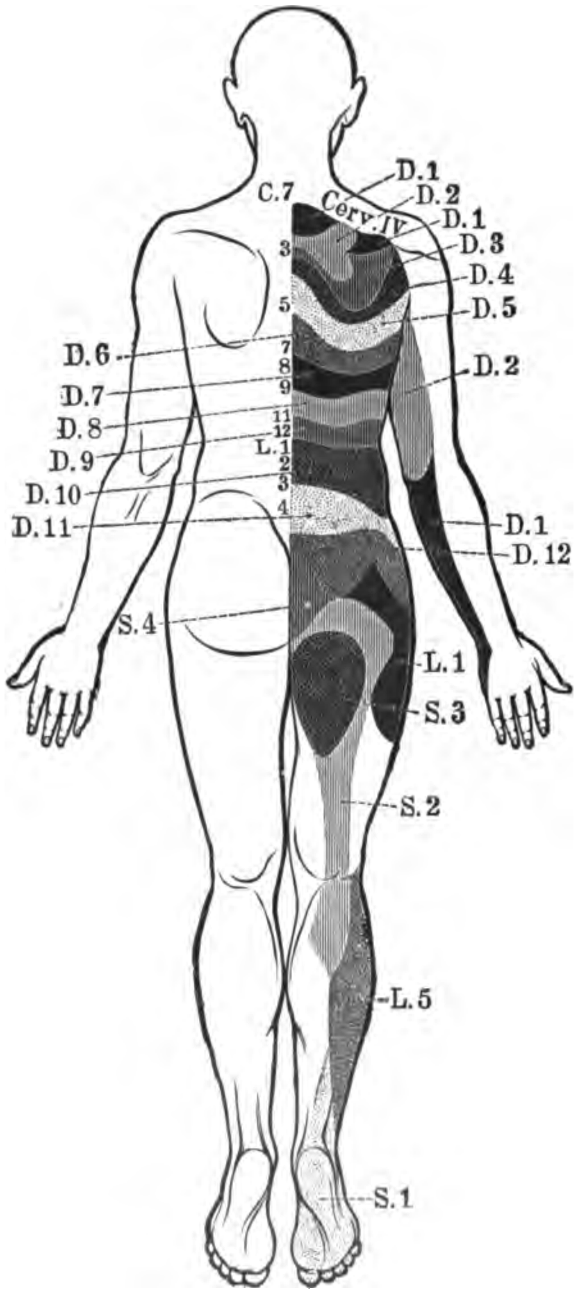


Fig. 156.—Diagram. (After Head.)

The *absence of the tendon reflexes* must be cautiously utilized, for this is observed even when there is no direct pressure upon the segments belonging thereto. When there is an acute transverse interruption of the spinal cord above the lumbar enlargement, the patella and Achilles tendon reflexes

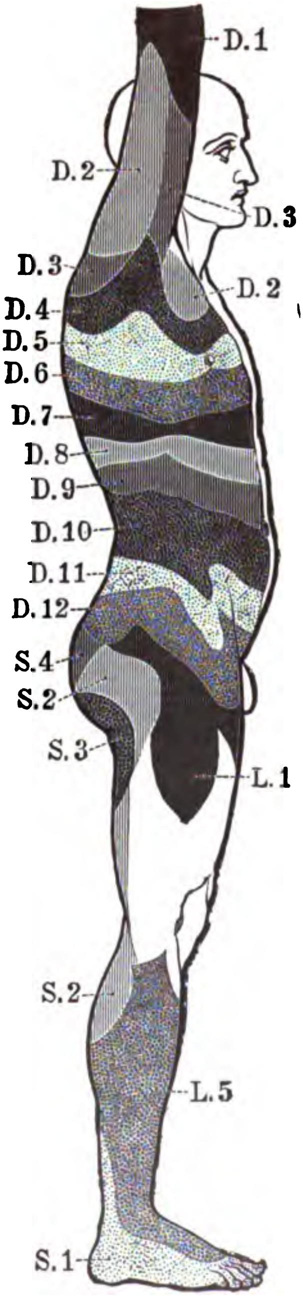


FIG. 157.—DIAGRAM. (After Head.)

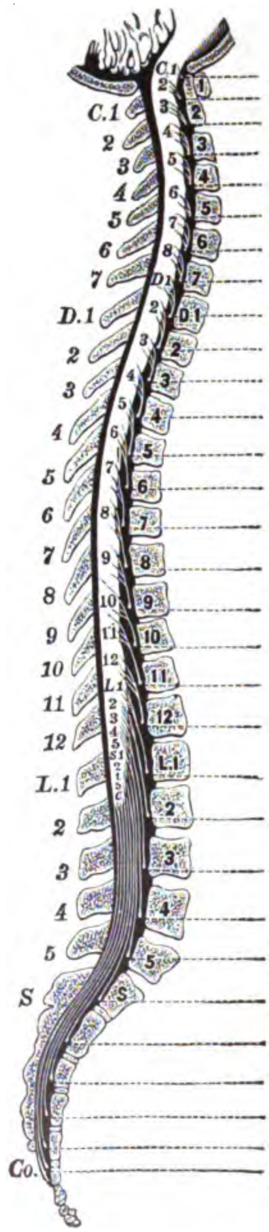


FIG. 158.—DIAGRAM. (After Gowers.)

usually disappear.[1] On the other hand, their presence or increase shows that there is no pressure in the corresponding segments.

Little is revealed by *pain* and *paresthesia* remote from the upper border of the tumor in deeper situated segments of the spinal cord and the cutaneous areas belonging thereto (for example, in the legs from tumors of the cervical cord); for these abnormal sensations may likewise appear when there is pressure much higher up, on the deeper situated centripetal *intraspinal* tracts, but not directly upon the segments. For instance, there may be a bilateral "girdle sensation" around the lower portion of the abdomen when a tumor presses upon the cervical segments.

Fortunately *circumscribed tumors* are much more common and grow more slowly than extensive ones, so that operative measures are not contraindicated when we find a very large tumor. Circumscribed multiple tumors which grow slowly are more rare than solitary ones, and they do not produce pressure phenomena. If they lie close to each other, they will of course produce the same symptoms as elongated connected tumors. When far apart (for instance, one in the cervical, and the other in the lumbar, enlargement), the exact location may be readily diagnosticated if *multiple* tumors also can be detected, or if there are metastases from malignant tumors which cannot be cured by operative removal.

When these can be excluded the local diagnosis of tumors of the dorsal cord and cervical enlargement can usually be made with exactness. For the middle and lower dorsal portions the following process is advisable: We ascertain as exactly as possible the upper boundary of the hypesthetic area on the trunk, and by referring to Head's tables we note which segment is supplied by the upper portion of the hypesthetic area that has been found; then by the aid of Gowers' tables we determine which vertebra and which of the spinous processes adjoin the damaged spinal cord segment. The surgeon must make his incision opposite this vertebral process, which is often sensitive to pressure. Usually the mode of operation is as follows: if, for example, hypesthesia is found pointing to the 10th dorsal segment, incision is made opposite the thoracic vertebra lying immediately above (the 9th), and also at the vertebral process next above (the 8th).

Local diagnosis is much more difficult at the *height of the lumbar enlargement*, of the *conus terminalis* and of the *cauda equina.* This is due to the fact that the anterior and posterior roots in this region do not at once leave the vertebral column when they emerge from the medulla spinalis, but they pass alongside a number of vertebral bodies before they disappear. Therefore, with a corresponding location of the tumor, and if there is pressure at the middle of the conus terminalis, the same symptom-complex may sometimes arise as when the tumor is located in the region of the last lumbar vertebra or the sacral vertebra. With such an exceedingly *difficult* operation as an extensive one in the region of the lumbar vertebral column, it is wise to make as exact a local diagnosis as possible.

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[1] I recently saw a case of *incomplete* transverse lesion due to external compression by a metastatic sarcoma at the height of the sixth spinous process of the dorsal vertebral column, and five days after the appearance of pressure phenomena the tendon reflexes of the lower extremity were abolished. Autopsy revealed a tumor at the height mentioned, but not in the lumbar enlargement.

Fig. 159 shows distinctly that a focus at the height of the 12th thoracic and the first lumbar vertebra—this point is indicated by shaded lines—may spare the crural nerve situated laterally to it equally as well as if it were situated below the exit of this nerve further downward, and that the same symptoms may be produced in other nerve regions. For this reason great pains have been taken to establish differential factors, and it has fortunately been shown that in those lesions which act slowly, as tumors usually do, the differentiation between *tumors of the lumbar enlargement* and *caudal tumors* is not so perplexing as when disturbances appear suddenly; for example, those of traumatic nature. In the latter a distinct change in the individual vertebræ not infrequently indicates the point of lesion, a change which is even more perceptible under X-ray examination. Moreover, experience teaches us that in certain kinds of trauma (for example, a fall upon the buttocks from a great height) only the fracture of certain bones is found. Large tumors may now and then produce a decided and visible prominence of some portions of the lumbar vertebral column or of the sacrum. But this is rare, and in circumscribed tumors even local sensitiveness to pressure may never appear.

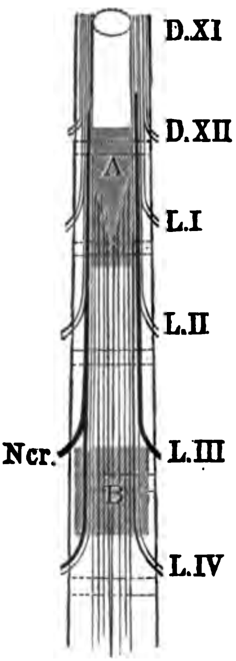

FIG. 159.

We must, therefore, resort to other methods of differentiation. This is evident from the fact that on pressure in the lumbar enlargement bilateral paralytic symptoms (at first of a *motor nature*) appear *more rapidly*—even if the tumor at first makes lateral pressure—than if it compresses the cauda equina. Dissociated *sensory paralyses* may develop more readily than in caudal tumors, while irritative phenomena are secondary (especially pain which in tumors of the cauda equina is usually *very severe*). On slight pressure upon the motor ganglion cells of the lumbar enlargement, irritative phenomena in the form of fibrillary and fascicular contractions are more common. Finally, a fact which Raymond noted in regard to the reflexes, namely, that when one of the deeper reflexes is lost those immediately higher may be increased favors disease of the lumbar cord. If the entire lumbar cord is not compressed but only its terminal portion, the conus terminalis, the following peculiar symptom-complex gradually arises: *paralysis of the bladder and rectum*, absence of the sexual reflexes as well as the *Achilles tendon reflex, without paralysis* of the muscles of the lower extremities. Simultaneously a varying degree of anesthesia is noted in the anus and its surroundings, the perineal region, the posterior skin of the scrotum, the mucous membrane of the urethra and bladder, and the posterior and internal surfaces of the thighs.

Since the nerve fibers originating in the conus extend far down into the sacral portion of the vertebral column, and at a certain point lie next each other, pressure far below the conus terminalis may produce the same phenomena.

Only a possible sensitiveness to pressure in the region of the corresponding vertebræ will then facilitate the decision. If the symptom-complex described be absent, it may be utterly impossible to make an exact topical diagnosis.

The difficulty of determining the exact point for operation will be better understood by the details of a case I recently observed (in which, unfortunately, exact diagnosis was impossible) than by long descriptions. It was the case of a man, aged 45, with no history of preceding infectious disease, certainly not of syphilis, but merely of prolonged alcoholism. There was no hereditary predisposition of any kind, nor had there been any trauma. In the winter of 1900–1 pain appeared in the right thigh. He felt this both when in the recumbent posture and when standing, but not while sitting or walking. He was even able to make many walking tours in the Schwarzwald—exhausting walks through deep snow and mountain climbing for as much as eight hours a day—without any pain. In the summer of 1901 the pains disappeared completely, but recurred the following winter. This time they were less severe but more continuous, and made sitting extremely difficult, while in the recumbent posture they were not felt, and they appeared in walking only when exhausted by several hours' marching. Shock from careless running down hill also produced them.

His condition remained stationary until the winter of 1902–3. In January, 1903, the pains increased so much as to render long walks impossible. The patient did not consult a physician until the spring. He was placed upon potassium iodid and sodium salicylate. Warm carbonated baths were employed without improvement. In the course of the year the pain attacked the *right leg and right foot*, finally the *left leg*. Treatment at Oeynhausen for five weeks in September, 1903, as well as treatment by aspirin in the following October, did not benefit him.

Pausing for a moment, we ask the question, What could have been diagnosticated during this time? Only sciatica, perhaps muscular rheumatism, was possible. But the course of the disease was opposed to such an opinion, since ordinary sciatica appears suddenly and disappears gradually. It is true relapses may occur, but they do not progress so slowly nor do they attack the other side, as in our case. Slight tenderness was at first also conspicuous, and this led us to believe that somewhere in the course of the sciatic nerve or its plexus or its roots compression had gradually developed, either in the bone, in the soft parts, in the sciatic nerve itself (perhaps in the form of neuroma), or in the vertebral canal. This view seemed to be substantiated when pain appeared in the left leg also.

Toward the end of January, 1904, the patient was admitted to the hospital in Bonn. Brief examination revealed the following: A robust man whose internal organs gave no evidence of change, and whose bones and joints, including those of the vertebral column, presented no demonstrable anomaly. There was no sensitiveness to pressure in the vertebral column nor in the bones of the pelvis. Digital examination of the rectum disclosed nothing abnormal. A *waddling gait*, due to atrophy and weakness of the medial gluteal muscles, especially the right, was conspicuous, but the larger gluteal muscles were also atrophic, and this made the ascent of steps very difficult. There was no DeR in these muscles, but a marked diminution of electric contractility on the left side and total absence on the right side with all the different currents was demonstrated. In the course of the right sciatic nerve there was marked hypesthesia of the external malleolus and of the external border of the foot. The pain and temperature senses were also decreased, and in the toes the sensation of position was defective. The leg could be flexed by the exercise of very slight power. The right Achilles tendon reflex was absent, and the left was weak. Babinski's reflex was abolished on the right side but distinct on the left. Nothing really anomalous was noted in the region of the left sciatic nerve except the so-called *sciatic phenomenon* (pain in the sciatic nerve upon raising the extended leg).

In the course of the crural nerve there was no demonstrable pathologic change. But it was difficult to raise the limb toward the trunk. Sensation about the anus, the perineum, and in the skin of the genitalia was absolutely normal, as were the bladder and rectal functions. The abdominal reflexes were good, the right cremaster reflex being well preserved, but the left very weak. There was pain in the right sciatic region which was especially distressing when lying upon the back.

This symptom precluded our designating the affection as a progressive disease of the sciatic nerves alone, because the gluteal nerves and those of the ilio-psoas were also implicated. We at first thought that a tumor was present, either in the lumbo-sacral plexus, within the sacrum itself, or pressing upon the roots in the vertebral canal. Chronic meningitis at the height of the cauda equina was contraindicated by the absence of syphilis, of tuberculosis, and of trauma, as well as by the circumstance that the affection did not more rapidly become bilateral but progressed with long pauses and gradations, although there was at first a decided and prolonged remission.

Of course, there might have been disease of the sacrum itself, or of the lumbar vertebræ, or of both, yet no sensitiveness to pressure and no change in form; even the X-rays revealed nothing abnormal. Nevertheless, such disease could not be absolutely excluded, although inability to find a primary focus (for example, in the prostate) was against the view of metastatic tumors. Normal conditions in the urine contraindicated rare myeloma. A tumor of the vertebral column could not have caused pressure over the lower half of the lumbar enlargement and the conus terminalis because the pathologic phenomena developed much too slowly. There were neither bladder nor rectal disturbances, neither motor irritative phenomena, dissociated sensory paralyses, nor the peculiar anesthesia previously described.

Compression of the fibers of the cauda equina, which, after excluding tumors with higher seat, finally comes under consideration, was favored by the severe pains so frequently observed in this condition; it was contraindicated by the absence of bladder and rectal phenomena. It was difficult to understand how the ilio-psoas fibers originating from the upper lumbar segments could be pressed upon, as well as the roots of the sciatic and gluteal roots, while the fibers of the crural and obturator nerves remained free.

The possibility, and even likelihood, of *multiple* tumors of individual roots and nerve trunks was suggested; of course the possible tumor or multiple tumors could not be exactly located, hence it was impossible to point out to the surgeon those lumbar vertebræ below the first and the sacral vertebræ (with the exception of the lowest which did not come under consideration) over which he was to operate. Opening the greater portion of the lumbar and sacral vertebral column simultaneously is such a serious operation that I did not think it advisable, particularly as the affection appeared to be arrested in the following months, and up to May there was decided improvement, especially a relief from pain.

Then the constipation increased, and about the middle of June marked sensitiveness in the buttocks reappeared, as well as in the distribution of the sciatic. Atrophy of the gluteal muscle simultaneously increased, and there was loss of weight in the previously strong and well nourished body. Slight *difficulty in urination* now appeared, and at the beginning of August circumscribed analgesia to the right of the anus; the sphincter ani no longer contracted. The weakness in the sciatic increased; paresis of the right peroneal region appeared, and the Achilles tendon reflexes were abolished bilaterally, while the left plantar reflex was still distinctly preserved. The patella reflexes were feeble. About the middle of December a small bed-sore appeared, and the general debility increased.

The pathologic picture now simulated a tumor at the height of the conus terminalis, and it seemed quite possible that the supposed growth might have gradually extended this far; but one or several tumors might also make pressure lower down in an indefinable position.

Considering this and the gradual decrease in weight, which could not well be accounted for, it seemed reasonable that a rapidly growing tumor, probably malignant and inoperable, might have developed from the sacrum or from the plexus and its intervertebral continuations. Opposed to this was the continuous absence of local pain on pressure of all the osseous structures which came into consideration, and the absence of deformity. Notwithstanding several attempts, no X-ray examination could be made on account of the great accumulation of feces which it was impossible to remove, and because of the great pain.

Even in this stage it might be deemed advisable to make incision at the level of the sacral vertebræ, and, if no tumor were found there, either at once or subsequently at the height of the second lumbar vertebra; but since the tumor is as likely to be opposite the 3rd, 4th, or 5th lumbar vertebra, and because the operation would probably be fatal, I cannot advise this method. At all events, it seemed certain that the

operation might at first aggravate the bladder weakness and the bed-sore, and thus weaken the patient; hence it was deemed wise first to try to heal the bed-sore, to say nothing of the fact that a local sensitiveness to pain might arise and give us more definite indications. Unfortunately, after September the disease progressed with exceeding rapidity, the patient constantly growing worse. The bed-sore rapidly enlarged, and finally necessitated the use of a permanent bath. The fever rose, the strength rapidly declined, the sciatic paresis increased, the patella tendon reflexes could no longer be evoked, and upon the 15th of February, 1905, the patient succumbed to septic fever.

The *autopsy* revealed the presence of a tumor 19½ cm. long, beginning about 1¼ cm. below the lower end of the conus terminalis, and compressing the entire cauda equina. Its greatest breadth above was 1½ cm.; below, 2½ cm. An illustration of it is given in Fig. 167. The upper end of the preparation is somewhat sunken in consequence of gravity while in an erect position. It was loosely adherent to most of the roots of the cauda equina, and was detached without difficulty. Microscopical examination showed it to be a sarcoma.

If, therefore, operation had been attempted a few months earlier, it is likely that even in August or September the tumor would have attained such a size that many vertebræ must of necessity have been opened, and death would just as certainly have resulted—as in a similar case which I observed. How extensive the tumor was when the patient came to us could not be absolutely determined, but it was probably then quite large. The autopsy revealed nothing accurate as to its point of origin. It is just as likely to have started opposite the 4th or 5th lumbar vertebra as in the upper half of the cavity of the sacral vertebræ; therefore at the operation luck would have been a prominent element.

For a long time no diagnosis of tumor of the cauda equina could positively be made. Our method of procedure was justified by the autopsy, especially as the prolonged absence of bladder weakness in the light of similar experiences warranted us in assuming that it was not a caudal tumor.

Fortunately the diagnosis is not always so difficult as in this case. Circumscribed sensitiveness to pressure (which we searched for in this patient with negative results) usually points to a possible tumor within the sacrum or opposite the lumbar vertebral column, although it permits no absolute conclusions as to its length. In a case reported by Laquer [1] of Frankfort-on-the-Main, tumor formation within the sacral canal was diagnosticated on account of "strictly localized pain" in the middle of the sacrum which had existed for two years; there was also pain on pressure in the same area. When the surgeon made incision at the corresponding point, an extradural tumor was found which extended to the vertebral canal, and had compressed the cauda equina, but could easily be removed. The patient had suffered from almost unbearable pain, as well as disturbance in gait and transitory paresis of the bladder and rectum, and a bed-sore had already formed in the region of the trochanter; he was almost entirely cured by the operation.

The local diagnosis is easier when, as in a case recently published by Engelmann,[2] a gunshot wound in the region of the sacrum indicates the location. In this case there was severe local and radiating pain. The ball had entered between the 5th lumbar and the first sacral vertebræ, and in this region also there was pain. Moreover the peculiar anesthesia previously described was noted, as well as cystitis and constipation, while motor paralytic phenomena were absent in the lower extremities, and of all the cutaneous and tendon reflexes in the legs only the Achilles tendon reflex was present.

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[1] *Neurolog. Centralbl.*, 1891.
[2] *Münch. medic. Wochenschr.*, 1904, Nr. 51.

These disturbances might have been attributed to an injury much higher up—in the lower portion of the conus terminalis—but the location of the wound indicated the region of the sacrum. The bullet, which was accurately located by X-ray examination, was found at the height of the first sacral vertebra, and removed. Four months after the operation the condition of the injured patient was almost normal. There was no pain; except for a periodic and slight dribbling of urine the bladder was normal, and there was only a small hypesthetic zone around the anus.

FIG. 160.—SARCOMA OF THE CAUDA EQUINA.

If, in this manner, we attempt to diagnosticate a tumor with certainty or even likelihood, we must also ascertain whether its location is *extradural* or *intradural*; and, if the latter, whether it lies between the pia and dura, or between the pia and the spinal cord. Fortunately the latter is very rare. I say fortunately, because in this case the differentiation between acute transverse myelitis and pressure from tumor can scarcely be made; for, on the one hand, there are few sensory irritative phenomena, and, on the other, a general inhibition of conduction, an interruption within the spinal cord, very rapidly takes place.

We are still unable to differentiate between extradural and intradural tumors, although great attention has been devoted to this distinction. Nor can we determine (important as this may be in deciding the question of operation for tumor) whether a tumor grows anteriorly or pos-

teriorly, or presses upon the spinal cord. We might suppose that in posterior pressure irritative phenomena and radiating pains would from the onset predominate, and that in anterior pressure these might be absent or only slight. But this is by no means always the case. I have been convinced that, even with posterior pressure, the initial and neuralgic stage of the disease or pain may be almost wholly absent.

It is easier to determine that a tumor grows laterally, or (at least chiefly) from one side. This for a time produces the familiar picture of *semi-lesion*, or *Brown-Séquard's paralysis*; i. e., motor paralysis predominates on that side of the spinal cord in which there is most marked pressure, on the other sensory paralysis, especially involving the pain and temperature sensations.

Furthermore it is important to demonstrate whether the cause of compression is *disease of the vertebral column* or not; above all, whether tuberculosis is present. This may be ascertained by an accurate history and close search for tubercular foci in the body, by the demonstration of fever, distinct changes in the form of certain portions of the vertebral column, and, after a long period of observation, by X-ray examination. Syphilitic disease of the vertebral column causing pressure upon the spinal cord is very rare, and, as a rule, is favorably influenced by antisyphilitic treatment. Exostoses develop with exceeding slowness, and in a given case can probably be recognized by radioscopy.

Carcinomata and *sarcomata* of the vertebræ are most common. Since, in our experience, the former only secondarily develop in the vertebral bodies, therefore usually in elderly persons, we should endeavor to ascertain whether there had been a prior operation in which a malignant tumor was removed, this fact being sometimes concealed. Above all, a possible primary malignant focus must be sought for, and unusual points of origin must not be excluded from consideration; for example, the prostate, the ovary, the thyreoid gland, the bronchi, etc. The primary cancer may have been strictly local. Persistent and severe pain on motion as well as during rest, particularly if extensive, is an especially noteworthy symptom. Vertebral cancer is usually multiple. Not rarely it is widely proliferated in the vetebral column, and may lead to sudden rupture with immediate symptoms of compression. Abnormally arched curvatures of the vertebræ may develop, while in tuberculosis of the vertebræ the angular kyphosis is much more likely. Sarcomata of the vertebral column may be primary or secondary. The former is perhaps more common. In the great majority of cases the secondary form is multiple, the primary less often so.

In the diagnosis of secondary sarcomata the same considerations influence us as in cases of carcinomata. All regions of the body must be minutely examined for primary tumors. Multiple primary sarcomata often produce distributed and various kinds of pain. Pain in the bones on pressure and motion prior to the appearance of root symptoms, particularly neuralgic ones, favors an osteogenetic origin. Of course a positive diagnosis is not always possible, nor can we exclude tumors which originate from the spinal membranes. As in rare enchondromata, X-ray examination may in some cases aid us; while extremely rare osteomata, exostoses, and chondromata are probably always recognizable by this means.

THE COMPOSITION

The diagnosis of the exact composition of tumors not originating from the bone but from the membranes cannot at present be made. Fibromata and fibrosarcomata are the most common, and, if the growth is not too rapid, can be diagnosticated. If other primary tumors, such as carcinomata, sarcomata, melanomata, are anywhere detected, and the previously described symptoms are present, we will not err in assuming metastases of these growths. Since in rare cases cysticerci and echinococci may be found, these also should be searched for.

In regard to the *course* of the pathologic phenomena produced by such tumors and their possible *cure*, it may be stated that syphilitic neoplasms are amenable to treatment. It is, however, unfortunately true that nearly all others enlarge, they increase the pressure upon the spinal cord, and finally prove fatal, although often only after the lapse of years. Even the so-called benign tumors may thus have a malignant effect. Some tumors, especially neurofibromata, may remain stationary; in an interesting case reported by Henschen this author assumed with good reason that retrogression had occurred.

Usually the pathologic phenomena gradually increase, although by no means uniformly. Comparatively often, especially in the first or neuralgic stage of the disease, there is prolonged or permanent *amelioration of the pain.* In the later stages when the symptoms of compression are prominent *remissions may occur*, probably because the tumor extends posteriorly, and there may be a temporary decrease in size, perhaps in consequence of softening, or because the spinal cord tract becomes accustomed to a steady, uniform pressure. If iodin is administered during such a period there may be an apparent improvement which may lead to an erroneous diagnosis of syphilis.

It is of great significance in the diagnosis that, in spite of this advance of the pathologic phenomena (certainly in *ordinary tumors which extend but slightly upward*), *the height of the region involved in the pathologic phenomena remains approximately the same*; even these tumors do not extend upward, and they produce only the picture of a constantly increasing transverse lesion. This peculiarity enables us to differentiate circumscribed tumor formation from other spinal cord diseases—above all syphilitic meningomyelitis and the various forms of transverse myelitis.

The fact, often so conspicuous, that pain is increased by sudden shock (for example, by coughing or sneezing) is not without value; but this is also observed in other diseases of the vertebral column, the vertebral joints, and the muscles of the back. *Spastic rigidity* of the muscles with *tonic and clonic reflex spasms*, often *exceedingly painful* and markedly developed, is not characteristic, although it frequently occurs; for these disturbances may also be observed in other diseases of the spinal cord.

As a rule, the disease is fatal, and terminates after extreme suffering in consequence of cystitis, pyelitis, or bed-sores.

TREATMENT

Except for antisyphilitic treatment in the rare gummatous tumors, our *only* hope is in *timely operative removal of the compressing tumor.* Of course, this treatment should be tried before operating, as syphilis cannot be excluded with absolute certainty. Even if the patient is syphilitic, and gummata are found in other portions of the body, pressure upon the medulla spinalis may nevertheless be due to other tumors.

While the diagnosis of tumors of the spinal cord as well as operative interference for the purpose of removal appeared impossible until a few decades ago, yet a neoplasm of the spinal cord membranes was for the first time correctly diagnosticated in 1887 by Gowers and Horsley. They localized it, and successfully removed it by operation. Others have since followed, and much more satisfactory results have been obtained than in analogous operations for tumor of the brain. Statistics up to the beginning of 1905 which I collected, and including a case not yet published which terminated unsuccessfully, show 62 such cases, of which 24 were cured or greatly improved, therefore 38 per cent. Here it must be remarked that the patients who improved were saved from certain death by operation. In 7 cases of my own in which operation was performed I was so fortunate as to reckon *3 recoveries* and one permanent improvement. In an eighth case, the one not yet published, death followed the operation. In a ninth case, also not yet published, there was a metastatic tumor which had not been recognized as such; operation did not aggravate the case, but, after the wound had healed, death occurred in consequence of a sarcoma of the thyreoid gland.

The conditions are most favorable when tumors are situated at the height of the *dorsal vertebræ,* less so when in the cervical and lumbar regions including the cauda equina. Nevertheless Putnam, Krause, and Park have successfully operated on tumors at the height of the *3rd cervical segment,* while Bardenheuer failed with a tumor diagnosticated by Minkowski and myself. On the other hand, Henschen and Lennander in Upsala, and Harvey Cushing in Baltimore, have successfully removed tumors in the lower portion of the cervical enlargement and at the height of the 5th and 6th cervical vertebral arches.

A tumor opposite the *sacrum,* extending from its middle almost to the lowest lumbar vertebra, diagnosticated in France by Laquer, was successfully removed in Frankfort by Rehn. One of our patients with a large tumor of the sacrum which extended upward to the first lumbar vertebra succumbed in spite of an operation performed by Schede. Extensive operations in this region, especially in bony persons, are very serious on account of the large and deep osseous wounds and the unavoidable aggravation of the preceding pathologic symptoms, while the opening of the sacrum and the removal of individual lumbar vertebræ are much less dangerous.

The *technic* of the operation is not especially difficult although the utmost caution is necessary. Too much pressure upon the spinal cord must be avoided, for there is but slight possibility of its regeneration, and the paralysis becomes profound and incurable. Especially in operations on the upper portion of the medulla spinalis and in intradural tumors, we must prevent the outflow of the cerebrospinal fluid; according to Sick this may be done by carefully tying the

dural sac with a thread prior to opening the dura. Finally, we must guard against severe hemorrhage during the operation; this will give the patient a better chance to recover from the aggravation of the paralysis and other symptoms of his disease immediately after the operation. I cannot enter into the details of these conditions which belong to my surgical colleagues. They have a particularly difficult case when it is ascertained that the tumor is not situated behind or alongside the spinal cord, but has grown anteriorly to the medulla spinalis, and is very difficult to detach. Sometimes the tumor cannot be entirely removed, or it may happen that in spite of all precautions there are severe secondary injuries.

But in the successful cases we always have the satisfaction of having saved the patient from great suffering and certain death. It is the duty of the internist to make a diagnosis as early as the data at hand will permit—chiefly to ascertain that there is neuralgic pain in the trunk and extremities, as well as increasing motor, spastic, or sensory paralysis with or without preceding neuralgia, constantly *to consider the possibility of tumor*, and to seek for its first symptoms. It is very humiliating to discover at the autopsy that we have failed to diagnosticate a tumor which might readily have been removed, and that in consequence of our lack of knowledge or carelessness the patient has succumbed to a painful death.

MYELITIS

By E. v. LEYDEN and P. LAZARUS, Berlin

HISTORY

A glance at the development of the pathology of the nervous system during the last century will reveal the wonderful and complete reconstruction that has taken place. This is especially true of diseases of the spinal cord, for a century ago the knowledge of the normal and diseased spinal cord was exceedingly scant; but to-day this is one of the best cultivated realms of our science. At the beginning of the preceding century all pathologic processes of the cord and its membranes were included under the same designations, "rachialgitis" and "spinitis" (Brera, Jos. Frank), also as notomyelitis (Hildebrandt). The first attempt at a separation of disease of the cord from that of its membranes, occurred in a dissertation by Harless (Erlangen, 1814) and by Klohss (Halle, 1820); but only in the celebrated writings of Ollivier (Edition I, which was awarded a prize by the Academy of Paris) and in the works of Abercrombie (1828) did myelitis attain a substantive position and a clinical description which was a classic for that period.

These researches contain a good description of the macroscopic changes of the inflamed cord. The altered consistence of the spinal cord was differentiated as *softening* (*myelomalacia*) and *hardening* (*sclerosis*), the latter according to the views of pathology at that time being regarded as the starting point of an inflammatory process. In reference to softening in the cord, and also in the brain, the red, yellow, and white variety were differentiated. White (central) softening was soon shown to be a post mortem change and without pathologic significance. Red, and white softening, almost up to the present time, were regarded as true representatives of spinal cord inflammation.

The next advance we owe to microscopy and in connection with this the improved methods in hardening and staining (M. Müller's chromic acid fixation, L. Clarke's clearing with turpentine, Gerlach's carmin staining), by which processes the normal and pathologic anatomy of the spinal cord were revealed.

The first important discovery in pathologic histology of the cord was due to Gluge in Halle (1850). He described, what was called after him, the "*inflammatory globules*" corresponding to our present *granular cells.* To demonstrate them a spread of tissue, to which a little dilute caustic soda has been added, is placed under the microscope and even with moderate magnification they are readily seen; by their black, granular consistence granular cells are sharply defined from the pale medullary substance (Plate I, Fig. 3 *a*). The great simplicity and rapidity of this method of examination renders it

still useful, provided it is followed by a minute microscopic investigation after hardening and staining. Granular cells are found in great numbers within the inflamed substance of the cord in myelitis, but they are also seen in other, pure, degenerative lesions in the brain and spinal cord.

This simple method led to one of the most important advances in the realm of diseases of the spinal cord; we refer to the discovery of *ascending and descending columnar degeneration*, by one of the most original medical investigators, L. Türck, in Vienna (1851),[1] who was also the founder of laryngoscopy. It is true Cruveilhier, in his celebrated Atlas on Pathological Anatomy (Tom. II, 1835–1842), had described and depicted columnar degeneration as "Dégénération fasciculaire," without, however, pointing out its special significance. Leyden, in one of his first publications[2] upon diseases of the spinal cord, connected gray degeneration of the posterior columns in tabes with Türck's ascending, secondary degeneration and thus constructed his theory of the pathogenesis of tabes (1863). Türck's discovery further led to the separation of column or system diseases from focal diseases of the cord, which is still in force to-day. Focal diseases to a great extent are included with acute and chronic myelitis, maladies with which we are particularly concerned.

PATHOLOGICAL ANATOMY OF MYELITIS

For a long time myelitis was regarded as identical with softening (*myelomalacia*). The inflamed spinal cord focus, especially in advanced cases, may be recognized on palpation by its decreased resistance, from a distinct swelling, as well as from an active injection of the meningeal vessels. On section of the focus the softening becomes even more marked, for the medullary substance protrudes upon the cut surface; the color is whitish-yellow or reddish, not rarely speckled red, in consequence of disseminated hemorrhagic foci or capillary apoplexies. In more advanced cases the pia mater surrounds the completely softened cord substance, which has a pappy consistence upon the cut surface, like a loose sac. In other cases, in which the softening is not well defined, the normal structure of the gray and white substance is more or less obliterated.

Not every softening is of inflammatory nature; there is also *necrobiotic, ischemic and traumatic softening without inflammation.* On the other hand there are not infrequently spinal cord inflammations *without* softening, to which Leyden called attention in his book upon "Diseases of the Spinal Cord" (1872–1876). In *myelitis without softening* there may be no visible macroscopic change; only the succeeding histologic examination, either in smear preparations or after hardening the cord, decides the diagnosis of myelitis. Mostly in this form, which belongs to the early stage, the histopathologic lesions of myelitic softening are found to an attenuated extent, i.e., only

[1] L. Türck, "Ueber ein bisher unbekanntes Verhalten des Rückenmarks bei Hemiplegien." *Zeitschr. d. Aerzte zu Wien*, 1850.—"Ueber secundäre Erkrankungen einzelner Rückenmarksstränge und ihrer Fortsetzungen zum Gehirn." Wiener Sitzungsberichte 1851, VI. u. XI. Heft.

[2] Leyden, "Die graue Degeneration der hinteren Rückenmarksstränge." Berlin, 1863 und *Deutsche Klinik*, 1863, 23.

swelling, or at most beginning destruction of the ganglion cells, medullary sheaths and axis cylinders, as well as disseminated round cell infiltration and more or less numerous granular cells. Plate I, Figs. 1, 2, and 3 gives an idea of the pathologic lesions in myelitic softening.

The alterations of the parenchyma in myelitis are of a *destructive* nature; those of the *interstitial tissue, that is of the neuroglia, are of productive kind, and the lesions of the vessels are of congestive, i. e., of exudative nature.*

The *ganglion cells,* according to the stage of inflammation, present degenerative pictures of varying kinds. At the onset of inflammation the ganglion cells swell, the multipolar anterior horn cells particularly become clumpy, they do not permit the recognition of Nissl's bodies, or when present they are cloudy, finely granular and often show hyaline swelling. The cells stain poorly and are subject to chromatolysis, that is achromatosis. The nucleus moves to the periphery of the cell, it contracts, and finally can hardly be differentiated from the body of the cell, which has been converted into a homogeneous, structureless clump. In the terminal stages of the inflammation the ganglion cells may perish and be completely destroyed under conditions of pigment degeneration and vacuole formation, or they contract including their processes (atrophy). According to Marinesco, proliferated glia cells are said to penetrate the protoplasm of the ganglion cells and analogous to phagocytosis these glia cells are able to take up the disintegrated substance ("neuronophagous glia cells").

The *nerve fibers* are also destroyed in a manner parallel to the cellular changes. In the beginning of acute myelitis the *axis cylinders* swell, they show varicose thickening and sometimes fine granulation, which indicates the threatened destruction. The *medullary sheaths* show globular-shaped swollen or contracted areas; they are finally destroyed with the signs of fatty degeneration (myelin globules) and of vacuole formation, so that the space occupied by the nerve fibers presents a cavity. In a transverse section these cavities appear like vesicles; they are larger than the normal transverse section of a nerve fiber and still contain a few axis cylinder fragments, without medullary sheaths or medullary sheath particles, which are changing into myelin globules; very often also granular cells are present. Fig. 161 (myelitis after gonorrhea) illustrates this change which was described and named by Leyden a "vesicular condition." (Compare also Fig. 162, syphilitic myelitis.)

This specimen was obtained from Paul E., a workman, aged 27, who was admitted to the Clinic on March 2, 1890, and died on March 9th. There was *complete flaccid paraplegia,* which had developed almost suddenly, 2 days prior to admission to the Clinic. The patella reflexes were abolished, and up to the navel, in a horizontal line, sensation was altered (hypalgesia, hypesthesia). The sphincters were incontinent. The purulent urethral secretion contained gonococci.

The necropsy revealed a hemorrhagic, ulcerative, *gonorrheal cystitis,* a phlegmonous para- and pericystitis, which had distributed itself to the retroperitoneal tissue, besides there was also a fibrinous peritonitis.

In the lower portion of the dorsal cord there was a moderately marked *myelomeningitis,* that is, a *leukomyelitis* to the longitudinal extent of several vertebræ. The "vesicular" condition of the diseased area (Fig. 168) is easily noted; with greater magnification the broadened transverse sections of the swollen axis cylinders and a few granular cells may be seen. The pia reveals great cellular infiltration, however, there are no vascular changes, nor bacteria. It could not be decided whether the myelitis, in this case, was due to a toxic metastasis or to a propagation of the gonor-

rheal inflammation, from the bladder to the spinal cord (by contiguity) as an ascending neuritis; in favor of the latter view are the phlegmonous pericystitis and the retroperitonitis (compare *Zeitschr. f. klin. Med.*, 1892, Bd. XXI).

The products of decomposition of the nervous tissue sometimes have the appearance of *corpora amylacea.* The destroyed masses of the medullary substance are taken up by the granular cells and removed; these cells are found

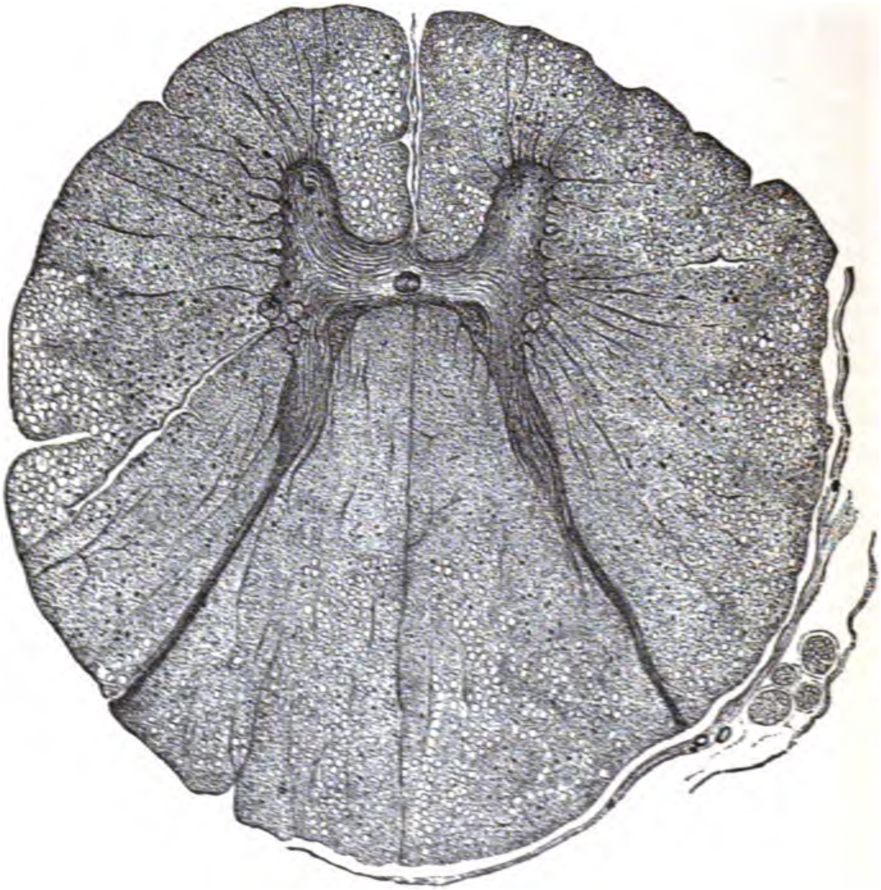

FIG. 161.—INFLAMMATION OF THE DORSAL CORD (VESICULAR CONDITION).

in every inflammation and also in non-inflammatory spinal cord softening, for instance, after tying the abdominal aorta (Stenon's experiment). We shall not discuss the point whether these granular cells, which serve as transporters, consist of leukocytes (wandering cells) or of fixed connective tissue cells, that is, products of transformation of glia cells (Senator).

In place of the destroyed parenchyma (in conformity with Weigert's law) a *substitute proliferation of the interstitial connective tissue* takes place. At the onset, the vesicular stage, the glia meshes appear dilated or irregularly lengthened, due to the swollen and destroyed medullary sheaths and

axis cylinders. The *glia cells* and *fibers* swell, especially in the vicinity of the vesicular spaces, with moderate increase of their nuclei. The protoplasm of the glia cells may further also lose its structure and their processes may undergo varicose enlargement.

In the stage of *softening* the glia stroma may finally break down, so that only a detritus of granular cells, medullary globules, fat drops, red blood corpuscles, leukocytes, pigment layers, as well as destroyed axis cylinders and medullary sheaths, remain. These fragment masses are taken up by the granular cells and are removed. To this a proliferation of the interstitial tissue is added, which begins in the periphery of the focus of softening, gradually proceeding toward the center and finally by retraction leads to a "*sclerotic cicatrix.*" Here there are often hyperplastic connective tissue cells having numerous processes, "spider cells or Deiter's cells"; further atrophic ganglion cells and nerve fibers, as well as degenerated vessels; occasionally a cyst-like space remains in the center of the sclerotic focus.

Sometimes a few axis cylinders (in multiple sclerosis) in the fibrous cicatricial tissue may retain the power of conduction and thereby regain their functional activity. Whether besides this form of restitution a *regeneration of ganglion cells and nerve fibers* takes place, is very questionable. In the lower animals a regeneration of the central nervous tissue has been demonstrated by the formation of new fibers with certainty. Fickler (*Deutsche Zeitschr. f. Nervenhk.*, 1899, Bd. XVI), maintained that the nerve fibers of the spinal cord, also in man, are capable of regeneration even up to a complete recuperation of function, so long as the blood vessels of the cord are still intact. Fickler, in a case of compression of the cord, describes these newly formed fibers which were in connection with ganglion cells below the area of compression.

The lesions of the *vascular apparatus* are sometimes only trivial in myelitis, in other cases these may dominate the anatomical picture. In pure, toxic, myelitis the inflammation is said to be entirely limited to the parenchyma, while in the infectious form the hemorrhagic, that is, the exudative variety predominates.

The *circulatory system* reveals the following lesions: congestive hyperemia, vascular dilatation, embolism, thrombosis, and rupture of the medullary vessels with capillary or even larger hemorrhages. *Hemorrhagic myelitis* can not be positively differentiated, macroscopically from hematomyelia. In connection with inflammatory spinal cord hemorrhages, as in the brain, there is serous infiltration of the surrounding medullary substance and further on a colliquative necrosis. The extravasated blood is absorbed, larger or smaller portions of the inflammatory focus undergo fatty degeneration and are transformed into yellowish masses (yellow softening). Then there is resorption of the fatty products of degeneration and further a proliferation of the neuroglia tissue with succeeding cicatrization. Included in the contracted cicatricial tissue, finally we find as the residue of former hemorrhages, either amorphous pigment or hematoidin crystals (compare Plate I, Fig. 3 *a*, Explanation of the plate).

Fig. 3 *a*, Microscopic picture of the softened masses (magnified 300 times) from a case of *hematomyelia during confinement* (published by E. v. Leyden, *Zeitschr. f. klin. Med.*, Bd. XIII, p. 225). The patient, Mrs. K., aged 28, during the first days of the

puerperium developed total sensory and motor paralysis of the legs with paralysis of the sphincters, loss of the tendon reflexes, girdle pain and bed-sores. The patient died of sepsis upon the 51st day of the disease. At the autopsy, there was found in the lower thoracic cord, a soft, pappy, almost fluctuating, focus of softening 5 cm. long. Histologic examination revealed that this soft, yellowish mass consisted of the following elements: (*1*) *Numerous large globules and granular cells;* (*2*) *fragments of nerve fibers with myelin drops and swollen, partly granular and fatty degenerated axis cylinders;* (*3*) *a few red corpuscles;* (*4*) *numerous, rhombic hematoidin crystals;* (*5*) *large cells with granular, rusty colored contents* (*pigment granules*)*;* (*6*) *medullated nerve fibers, the axis cylinders of which are stained yellowish brown and reddish brown* (*hematoidin*), while the medullary sheaths remain unstained.

Fig. 3 *b–g, Sections through the spinal cord;* the green color is due to the chromic stain. Abbreviations: *s. D.* = secondary degeneration (ascending and descending); *R. d.* = border degeneration; *H.* = hemorrhagic focus (Fig. 3 *b, c, d,* transverse section above the focus [cervical and thoracic cord], *e, f, g* = transverse section below the focus [lumbar cord]). In the cervical and thoracic cord the ascending degeneration of the posterior columns is recognized from the lighter color; in the vicinity of the focus of softening the posterior columns are attacked *in toto,* upward the degeneration passes to Goll's columns. Downward from the focus of destruction, in the latero-posterior columns, traces of descending degeneration can be demonstrated. The border degeneration is very marked above as well as below the focus. Further, a hemorrhage is noted passing through the entire cord, from the left Goll's column in the cervical cord to the posterior column in the lumbar cord, which originated from the focus of softening (*central tube hemorrhage*) and which has distributed itself in the vault of the posterior columns, upward and downward. The white medullary mass has suffered more than the gray.

Of further lesions in the vessels, apart from the congestive hyperemia of the initial stage, *infiltration and inflammation of the vascular wall* itself is noteworthy. This is characterized by small cell infiltration of the vessel sheaths, further by serous transudation (inflammatory edema), or leukocytic exudation into the surrounding medullary tissue. Further hyaline degeneration and finally even obliteration of the vessels may take place; then by ischemic processes either a necrotic inflammation or a cuneiform focus of softening develops, the apex being directed toward the center of the transverse section of the cord.

It must, however, be emphasized, that this perivascular leukocyte infiltration may not take place and that there are inflammations of the cord in which also the other circulatory disturbances are absent and only an (acute) degeneration of the nerve parenchyma can be demonstrated. Lubarsch regarded these cases as degenerative inflammations, in contrast to the exudative forms, which run their course with small cell infiltration.

The *central canal* is sometimes dilated in myelitis, occasionally even filled with a fibrino-purulent exudate. In central myelitis the vascular lesions and the nuclear increase are most marked in the commissures.

If the inflammation begins in the white substance of the cord (*leukomyelitis*) there are usually lesions in the membranes of the cord. A primary meningitis may distribute itself along the vessels or the connective tissue septa, to the substance of the cord (border myelitis, perimyelitis, *meningomyelitis*). In this complication the meningeal vessels are dilated, sometimes thrombosed, the pia mater showing small cell infiltration, thickened, being turbid and adherent to the medullary substance. Not rarely hemorrhages arise from the inflammatorily altered pia blood vessels.

Myelitis may, especially in disease of the cervical and lumbar enlargements, attack the *nerve roots* and even involve their extraspinal continuations; in the latter we recognize histologically, swelling of the axis cylinders, destruction of the medullary sheaths and proliferation of the interstitial tissue, particularly around the vessels. These degenerative and neuritic lesions, in disease of the anterior horns, for example in poliomyelitis anterior, follow the affected nerve to its terminal distribution in the muscle and this structure presents atrophy.

The bacteria found in the cerebrospinal fluid in myelitis will be described later on.

The pathologic lesions that have been described may occupy the entire transverse section in focal myelitis, or may be limited to a special area. Small foci usually do not show softening or only to a very slight extent. If the areas in the vicinity of the myelitic focus are microscopically investigated, we will find that the pathologic change always extends further than can be observed with the naked eye. To these, after myelitis has existed for a few weeks, *Türck's secondary degeneration* is added, which distributes itself upward in the sensory, and downward in the motor, fiber systems.

Descending degeneration takes place in the motor tracts, such as the pyramidal-anterior and lateral column tracts, and those from the cerebellum, the optic thalamus, the corpora quadrigemina and the red nucleus (v. Monakow's rubrospinal tract) and in the descending conduction tracts in the anterior and lateral columns. *Ascending degeneration,* on the other hand, occurs in the sensory conduction tracts, and especially all posterior column tracts (including Lissauer's zone), whose cells of origin are to be sought in the extradural spinal ganglia; further the cerebellar lateral column tracts, whose trophic center is situated in Clarke's columns, finally Gowers' bundle and a part of the anterior column bundle. Only exceptionally is a retrogressive degeneration observed in the motor and sensory tracts opposite to the direction of conduction, which at most only amounts to a few centimeters. An idea of the altered form and distribution at various heights of the cord, of ascending and descending degeneration, is shown by the illustrations on Plate I, Figs, 1, 2, and 3 (compare also Fig. 168). The form thus described corresponds to *focal (transverse) myelitis,* the first and most typical variety of spinal cord inflammation.

The *second form* in which myelitis appears is *acute disseminated* or *multiple myelitis,* in which a number of inflammatory foci are distributed throughout the cord. This has much similarity to multiple sclerosis; the latter may be regarded as a frequent termination of acute disseminated myelitis, such as occurs especially after acute infectious diseases. We are justified in assuming that sclerosis may originate from a spinal cord disease of long standing, and for this reason the condition belongs to chronic myelitis; according to Schmaus's recent investigation multiple sclerosis cannot be denied an inflammatory character.

The individual foci of disseminated myelitis, as a rule, have but moderate size and extent, they rarely include more than half a transverse section of the cord and one half the height of a vertebra. Their distribution varies greatly; sometimes they are found in several small "plaques" in the vicinity of a larger focus. In other cases they are found all through the cord, even

up to the medulla oblongata, to the pons, mid-brain and brain. Generally their number is greater in the upper part of the spinal cord (cervical enlargement); rarely are the foci more numerous in the mid-brain and *bulbus* (symptoms of bulbar paralysis).

Besides focal and disseminated myelitis we also recognize a *third form, inflammation of the gray substance* (*poliomyelitis*), which from its localization and symptoms differs materially from the two others. It presents the anatomical condition of "essential infantile paralysis" first described by Heine (1840), as well as of "essential atrophic paralysis of adults" (Duchenne); the clinical picture was accurately investigated by Roth (Basle), L. Clarke, Hayem, Charcot, and Kussmaul, the last author naming the disease anterior poliomyelitis; Leyden in a number of rapidly fatal cases of infantile paralysis was able to demonstrate the inflammatory character by discovering cellular infiltration in the anterior horns (*Westphal's Archiv,* 1864). Multiple myelitis and poliomyelitis present such a totally different clinical picture from transverse myelitis that they require a special description. (This will be found in another chapter of this book.)

The following questions are justified from the description of the pathology of myelitis:

(1) *From which tissue elements does myelitis take its starting point?*

(2) How do we recognize that we are dealing with *an inflammatory process?*

(3) *How do we differentiate myelitis from other degenerations or from changes which occur in connection with hemorrhages or compression of the spinal cord?*

The answer to these questions is quite indefinite. Thus the *starting point of the inflammation* was referred in part to the *ganglion cells* (Charcot, Rissler, Kahlden),[1] partly to the *circulatory apparatus* (P. Marie, Schmaus, Mayer), and partly in the *glia tissue* (Marinesco, Goldscheider). Accordingly a *parenchymatous, vascular and interstitial* form of myelitis was recognized. In our opinion one and the same inflammatory irritant may attack the ganglion cells and the nerve fibers, at one time or the neuroglia, that is the interstitial tissue, or the blood vessels with greater severity and correspondingly lead to a predominance of parenchymatous, interstitial or vascular lesions; all three are, however, so coördinated as in inflammations of other organs, for example, of the heart or of the kidney, in which the inflammation is not exclusively limited to the interstitial tissue, that is to the parenchyma alone, but constantly attacks both, although not always to the same extent. In this the circumstance must be included that in myelitis an irritation to the supporting structure and upon the walls of the vessels acts differently than upon the specific nervous tissue; while the first react to irritation with increased activity—hyperemia, exudation and cell segmentation—the specific nervous elements are destroyed as soon as the irritation has attained a certain degree (Rissler).

Nor can the boundary between *acute degeneration* and *inflammation of*

[1] An accurate review of the literature will be found in Redlich's article *Centralbl. f. allgem. Pathologie und pathol. Anatomie*, 1898, Bd. IX, Heft 3 u. 4, and in W. Mayer's article "Myelitis acuta" in the "Arbeiten aus dem Institut für Anatomie u. Physiologie des Centralnervensystems," 1900, Heft VII.

nerve fibers and ganglion cells be sharply defined. According to Ziegler's definition, inflammation is to be regarded as a primary, degenerative tissue lesion which is only secondarily succeeded by changes of the vessel walls and further on by exudative processes. Thus, in disseminated myelitis Kahlden was able to discern foci of varying intensity and of different ages. The initial signs are exclusively marked by degenerative cell changes; then comes destruction of the axis cylinders, and finally the lesions of the vascular apparatus. Also in myelitis experimentally produced in animals, by the injection of bacteria or their toxins, all grades of inflammation were found, from simple degeneration to hemorrhagic softening.

While according to this view the different changes of the individual tissue constituents in myelitis are coördinated and only represent varying forms of the same fundamental process, other authors regard as the essential signs of inflammation the "*inflammatory circulatory disturbance*" (Schmaus). This consists of congestive hyperemia, extravasation of blood, increased lymph transudation, and migration of leukocytes, which especially infiltrate the adventitia and the perivascular lymph spaces. Finally the walls of the vessels may become thickened and show hyaline degeneration. A recent author, Douglas Singer, even refers the majority of cases of myelitis, not to inflammation, but to a thrombosis of the spinal arteries and necrosis in their area of distribution.

The view of the vascular character of myelitis in our opinion is only proper in infectious or toxic myelitis which arises through the circulation, especially in poliomyelitis, as well as in the disseminated forms, in which the inflammatory changes of the vascular apparatus are particularly prominent. In a similar manner primary disease of the walls of the vessels (syphilitic endarteritis, arteriosclerosis, thrombosis), further emboli in the region of the spinal cord arterioles, terminal arteries (Adamkiewicz, Kadyi), lead to nutritive disturbances and multiple degeneration.

In contrast to these there are undoubtedly myelitides, even of the most acute type, in which the vascular lesions are secondary. On the other hand, the previously described lesions of the ganglion cells and nerve fibers, as well as of the neuroglia, further the epithelioid and fatty granular cells, can not be regarded as absolutely characteristic of spinal cord inflammation, as these are also observed in other non-inflammatory degenerations; even inflammatory degeneration can not be differentiated from the descending form. Further in disseminated myelitis one focus may only present the signs of acute degeneration of the nerve fibers and ganglion cells, while another focus shows interstitial inflammation. In this condition, as Obersteiner and Redlich quite justly remark, there are only quantitative differences, so that a positive differentiation of acute inflammation from acute degeneration is impossible. For this reason Virchow's opinion is also applicable to myelitis, that inflammation is *not a uniform process* presenting constant factors.

Finally the differentiation of *primary spinal cord hemorrhage* from hemorrhagic myelitis is very difficult especially in old foci. Ollivier separated hemorrhages in the spinal cord (*hematomyelia*) from true acute myelitis. His reports are based upon the results of autopsy, while the clinical observation of the cases was not always clear in regard to their development and course. According to Leyden's investigation and also to our present experiences,

primary hemorrhages of the spinal cord are exceedingly rare, excepting those due to concussion. Post-infectious myelitis, in the transverse section, may present itself as hematomyelia. Thus Leyden saw a case of myelitis of this kind after the patient had suffered from influenza, the disease rapidly terminating fatally on account of the ascent of the process to the medulla oblongata which gave rise to respiratory paralysis. Almost throughout the entire length of the spinal cord numerous smaller and larger hemorrhages, in part between the pia and medullary substance and in part around the spinal cord vessels, were found. Careful investigation, however, unquestionably revealed the signs of an infectious myelitis (Plate I, Fig. 1).

On the other hand, after serious traumatic lesions, a disease of the spinal cord with a rapidly fatal course may appear, in which at the post mortem hardly the traces of an effusion of blood will be noted, but, on the other hand, the distinct signs of myelitis. Leyden saw this condition in a case of compression of the cervical cord due to a detached odontoid process in a man, who broke his neck from the results of a fall. It has also been demonstrated in other organs, that in connection with traumatic hemorrhage, inflammatory lesions may appear.

Larger effusions of blood, analogous to apoplectic cerebral foci, are rare in the spinal cord. Such conditions have almost always been seen only after rupture of the hemorrhage from the pons or the medulla into the fourth ventricle. In regard to the distribution of hemorrhage in the spinal cord, the blood gradually forces itself to the point of least resistance, and this is the top of the posterior columns. In this region the hemorrhage may take the shape of a red thread, which traverses the cord in this area in a longitudinal manner. Leyden has reported a case of this kind (*Zeitschr. f. klin. Med.*, Bd. XIII). This was one of secondary dorsal myelitis occurring during the puerperium (compare Plate I, Fig. 2).

Such observations lead us to the conclusion that anatomically a sharp separation between primary hemorrhage with subsequent myelitis from primary hemorrhagic myelitis is only possible in the rarest cases, so that this differentiation can not be maintained for clinico-practical purposes.

The conditions are similar with another form of spinal cord softening, namely, *compression softening*, that is the much discussed *compression myelitis*, which H. Oppenheim regards as more frequent than primary myelitis. Several authors have separated this form from true myelitis and have designated it as *stasis edema of the spinal cord.* Leyden has included compression softening with myelitis on account of the similarity of the clinical picture. Prominent histologists, such as Schmaus, also consider it identical with myelitis. In pressure paralysis we must, however, differentiate two processes:

(*a*) *Pure compression* from tumors, which, as a rule, are situated within the spinal canal, forming upon the dura or pia mater (endotheliomata, fibrosarcomata, fibromata). They may compress the spinal cord to a narrow band and occlusion of the vessels produces ischemic processes, in which inflammatory lesions hardly arise. This is simple compression of the spinal cord, which, as is well known, may be relieved by surgical measures. On the other hand

(*b*) In compression due to caries of the vertebræ the inflammation may pass to the membranes of the cord (*pachymeningitis tuberculosa, epidural*

exudates and fungous proliferations) and may even permeate the cord itself. Secondary myelitis also occurs after fracture of the vertebræ, causing compression and hemorrhage to which Fr. Kraus has particularly called attention (*Zeitschr. f. klin. Med.*, Bd. XVIII, p. 360). The compressed medullary substance is often greatly softened and contains numerous granular cells. Reactive inflammatory changes arise in the immediate vicinity of the focus of compression and beyond these Türck's ascending and descending degeneration. Schmaus quite correctly emphasizes, in his recent article upon myelitis (*Deutsche Zeitschr. f. Nervenheilkunde*, Bd. XXVI, 1904), that the pressure upon the spinal cord may lead, not to stasis edema, but to true, secondary inflammation.

Also according to Schmaus, shock or compression of the cord, as well as hemorrhage, may give rise to distinct softening, to which secondarily an inflammation, with leukocytic and granular cell infiltration, may be added, entirely identical with the anatomical picture of genuine myelitis.

From a clinical standpoint we also maintain that a sufficiently distinct separation between compression softening and myelitic softening cannot be substantiated, that, therefore, *pressure paralyses of the cord may be included with myelitis*, as well as that we cannot, clinically or anatomically, separate hemorrhagic softening from true myelitis.

Syphilitic myelitis also presents peculiarities. It occasionally arises in connection with syphilitic *meningitis* or a *gummatous nodule*; according to Erb, the apparently simple, primary degenerations and atrophies (sclerosis, column degeneration, inflammation) may be of syphilogenous origin. Syphilitic myelitis often arises in small foci, which occur in connection with luetic *vascular disease* appearing unilaterally or bilaterally, solitary or multiple (Fig. 162).

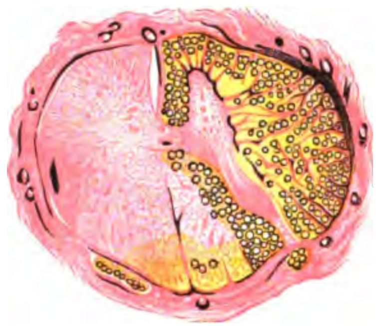

FIG. 162.—SYPHILITIC MYELITIS OF THE LUMBAR ENLARGEMENT, SHOWING CICATRICIAL ATROPHY AND SCLEROSIS, RIGHT HALF (RELAPSE) IN THE STAGE OF SUBACUTE INFLAMMATION (VESICULAR STAGE).

As an example of this form, a case reported by Leyden may serve (Charité—Annalen, III. Jahrg.). The course of the disease as well as the anatomical investigation permitted the recognition of three periods of development. It was the case of Mrs. Emma K., æt. 30, who 15 years previously had passed through a luetic infection. Three years ago she was attacked with acute paraplegia with thoracic girdle sensation, and pain in both legs which rapidly emaciated. This condition, in the next few months, improved to such an extent, that the patient was able to take long walks. A relapse suddenly appeared 2 years later, the left leg being completely paralyzed; after treatment for 2 months incomplete restitution occurred, which, however, became aggravated after a prolonged journey and was succeeded by a complete, flaccid, atrophic paralysis of both legs with sensory disturbances, loss of reflexes and paralysis of the sphincters (2d relapse). The patient died 4 months later, the result of sepsis which took its starting point from a bed-sore.

Corresponding to the 3 "relapses" of the clinical course, the histologic examination of the myelitic focus, which affected the lumbar enlargement, revealed 3 *stages of development*. As the first a *fibrous cicatrix* at the periphery of the left lateral column may be regarded; in this cicatricial tissue not a trace of nerve fiber could be found,

on the other hand, a *completely obliterated* arterial vessel thickly permeated with yellow pigment granulations could be seen. We are justified in regarding this process as the starting point of softening in consequence of an *arteriitis obliterans syphilitica.* The remaining portion of the left, completely sclerosed and atrophic half of the cord, corresponds to the second period of the disease, while the right half was in a condition of subacute inflammation (vesicular stage) and coincides with the second relapse, which occurred 4 months prior to death. The remainder of the spinal cord revealed ascending and descending degeneration; the pia mater thickened over the extent of the focus, was rich in cells and adherent.

The foci which only attack *one-half of the cord* are also of great interest. Fig. 163 shows the transverse section of a syphilitic myelitis with preponderant destruction of the left half. Unilateral disease leads to the form first described and named after Brown-Séquard; this is rarely of inflammatory origin (unilateral myelitis), it is usually the consequence of a unilateral division of the cord (from a stab wound in the cord or the like).

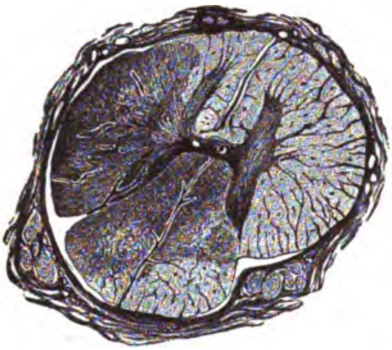

Fig. 163.—Syphilitic Myelitis, Predominant Destruction (Dark) of the Left Lateral and Posterior Columns, Partial Destruction of the Right Posterior Column.

A few words must be devoted to a quite rare, but nevertheless remarkable form of myelitis, **caisson paralysis.** This appears in persons working under high atmospheric pressure (in building tunnels and bridges), when they come at once from the caisson filled with compressed air into the open air. Under the influence of this rapid lowering of pressure, various disturbances arise in the internal organs, among which the lesions in the nervous system are of particular interest. Such observations have been reported by v. Schrötter, Mayer and Heller in their "Handbuch der Caissonkrankheiten." Besides cerebral paralysis, spinal paralysis of the more or less complete paraplegic type is noted. The sudden appearance of these paralyses may be referred to rupture of the cord substance in consequence of the exit of air from the blood vessels, with or without extravasation of blood, accordingly to traumatic influences; from the embolic occlusion of the air vesicles of the arterioles of the spinal cord, circumscribed necroses may appear. The autopsy reveals circumscribed foci with the typical lesions of myelitis.

We, therefore, do not err if we include, clinically, these various forms of spinal cord lesions and designate them myelitis. Clinical description will entirely justify this procedure. The etiology will often enough present points of support to render a differential diagnosis of the various forms possible.

THE CLINICAL PICTURE OF ACUTE MYELITIS

The starting point of the clinical diagnosis of acute myelitis is always formed from the more or less rapid and complete development of *paraplegia.* This type has been known for a long time. First described as the result of injury to the vertebral column and experimentally produced in animals

by division of the spinal cord, it was subsequently observed as a spontaneous disease. In the most developed cases there is a complete functional separation of the upper and lower body portions, so that the latter are entirely beyond control of the will and devoid of sensation. Although we adhere to this type of myelitic transverse paralysis there are nevertheless manifold differences; thus the degree of the paraplegic paralysis may vary in an equal or unequal implication of the lower extremity, according to the *height, the transverse extent, and intensity* of the pathologic process. The clinical picture of myelitis is especially modified according to the involvement of the central gray, and the peripheral white, substance; it further depends upon the implication of the anterior or posterior horns, as well as of the motor and sensory fiber columns, upon one or both sides. For this reason symptoms may appear singly or in combination in myelitis, conditions which are also observed in other diseases of the cord, for example, spastic spinal paralysis.

In general the inflammation attacks both sides, not rarely even the entire transverse section of the cord; this is easily understood from the narrow extent of the cord; hardly thicker than the circumference of the little finger, only exceptionally may one-half be affected (unilateral myelitis). For this reason—in contrast to cerebral monoplegia and hemiplegia—*motor and sensory paraplegia* combined with paralysis of the sphincters remains the type of spinal cord paralysis.

Before proceeding to the special description of the clinical pictures, according to the region of the cord involved, it appears advisable to enumerate briefly the common features of transverse myelitis.

Transverse myelitis in its typical form, no matter at what height it is situated, produces the following symptoms:

(1) In the muscles supplied by the cord segments below the lesion there is, as a rule, *spastic paralysis* without disturbance of electric contractility, frequently, but not always, with increased reflexes (descending pyramidal tract degeneration);

(2) In the muscle regions of the diseased spinal cord segments, flaccid paralysis with DeR and atrophy of the muscles, as well as loss of reflex contractility;

(3) In the area of innervation of the diseased, as well as of the entire spinal cord below, *anesthesia* of the skin and also of the deeper parts (muscles, bones, joints). To this anesthetic or hypesthetic zone there is often an adjoining narrow, hyperesthetic band (girdle pain, see Fig. 166);

(4) Bladder and rectal disturbances;

(5) Vasomotor (edema, cyanosis) and trophic disturbances (bed sore, cystitis).

Transverse myelitis most often affects the *dorsal cord,* which represents the longest and simultaneously the narrowest portion of the cord; for this reason disease of the entire transverse section in the dorsal cord is much more frequent than in other portions.

The transverse size of the section of the dorsal cord, according to Stilling, amounts to 28.7–36.2 mm., while in the cervical cord it varies between 44.7 and 62.49 mm.; in the lumber cord between 35.2 and 42.9 and in the three upper sacral segments from 22.4 to 8.6 mm. (measured in a child). To this must be added the circumstance that the dorsal cord is situated in the long-

est and most exposed portion of the vertebral column. The dorsal cord externally corresponds to a line from the *seventh* cervical spinous process to the *tenth* thoracic vertebra.

We shall, therefore, describe

THE CLINICAL PICTURE OF DORSAL MYELITIS

somewhat minutely and in this connection the variations in disease of other portions of the cord, particularly emphasizing segmental localization of the spinal functions.

We may differentiate *four stages* in the course of myelitis:

(1) The *prodromal stage;*

(2) The *stage of paralysis,* in which the motor and sensory paralyses, ascending and descending secondary degenerations and the degeneration in the anterior horns take place to the fullest extent depriving the muscles and nerves of their nutrition (*neuroparalytic stage* according to Goltz).

In connection with these is,

(3) The *regenerative stage* and finally

(4) The *stage of arrest* (*stationary stage, sequels*).

I. THE PRODROMAL STAGE

The signs which signalize a threatening myelitis, apart from preceding trauma or infection, are in part sensory, in part of motor kind. The patient drags the member attacked by paralysis, and has a permanent or transitory sensation of leaden weight, muscular tremor, of stiffness and of rapid exhaustion; the legs temporarily refuse to function and there is a "kink" in one or the other knee-joint. Among the sensory symptoms there are various disturbances of sensation, formication, circumscribed anesthesia and dysthesia, further a tormenting girdle sensation and lightning-like pains, which appear particularly at night and are often combined with involuntary muscular contractions. These pains can not be referred to disease of the cord, for this structure, like the brain, is but little or not at all sensitive; on the contrary they indicate an implication of the membranes of the cord or of the nerve roots. Myelitis not infrequently begins with *crises-like gastric pains,* nausea and vomiting. Sometimes even in the prodromal stage of myelitis pain or symptoms of retention appear on the part of the *bladder and rectum.*

Often *fever and chills* open the scene, especially in inflammation of the cord the result of acute infectious diseases, for example, enteric fever, influenza, gonorrhea, malaria; rise in temperature is noted especially often in acute inflammation of the anterior horns (poliomyelitis) and in acute disseminated myelitis. The height of the fever varies, in some cases it rises to 104° F.; usually, however, the rise is moderate and in a few days drops to normal.

The appearance of fever and chills in the further course of myelitis indicates a complication, either of *sepsis* (usually originating from a bed-sore or from cystopyelitis), or *hypostatic pneumonia* or a disturbance of the *function of the bowels,* more rarely a *complication on the part of the cord* (meningitis, abscess of the cord).

The symptoms that have been described, in varying degrees and in manifold arrangement, constitute the prodromal stage, and according to the rapidity with which myelitis develops, may be of briefer or longer duration. Sometimes the disease appears in the midst of health; in a few hours the signs of paralysis attain such a grade that the patient almost collapses. Such paraplegias and anesthesias with sudden onset resemble in their appearance spontaneous apoplexy of the cord; there may even be a combination of both processes, a hemorrhage into a focus which up to that time has been latent. As a rule, however, in these forms of myelitis with an apoplectiform onset, there are symptoms in the history, in contrast to primary hemorrhage of the cord, such as have been described previously, to which but little attention had been given. Further the paralysis does not advance in primary hematomyelia, on the contrary—the pressure symptoms, the edema of the cord improve, and there remain, similarly as in cerebral hemorrhage, only the symptoms due to the anatomical destruction of the cord.

What in this *myelitis acutissima s. apoplectica* is the work of destruction of but a few hours, in the acute variety requires a few days, in the subacute form a few weeks, and in chronic myelitis an insidious development of a few months. This principle of development proposed by Leyden [1] for the 4 forms only refers to the onset and the rapidity of development of myelitis, however, not to the further course. The disease varies in regard to the rapidity of its onset; when the paralysis has reached its acme it is usually chronic, lasting for months or years, excepting those rare cases which terminate fatally during the early period.

The *apoplectiform development* has been observed especially in central hemorrhagic myelitis, which preferably attacks the gray substance. Caisson paralysis also develops acutely; the patient coming out of coma in a paraplegic condition.

In the *subacute* and *chronic forms* the signs of paralysis are sometimes not uniform, but are paroxysmal, presenting remissions and intermissions; the paralysis may even improve until a relapse brings out the entire clinical picture (*myelite à rechutes*). This *paroxysmal course* occurs especially in metasyphilitic inflammations of the cord (compare the previously described history). In another case of this kind under our observation the paresis of the left leg developed suddenly to a paraparesis, which again improved under mercurial treatment. After some time paralysis reappeared and finally led to complete paralysis of both legs and was followed by paresis of the left arm.

A *beginning myelitis* can, therefore, not be taken too seriously and particularly the transitory improvements in its course are by no means a source of unalloyed joy. During the entire course of the malady a new attack may occur, especially as the spinal cord is much more sensitive than the brain and an intercurrent disease, or carelessness on the part of the patient, or in the treatment, may readily rekindle the inflammatory process.

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[1] Article upon myelitis, *Verhandlung des Congresses für innere Medicin in Berlin*, XIX. Bd., 1901.

2. THE STAGE OF PARALYSIS

Dorsal myelitis produces paralysis of both legs, the muscles of the trunk up to the height of the pathologic focus, as well as of the bladder and the rectum.

In the *most marked cases* the paralysis is complete and implicates all of the sensory and motor qualities: the legs no longer obey the impulse of the will, they are anesthetic, immotile and only by dysesthesia, pain and involuntary contractions do they show signs of life. The sphincters are insufficient, cystitis and bed-sores appear, the consequences of which (pyelonephritis, sepsis) are frequently sufficient to cause death. Fortunately these severe cases with fatal paralysis are rarer than those in which some movement is present.

Motion is usually earlier and more severely affected than sensation. Motor paralysis may even be complete, while sensation is but little or not at all involved. For this incongruence of sensory and motor paralysis at the present time we cannot give a well-founded explanation. The paralyzed muscles do not obey the will, but by no means are they always incapable of stimulation; on the contrary, in the majority of cases they reveal increased reflex excitability. The hypertonic muscles soon become rigid, probably a sign of a beginning secondary pyramidal degeneration; perhaps also a sign of an extrapyramidal, beginning regeneration. Only those muscles remain flaccidly paralyzed whose anterior horns are destroyed, while the others, centralized below the medullary disease, may even show increased tonus, increased reflex irritability and even spasm. The last named is sometimes spontaneous but more frequent after attempts at motion, for example, when the patient endeavors to sit up or after various reflex stimulations, stroking the sole of the foot or the popliteal space, from the effect of cold, shaking the bed and the like. The legs are spasmodically raised or drawn up with marked muscular tremor so that the heels approximate the buttocks, the toes being spasmodically flexed. In other cases the legs are alternately flexed and extended, which in children may jerk the limbs about (flying legs). This spasmodic condition is followed by painful muscular rigidity, which produces a cadaver-like rigid condition. The clonic spasms have been called by Brown-Séquard *spinal epilepsy.* It is the result of increased reflex irritability of the normal muscle. Contraction of the legs indicates the seat of the lesion above the lumbar cord; if this latter region is also attacked or destroyed, the reflexes disappear, the paralyzed legs are flaccid and do not react to external stimulation.

In the *medium severe cases* of inflammation of the thoracic cord, by a minute investigation of the power of movement, the implicated muscle groups may be determined and we may thus inform ourselves in regard to the height and transverse distribution of the lesion in the cord (see the section on regional diagnosis). Most frequently slight flexure movements are retained in the toes, ankle, knee, and hip joints; but it is most difficult actively to raise the extended leg from its support. Attempts at movement are slow and tiring, the legs appear heavy as lead to the patient and he is soon exhausted.

In *milder cases* the patients are still able to walk with the support of a cane or even without, the legs being held stiff like stilts. Raising the tip of the foot is difficult, so that a drawing of the trail of the foot shows an almost continuous but irregular path. This spastic gait renders the ascent of stairs

very difficult as well as jumping and rapid walking; tremor soon appears, the patient is quickly exhausted (myasthenic symptoms), he stumbles and threatens to fall; sometimes one or the other leg fails and the patient actually falls. The sensory symptoms in these milder cases are actually slight but on the other hand there are frequently pains in the distribution of the sciatic nerve. The bladder is almost always affected.

A common outcome of *spastic paralysis* is in contracture, which in contrast to hemiplegic extensor contracture may also induce a flexure position. The hip and knee joints may be flexed in such a manner that the heels approximate the buttocks; the knee joints are sometimes contracted in a position of adduction so that they bore into each other sufficiently to produce pressure ulcers and can only be held apart by producing active pain. The feet in consequence of contraction of the gastrocnemii assume a pes equinus position; the contracted muscles finally stand out like fibrous bands so that relief of the contracture is no longer possible by manual redressment but only by operative means.

Atrophy attacks the over-extended antagonists to a greater extent than the contracted muscles; according to the extent of the anterior horn lesion it may at one time attack this, at another time that, muscle to a greater extent, so that various deformities and contractures may develop. In the paralyses which are flaccid from the onset, from external mechanical influences paralytic contractures may result, for example, from pressure of the bed clothes upon the dorsum of the foot pes equinus may develop.

In dorsal myelitis, besides the muscles of the legs those of the *trunk and of the abdominal wall* may be involved. Paralysis of the intercostal muscles renders respiration difficult and occasionally leads to dyspnea; usually, however, the vicarious function of the diaphragm and the auxiliary muscles of respiration compensate for this defect.

A lesion of the 5th–12th dorsal segments paralyzes the abdominal muscles. *Paralysis of abdominal pressure* renders expiration and expectoration difficult, as well as contraction of the abdomen. Bronchitis, hypostatic pneumonia, hindrance of the act of vomiting, meteorism, constipation and the retention of urine are the consequences. Wagner and Stolper report a case, in which the muscles of the abdomen, in consequence of a partial injury of the lumbar cord were only paralyzed on the right side and were flaccidly protruded. Stolper even observed ileus-like symptoms after spinal cord hemorrhage. Paresis of the abdominal muscles may be noted from the inability of the patient to rise from the recumbent position unaided. The rectus abdominis may present segmentary paralysis; Kocher reports the case of a paraplegic, following dislocation and fracture of the 11th and 12th thoracic vertebræ, in whom the rectus abdominis did not contract below the umbilicus but responded well above this point.

When the inflammation attacks the upper thoracic cord from the second thoracic vertebra upward, then to the paralyses of the trunk and legs there are also added pareses of the flexors of the fingers, of the thumbs and the ball of the little finger, and of the interossei, as well as of the pronator quadratus. In disease of the first dorsal segment there are also oculopupillary symptoms (see cervical myelitis).

The behavior of the paralyzed muscles to the *electric current* is of great

diagnostic, prognostic and therapeutic import. Electricity in a certain respect is a test for the condition of the cord and of the muscles. A permanent, normal, electric reaction indicates that profound damage of the muscles and nerves, that is of the anterior horns which innervate them, is extremely unlikely. In general, therefore, with the lesion high, the electric contractility of the lower members is retained. Only in lesions of the lumbar cord and of the cauda equina, may electric alterations appear, and especially after destruction of the gray anterior horns. The appearance of DeR indicates a profound destructive lesion, with degeneration of the muscle and nerves, as may be readily determined macroscopically.

As is well known complete *DeR* consist in the loss of *electric response* of the nerve and of faradic contractility of the muscle; contraction of the muscle to galvanism is all that is retained, and at first may even be increased. This increase of galvanic irritability is often accompanied by an increase of mechanical irritability; it is not to be confounded with the increase, which is regarded as a sign of general heightened irritability occurring with both currents in spastic, cerebral, and spinal paralyses. Besides, an alteration in the form of contraction and in the law of contraction is characteristic of the DeR; the first is sluggish, crawling, the latter shows an increase of the AnCC over the CaCC.

DeR may take place during the first weeks of myelitis and points with great likelihood to the arrest of the trophic influence of the anterior horn cells as well as to a severe course for the muscular paralysis. In favorable cases the increase of galvanic irritability gradually decreases, the formula of contraction again becomes normal and faradic contractility reappears. In the unfavorable cases the contractions remain sluggish and the electric formula transposed; galvanic contractility may gradually decrease until in the terminal stage of atrophy, neither from nerve nor from muscle, by the strongest faradic or galvanic currents, can a contraction be obtained. It is self-evident that complete DeR in myelitic paralysis only occurs in those muscle bundles the related anterior horns of which are destroyed. If a portion of the anterior horn is unaffected, the muscle fibers pertaining to it are retained, so that a muscle in one fiber bundle may show normal electric reaction and in another DeR, as may be the case also in spinal progressive muscular atrophy (Duchenne-Aran type).

Simple, quantitative decrease of electric contractility with a normal electric formula is less unfavorable; this condition is particularly observed in *inactivity atrophy*, as in the late stage of those paraplegias, in which the lesion is above the leg center, and also above the lumbar cord.

The disturbances in the *sensory* field are even more varied than those of the motor. In the paralyzed lower extremities, sensation (conduction of sensory stimulation from consciousness) may be more or less reduced; and in all areas of distribution, upon one or both sides or even only in one part of a lower extremity, as upon the posterior surface of the legs or the sexual organs and the perineum, etc., these conditions may prevail. The individual sensory qualities (pain, cold, heat, vibration, and localization) may be involved to a varying extent. If the inflammation is limited to the central gray of the cord, pain is mostly absent, as the spinal cord substance in itself is insensible. Only when inflammation or edematous swelling is distributed to

the spinal membranes which are richly supplied with sensory nerves or to the posterior roots do pains appear. For this reason, in uncomplicated myelitis, in contrast to meningitis, the vertebral column is not held stiff nor is pain present upon movement or pressure.

Among the *sensory irritative symptoms* different forms of paresthesia appear in the paralyzed limbs. Some patients complain of numbness, tingling, furry sensation in the toes, others of tormenting heat and cold, burning, itching, of spasmodic sensations in the bladder and rectum. In the region of the spinal nerve roots sensations of this kind are experienced as girdle pains or as a painful constriction, which may even lead to respiratory difficulty. This girdle sensation may often be referred to an irritation of the nerve roots at the upper boundary of the lesion and then indicates its height. Externally this girdle sensation often shows the boundary of the anesthetic and normal cutaneous zone (see Fig. 173). Sometimes *pains are early symptoms of myelitis*, especially of syphilitic perimyelitis and meningomyelitis. The pains are aggravated at night and are sometimes unbearable; thus many a myelitic becomes a morphin habitué. The pain is especially severe in the legs, it is experienced as "shooting" sciatica, or as a feeling of constriction around the knee and ankle joints.

The anatomical cause of these pains may be sought in an irritation of the posterior nerve roots, either by an encroachment of the inflammation, by pressure of the tissue swelling, by the edema or inflammatory exudate, or from adhesion of the spinal membranes to the cord. This pressure is most decided upon the nerve roots in the case of disease of the vertebræ; the pain being most severe in *carcinoma of the vertebræ* (*paraplegia dolorosa*); they are not so intense in *spondylosis rhizomelique*, less, even absent, at least during rest, in spondylitis tuberculosa.

This pain may be produced or increased by pressure, palpation over the vertebræ, by bending forward or by jumping. Occasionally the localized vertebral pain, which is momentarily absent, may be provoked by passing a hot sponge over the region or by the contact of a vibrating tuning fork. According to an ingenious observation of W. Gull's the severe neuralgic pains, *in cancer of the vertebra*, are almost always experienced *close to the vertebral column*, while in caries of the vertebra they are situated laterally in the wall of the thorax. Here the patient notes a painful burning or tearing or a compressing girdle sensation about the thorax. In utilizing these sensations of pain, in a diagnosis of the height of the lesion, it must be remembered that the nerve roots which pass out of the cord, in the thoracic and lumbar regions, run obliquely from above downward. *The focus in the cord, therefore, is always higher than the point of exit of the nerve roots which belong to it in the canal of the vertebral column.*

In myelitis the internal organs may also give rise to pain; thus in *dorsal myelitis there are attacks of angina pectoris and gastric crises.* It is quite possible that the internal organs, in diseases of the spinal cord segments that supply them, react by sensory disturbances in a similar manner to the skin. Both are linked with nerve tracts, which was made clear by Head's investigations upon the hyperalgesic zones of irradiation of the skin in disease of the internal organs. Head refers the spinal innervation of the heart to the 1–3 thoracic segment, of the lung to the 1–5 thoracic segment, of the stomach

from the 6–9 thoracic segment. According to Kocher 1–4 dorsal segments send sympathetic nerves to the head and neck as well as to the heart and lungs; the 5–9 dorsal segments to the glands of the intestines and abdomen (splanchnic nerves), and the 10–12 dorsal segments to the testicles, the bladder and the rectum (inferior splanchnic nerves; internal spermatic plexus; inferior mesenteric plexus). These reports of the cord innervation of the heart, the stomach and the intestines, of the spinal course and the location of the conduction tracts for the intestine, require further investigation. Probably spinal vasomotor innervation plays an essential part in these intestinal reflex processes.

In addition to these attacks of pain there are also *hyperesthesia, hyperalgesia* and *spontaneous muscle contractions* in the paralyzed members. This may go so far that even slight contact or pressure may produce painful formication (Charcot's dysesthesia), and even contractions. The bones and muscles are often hyperesthetic. This hypersensitiveness, in contrast to the neuritic pains, is not in connection with the pressure points of the peripheral nerve trunks, but with the segment zones.

The combination of these spontaneous pains with objective anesthesia in the region of the paralyzed limbs is peculiar. This condition known as *anesthesia dolorosa* is referred to a process of irradiation due to a central irritation of the posterior roots, which have simultaneously become incapable of conduction for peripheral irritations. This may be due to compression of the posterior roots from masses of exudate or contracting cicatricial bands; the symptom-picture of *paraplegia dolorosa* is especially common in compression of the spinal cord due to carcinoma of the vertebra (Charcot, v. Leyden).

The appearance of sensory paralysis indicates a profound destruction of the cord. The extent of the sensory disturbance is of the greatest importance in regard to the height of the lesion in a dorsal myelitic focus because motor paralysis in all diseases of the dorsal cord, from the third segment downward, presents very nearly the same picture of paraplegia. *In dorsal myelitis anesthesia may extend from the tip of the toes to the clavicles*, varying in height, according to the location of the focus (compare Seiffer's diagram of sensory topography).

In *disease of the lowest dorsal cord segment* the anesthesia is limited to the legs, anteriorly it reaches to Poupart's ligaments, posteriorly to the height of the 5th lumbar vertebra. With each higher segment that is diseased the border of anesthesia rises. In disease of the *9th and 10th dorsal segments the anesthesia reaches to the umbilicus*, of the *8th and 7th dorsal segments to the line of the xyphoid*, of the *4th, 5th and 6th dorsal segments to the breasts and in disease of the 3d dorsal segment to the clavicles.* In an affection of the upper thoracic segments the anesthesia reaches posteriorly to the first thoracic vertebra; a hypesthetic line reaches to the axilla and to the ulnar inner surface of the arm including the ulnar half of the forearm and the second to the fourth finger, similar to the condition found in ulnar nerve paralysis.

As the dorsal segments are situated higher than the vertebræ which correspond to them, and as besides a completely anesthetic zone always points to an implication of adjacent segments, *the focus in the cord is always to be sought for higher than would correspond to the actual anesthetic zone.* Thus in total anesthesia of the mammæ (border limit of the 5th dorsal segment)

the medullary lesion is to be sought at the height of the 4th thoracic vertebra or even of the next higher vertebra.

The *lower boundary of the lesion* is very difficult to estimate, as the motor and sensory paralysis implicates the entire body below the focus. Only the retention of the reflex arc below the point of lesion, as well as the behavior of the paralyzed muscles and nerves upon electric testing, and the condition of the sphincters furnish points for the recognition of the lower boundary of the lesion.

Often enough the extent of the sensory disturbance does not correspond with the motor paralysis, because the inflammation may at one time attack the sensory, at another time the motor, portions in the cord more intensely. The height of the focus is naturally determined by the highest segment that is diseased. If the myelitic focus is oblique, or if the disease is more marked on one side of the cord, then also the leg upon the side of the more severely involved cord lesion shows the paralysis to a greater extent. In inflammation limited to only one-half of the cord Brown-Séquard's paralysis will be produced; this occurs more frequently in unilateral spinal cord injuries, for example from a stab wound or traumatic dislocation of the vertebræ. *Motor paralysis* (spastic paresis with increase of the tendon reflexes, arrest of the muscle sense, the tract running in the posterior column of the same side) *is situated upon the side of the injury, while sensory paralysis* (analgesia) *affects the opposite side,* as the sensory tracts first cross in the spinal cord.

These peculiar sensory disturbances are shown by a case under our observation. It was one of Brown-Séquard's paralysis that had existed for 25 years; remarkable besides the prolonged duration, for tormenting hyperesthesia and a spastic contracture of the lumbar muscles upon the paralyzed side, as well as for the favorable outcome (Figs. 164 and 165).

The case (I) was that of a carpenter, Paul H., æt. 50, who 25 years previously had suffered from a stab wound in the thoracic vertebral column. Immediately after this injury there was paralysis of the right and analgesia of the left leg. The wound healed rapidly. Patient was in the hospital for 7 months and was finally able to walk with the aid of a cane. Functions of the bladder and rectum were intact. Upon the paralyzed side there was often severe hyperesthesia and paresthesia (burning, numbness, formication). To this were added pain and stiffening in the hip, knee, and ankle joints upon the right side; occasionally, for several seconds, there were painful spasmodic extensions of the right leg, in consequence of which the patient suffered for years from insomnia. Upon the 8th of June, 1904, he was admitted to the Charité.

In this powerfully built patient, there was a cicatrix 3 cm. in length, somewhat to the right of the median line (Fig. 165), between the spinous processes of the 2nd and 3rd thoracic vertebræ. The vertebral column was nowhere sensitive to pressure. There was spastic paresis of the right leg with slight atrophy (inactivity). The circumference of the right thigh was 46.5 cm. and 39 cm., on the left 47.5 cm. and 41 cm. The muscles of the calf were equal upon both sides (circumference 33 cm., each). The patient, like cerebral hemiplegics, dragged the right leg scraping the floor with the tip of the foot in a semicircle. The flexor muscles were more markedly paralyzed than the extensors. In the lumbar muscles and those of the leg on the right side, there were spontaneous contractions. The patient limped—in consequence of contracture of the right sided lumbar muscles—as the right half of the pelvis was higher than the left; the right leg, therefore, appeared shorter and the right *malleolus tibæ* was 2 cm. higher than the left. The length of the legs, measured from the iliac spine to the ankle was equal upon both sides.

Pupillary reflexes normal. Patellar and Achilles tendon reflexes were greatly increased upon the right side, normal upon the left. Babinski's reflex was pathologic

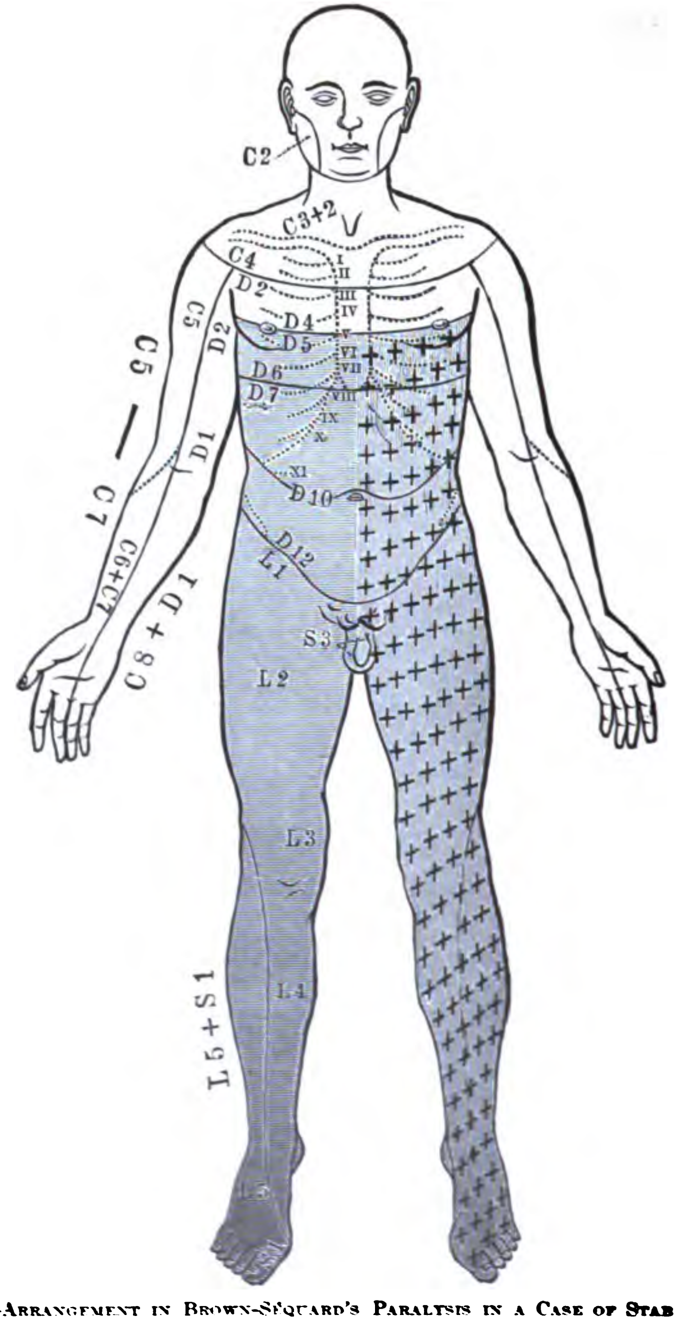

Fig. 164.—Arrangement in Brown-Séquard's Paralysis in a Case of Stab Wound to the Right Half of the Spinal Cord (between the Second and Third Thoracic Spinous Processes) in Seiffer's Diagram.

C, cervical, *D*, dorsal, *L*, lumbar, *S*, sacral, segment. Upon the side of the injury (right) there was *spastic paralysis with hyperalgesia* (transverse shading) and diminution of sweat secretion, upon the left side there was *analgesia* (longitudinal shading) and thermo-anesthesia (crosses). The sensations of contact and position were intact.

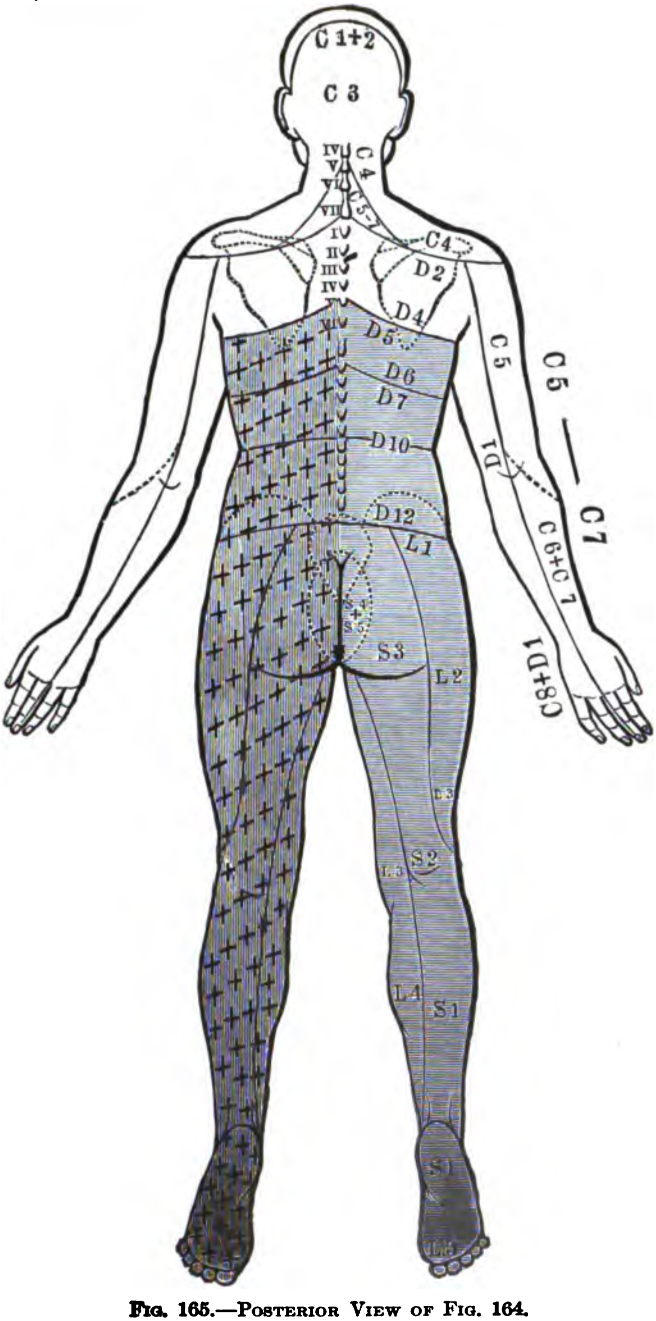

Fig. 165.—Posterior View of Fig. 164.
(For caption see page opposite.)

upon the right, normal upon the left, side. Cremaster reflex normal upon both sides. Abdominal reflexes absent upon both sides.

Electric contractility in the muscles and nerves of the right leg was increased. Secretion of sweat in the right leg was almost arrested. The sensory conditions may be seen from the diagram (see Figs. 164 and 165). The sciatic points upon the right side were extremely painful.

The *treatment* consisted in *methodic exercise*, bath-gymnastics, and carbonated baths, vibration massage, inunctions of iodin-vasogen, application of menthol salves and the administration of veronal and trigemin. The pain and paresthesia in the right leg almost disappeared, and walking improved to the extent that the patient was able to ascend stairs unaided. He was discharged from the hospital July 16th, greatly improved and was able to follow his occupation.

Symptoms on the part of the bladder and rectum may be entirely absent in unilateral myelitis, as the corresponding tracts run bilaterally (according to Kocher in both cervico-dorsal-lumbar tracts); these bilateral tracts act synergistically and may function vicariously for one another. The appearance of bladder and rectal symptoms, therefore, renders a bilateral spinal cord disease likely.

Anesthesia, in a complete transverse interruption, involves all qualities including deep sensation (position, muscle and locality senses). In a partial disease of the cord the individual qualities of sensation (tactile, pain, temperature, vibration sensations) may show all forms of transition, from an absolute to slight loss, in all variations of distribution; sometimes a slowing of conduction is noted. A case reported by J. Schnitzer is remarkable. It was one of spontaneous delivery in a woman suffering from compression myelitis (dorsal cord); there was anesthesia extending 3 cm. above the xiphoid line. Labor was rapid, without pain and without the aid of the abdominal muscles. Partial sensory disturbances are more marked as a rule toward the periphery, i. e., toward the toes and less so centrally, but even here there are exceptions to the rule.

The transverse topography of the focus may be inferred from the nature of the sensory disturbance; thus a loss of pain and temperature senses, with retained contact sense, point to disease of the posterior horn adjacent to the central canal. In other cases the temperature sense may be alone involved or only deep sensation may be affected, with retention of the other sensory qualities. From these clinical facts we may conclude that every sensory quality has special conduction tracts.

The condition of the *reflexes* varies according to the localization, intensity and duration of the disease; sometimes the reflexes are normal, at other times increased to clonus, sometimes abolished. Normal reflexes permit us to conclude that their reflex arcs in the gray substance are normal. Lesions outside of the reflex arcs, for example in the anterior columns, may be present without influencing the reflexes. Hence, absence of reflexes indicates a lesion of the reflex arc, such as may result from disease of the posterior roots (anesthesia), or of the anterior roots (motor paralysis), that is of the gray substance or of Burdach's tract. *Exaggerated reflexes* are due either to greater irritability of the gray substance, or to interruption of the pyramidal tract.

The law that the reflexes are abolished in the innervation area of the diseased section of the cord, being retained above and below in consequence of the interruption of the cerebral reflex inhibition and increased below, is

true of some, but by no means of all, cases of myelitis. Thus in *dorsal myelitis*, the patellar and Achilles tendon reflexes are usually active (their center being in the lumbar and sacral cord), often being even increased to a clonus, sometimes even to the extent that slight peripheral irritation, such as slight contact with or cooling of the legs, produces convulsive muscular contractions. The increase of reflexes may persist for years, even after restitution of motion, as the last sign of recovery from a myelitis. *Exaggerated knee jerk*, naturally, may not be present, when the inflammatory process has involved the lumbar enlargement. On the other hand, in a *high complete transverse separation of the cervical cord* or of the upper dorsal cord, a *loss of the patellar reflex* has been observed, in spite of the anatomical integrity of the lumbar reflex arc. According to Kocher, the absence of the tendon reflexes in the paralytic area especially characterizes complete transverse lesion of the cord, while in unilateral or partial cord lesions the reflexes are retained and commonly exaggerated.

Various hypotheses have been proposed to explain this peculiarity of the reflexes; Bastian, Bruns, van Gehuchten believe that the loss of the reflexes is due to the absence of the muscle tonus tracts which originate from the cerebellum. According to Hoche, perhaps the long, descending posterior column tracts are related to the abolishment of the patellar reflex. Other authors (Gerhardt, Sternberg) believe that there is a reflex inhibition due to irritation originating from the point of lesion passing to the reflex centers. The early absence of the tendon reflexs at the onset of myelitis is regarded as a sign of shock, that is, an *irradiation of the central irritation*. Egger believes in a functional paralysis of the gray substance, and Bischoff in a damage of the latter, as the result of vasomotor and consecutive circulatory disturbances. None of these views have as yet been supported by unquestioned anatomical findings. In many cases the total loss of reflexes (including those of the skin, tendons, vasomotor apparatus, bladder and rectum) in high, complete transverse lesions of the cord, must be regarded not only as the sign of a sudden action, an irradiation of the central irritation, but as the result of a disease descending in the gray substance to the lumbar cord. Thus, v. Leyden in 2 cases of tumor compression of the cervical cord, which ran their course clinically with flaccid paralysis and absent reflexes, found lesions in the anterior horn cells, extending down into the lumbar cord (*Archiv für Psychiatrie*, 1878, Bd. VIII, and *Berlin. klin. Wochen.*, 1878).

In regard to the *skin reflexes* the theories also are far apart. The interruption of the pyramidal tract, for example after hemorrhage of the internal capsule, increases the tendon reflexes, while the cutaneous reflexes are diminished or abolished. In disease of the pyramidal lateral column, the cutaneous and tendon reflexes are usually increased. In diffuse inflammation of the cord, according to the implication of the gray or white substance, the condition of the cutaneous and tendon reflexes will vary. Thus the skin reflexes disappear in cutaneous anesthesia, while the tendon reflexes may be increased. On the other hand in disease of Burdach's columns, the tendon reflexes may be abolished and the skin reflexes retained, as the tracts of the latter radiate directly into the posterior horns and thence into the anterior horns.

To this must be added the physiologic differences in regard to number and strength of the cutaneous reflexes in the normal person. The pathologic

reaction of Babinski's toe reflex, i. e., sluggish primary extension of the large toe upon stroking the ball or the external border of the foot, is not absolutely significant of a pyramidal degeneration. Babinski's reflex also occurs in other diseases of the spinal cord, in healthy nurslings, in sleep, and exceptionally even in some functional diseases of the nervous system (hysteria). From the absence of the skin reflexes, therefore, we can not draw such definite diagnostic conclusions in regard to the nature and seat of the pathologic focus, as from the loss of the tendon reflexes. Only rarely may we obtain an idea of the location of the cord lesion from the segmentary testing of the skin reflexes, for example, when Babinski's and the cremaster reflexes are retained, while the abdominal reflexes are absent; a condition that may occur in a circumscribed myelitis of the lower dorsal cord. Occasionally the skin reflexes in dorsal myelitis may be increased to such an extent that even slight cutaneous stimulation, such as the pressure of the bed clothes, gives rise to contractions, especially in the flexors of the knee joint and in the extensors of the great toe.

Among the *most important and at the same time most serious sequels of myelitis* are the *symptoms on the part of the bladder and rectum.* The bladder disturbance is often an early symptom of myelitis as well as of other cord diseases, especially those involving the posterior columns. At the onset there are usually spasmodic symptoms, tenesmus (spastic contraction of the detrusor) *retentio vesicæ* (sometimes also *ani*). In the further course incontinence occurs. The retention may be *spastic* as the result of sphincter spasm or *paralytic* the consequence of paralysis of the detrusor vesicæ. In this condition, especially if there be anesthesia of the mucous membrane, the bladder may be distended to the umbilicus and even beyond, until the over-distended sphincter vesicæ relaxes (Landois) and dribbling of urine occurs (*incontinentia paradoxa*). In some cases from reflex irritation, for example, upon attempts at movement a discharge of urine, a spasm of the bladder takes place, sometimes also an erection of the penis.

When the sphincter center in the sacral cord is affected, sphincter paralysis takes place, and may be recognized from the constant paralytic incontinence. If the lesion is above the sphincter center, strangury is often unnoted and the discharge of urine occurs involuntarily, but at long intervals and in a full stream. With an *anesthetic mucous membrane of the bladder* prolonged retention of urine occurs, which, in consequence of the pressure, may give rise to circulatory disturbances in the wall of the bladder with subsequent hemorrhage, necrosis and ulceration, especially in the vertex of the organ. Finally, especially at the acme of the disease, paralysis of the sphincters and of the detrusor, with permanent incontinence of urine, appears.

Paralysis of the sphincter is of the greatest importance in diagnosis, prognosis and treatment of myelitis. In the diagnosis, paralysis of the sphincter not infrequently is decisive in differentiating neuritic and toxic paraplegias from myelitis. These maladies were formerly included with spinal paralysis or were regarded as "reflex paralysis." In neurotic paralysis, in contrast to disease of the cord, only exceptionally do bladder and rectal symptoms appear.

The second point of importance in sphincter paralysis is in prognosis, as *incontinence of urine and feces* is a direct danger to the life of the patient.

Evacuation of the bladder in spite of the constant dribbling of urine is not complete. In the stagnating contents of the bladder, partly from the urethra, especially in the female, partly from the lymph channels, by way of the rectum, microbes (bacterium coli) enter. Besides the normal bladder contains numerous germs, which in the healthy are evacuated without damage; in paralysis of the bladder, however, they are retained in the residual urine and produce *cystitis*. According to Charcot, catarrh of the bladder is analogous to the bed-sore and is to be regarded as a trophic disturbance, directly dependent on the spinal lesion. As a matter of fact cystitis may appear without preceding catheterization. Cystitis is diagnosticated from the presence of fever, chills, and the change in the urine. The further consequences are ammoniacal fermentation of the urine, precipitates of calcium phosphate, putrid, even ulcerative and necrotic catarrh of the mucous membrane, ascent of the inflammation to the ureters, to the pelvis of the kidney, and to the kidney itself, as well as a distribution to the perivesical tissues (pericystitis, paravesical phlegmons). Pyelonephritis is a common cause of death in myelitis; such patients become debilitated by fever, chills, metastatic abscesses, septic diarrhea, anorexia and insufficient ingestion of food, until finally collapse or uremia hastens the fatal issue.

Another, quite as serious complication is due to disturbance in the *discharge of the feces*; this is due partly to weakness of abdominal pressure, partly to spastic or paralytic conditions of the sphincter ani. In the beginning the irritative symptoms are most prominent: *tenesmus ani* and *retentio alvi*; tympanitis, in consequence of retention of intestinal gases and obstinate constipation, appears and may even continue for weeks. This condition becomes even more disagreeable and dangerous from paralysis of the sphincter, for the patient often loses the sensation of tenesmus as well as of the passage of feces. The patient lies in the urine and feces which have been involuntarily voided and in spite of the greatest care in nursing, in a brief time, ulcers form *ad nates* (bed sores), the sequels of which (erysipelas, sepsis) cause the death of many a myelitic.

The *male potency*, the power of erection as well as of ejaculation, may sometimes be retained in dorsal myelitis. More often, however, this is lost; occasionally there is spermatorrhea without *libido* and without erection. More rarely there is a reflex erection of the penis or *priapism*. Sometimes there is anesthesia of the penis and testicles; the latter organs are innervated by the upper lumbar segments and, therefore, in isolated disease of the sacral cord, remain sensitive to pressure in the anesthetic scrotal skin (innervation area of the III sacral segment).

Menstruation may be retained for a long time. In the terminal stages of myelitis it commonly ceases in consequence of the debility of the entire organism. Myelitis does not hinder the power of conception; but *pregnancy* usually aggravates the malady.

Vasomotor and *secretory anomalies* also appear in the paralyzed members. The temperature of the skin may be raised at the onset of myelitis and in the further course may drop 2° F. below the normal. The limbs are cool and cyanotic. Very often edema is observed in them, which Vulpian refers to the decreased nutrition of the walls of the vessels. Edema of the legs may be the result of a sluggish circulation and of absence of motion. Thromboses

have been repeatedly noted in paraplegics, in the femoral vein or its branches. Arterial thromboses in the paralyzed limbs are rare; these only develop in endarteritic, that is, sclerotic processes.

Arrest as well as increase of the secretion of sweat has been observed in the paralyzed areas as *anomalies of secretion.* Sometimes while the body is in perspiration, the paralyzed members do not sweat; in other cases the latter are bathed in perspiration and the remaining portion of the body is quite dry. The cause of this disturbance has not yet been made clear.

Among the usual accompaniments of myelitis are *trophic disturbances in the skin, the joints and the bones.* In the skin there are often pemphigus-like or herpetic vesicles, bluish-black infiltrates and even necrotic inflammations. These appear most readily in regions that have suffered from slight mechanical injury. We must, therefore, guard against the most insignificant lesions, for example, pressure of the bed clothes, prick of a pin, etc., for, on account of the loss of sensation in the skin, these are not noted and, therefore, easily become infected and even gangrenous. Further, eruptions of various kinds are noted, urticaria, not infrequently petechia, phlegmons, extravasations of blood and eczema; but we cannot enter upon a description of these at this point. Trophic disturbances are further revealed in the falling out of the hair, in desquamation of the epidermis and of the nails.

The *acute bed-sore* (*decubitus*) is of especially serious import. It was described by Samuel (Königsberg) and mentioned by Charcot in a masterful description, as the characteristic of the malignant form of myelitis. Charcot, and following him numerous other authors, recently also Déjérine, regard acute bed-sore as the result of a trophic disturbance of the cutaneous nervous system. There is much in favor of this view. In many other spinal diseases as in syringomyelia, characteristic trophic disturbances appear. Besides decubitus often appears in myelitis quite acutely, in the first few days, in fact very much more rapidly than in patients bed-ridden from some other cause, even in comatose persons. Thus we saw a case in a woman suffering from dorsal myelitis, in whom during the first week bed-sores appeared in various areas of the lower extremity; first on the heel, then upon the ankle; from the pressure of the bed clothes, then upon the trochanters and the sacrum. First there was a reddish discoloration of the skin which in a few hours became a blackish-blue. Soon the epidermis was raised in vesicles, which after desquamation gave place to ulcers, rapidly gaining in extent. Besides the areas that have been named the iliac fossæ, the *tuber ischii* and the internal condyles of the femur are often the seats of bed-sores.

Without treatment the bed-sore rapidly increases in extent and depth; it finally undermines the soft parts and attacks the periosteum until the bare, necrotic bone can be seen in the floor of the ulcer. The *sacral bed-sore* may attack the retrorectal tissue and give rise to an exceedingly painful, purulent periproctitis.

An *early, systematic* and *careful prophylaxis*, that is, the *treatment* of the *beginning bed-sore* is one of the chief objects in the *therapy* of myelitis and here the *importance of paralysis of the sphincters* and of bed-sores must occupy the main attention of the physician; this greatly influences the *prognosis* and very often even the question of life may depend upon this; bed-sores and cysto-pyelitis are the most frequent causes of a fatal termination in myelitis.

Trophic changes also occur in the bones and joints (osteoporosis, arthropathies), but with less frequency than in tabes, in which the osseous anesthesia with retained muscle power favors the development of arthritic deformities; while in myelitis early motor paralysis compels the patient to remain in bed.

In connection with this description of the symptomatology of myelitis, we shall report a case of *traumatic hematomyelia,* that is *myelitis,* which in many respects is quite remarkable (Case II).

TRAUMATIC HEMATOMYELIA

Man, aet. 35, fracture of the XI thoracic vertebra, complete motor and sensory paraplegia with loss of reflexes and incontinence of the sphincters. Death after 15 months. Destruction of the cord from the 9th thoracic segment downward without demonstrable compression. Tubular posterior columnar hemorrhage.

A sailor, Kr., aged 35, suffered from an accident on October 27, 1896; he fell backward over a mast, while a falling spar struck him on the abdomen. Immediately after this accident his legs were paralyzed and without sensation, there were besides severe pains in the back and side, as well as incontinence of urine. Examination revealed *total, flaccid paraplegia with complete loss of sensation from the soles of the feet to the umbilicus*; above this region there was a narrow hyperesthetic zone (Fig. 166). Posteriorly the loss of sensation reached the upper border of the pelvis. The tendon reflexes in the legs as well as the abdominal reflexes were absent. There was incontinence of urine and feces. In the vertebral column, in spite of repeated examination even by a surgeon, no curvature nor dislocation of the spinous processes was found There was slight sensitiveness to pressure of the 11th and 12th dorsal vertebræ. Lumbar puncture performed early after the trauma, produced 20 c.c. of a clear, watery fluid with but slight pressure.

The paralysis and the sensory disturbances remained stationary during the following 15 months. Rapid atrophy occurred in the muscles of the legs (see Fig. 166), they lost their faradic contractility. All therapeutic attempts (extension, corset treatment, careful nursing and nourishment) were futile. Soon putrid cystitis, gangrenous pressure ulcers, bronchitis and apathy appeared; finally the patient refused nourishment and died upon January 7, 1898, 15 months after the injury. The necropsy revealed, besides general macies, a purulent inflammation of the bladder and kidneys, amyloid degeneration of the spleen and liver, a few bronchopneumonic foci in the left lung and a pelvic phlegmon.

No abnormality was visible externally on the vertebral column at the autopsy. Only after sawing through the vertebral column in a median direction was a *cuneiform deformity* noted in the 11th thoracic vertebra, so that anteriorly it was 12 mm. and posteriorly 20 mm. in height. The spinous and transverse process showed no deviation, only the 11th thoracic vertebra protruded 2 mm. into the vertebral canal. Nowhere was there compression of the cord; at the height of the 8th and 9th thoracic vertebræ the cord was quite flat, contracted to a ribbon. The pia mater showed no changes; nowhere were there traces of hemorrhage. After hardening in formalin and Müller's fluid, there were shown in transverse sections through the cord, that from the 8th dorsal segment downward to the conus medullaris the structure had disappeared (Fig. 167 *f*, *g*, and *h*). This portion of the cord was contracted, and stained a uniform light yellow. In the higher sections (Fig. 167 *a–e*) up into the cervical cord the usual picture of ascending degeneration in the posterior columns as well as in the periphery of the lateral columns was recognized and could be noted by the paler stain. Besides a focus of softening about the size of a millet seed (Fig. 167 *e*) was found from the 8th dorsal segment, extending downward in the left posterior column near the commissure being distributed to the dorsal cord, and at the 4th dorsal segment widening into a cavity

(167 *d*) thence extending over the middle of both posterior columns and filled with yellowish masses. This tubular focus terminated in the lower cervical cord.

Leyden, upon histologic examination (according to Nissl, Marchi, Weigert, etc.), was unable to find nerve fibers or ganglion cells in the lower dorsal, lumbar or sacral cord; the tissue in the entire transverse section consisted of numerous granular cells,

Fig. 166.—A.K., *Traumatic hematomyelia* after fracture of the 11th thoracic vertebra. *Flaccid paraplegia*, sphincter paralysis. Decubitus. The marked atrophy of the thigh and leg is readily noted. The *anesthesia* reached to the umbilicus (marked by a black line); above this there was a narrow hyperesthetic girdle (dotted line).

proliferated glia meshes, myelin granules and a few hematoidin crystals. Only in a few longitudinal sections were there scant fragments of destroyed axis cylinders and medullary sheaths. The pia mater was greatly contracted, the posterior roots were only in part destroyed, while the anterior roots were transformed into an almost complete dense, fibrillary tissue. This destruction of the spinal cord continued in the lateral columns to the 8th dorsal segment, while in the posterior columns it could be followed, in the form of a peg-like focus to the lower cervical cord. The largest focus

also situated in the 4th dorsal segment (Fig. 167 *d*) consists of granular cells, remains of medullary sheaths and glia fibers, the latter becoming denser around the focus. With this there is the usual ascending degeneration in the posterior columns (Fig. 167 *a–e*) and in the lower border zone (cerebellar lateral column, Gowers' bundle). The *muscles* of the leg were in a condition of degenerative atrophy; the transverse

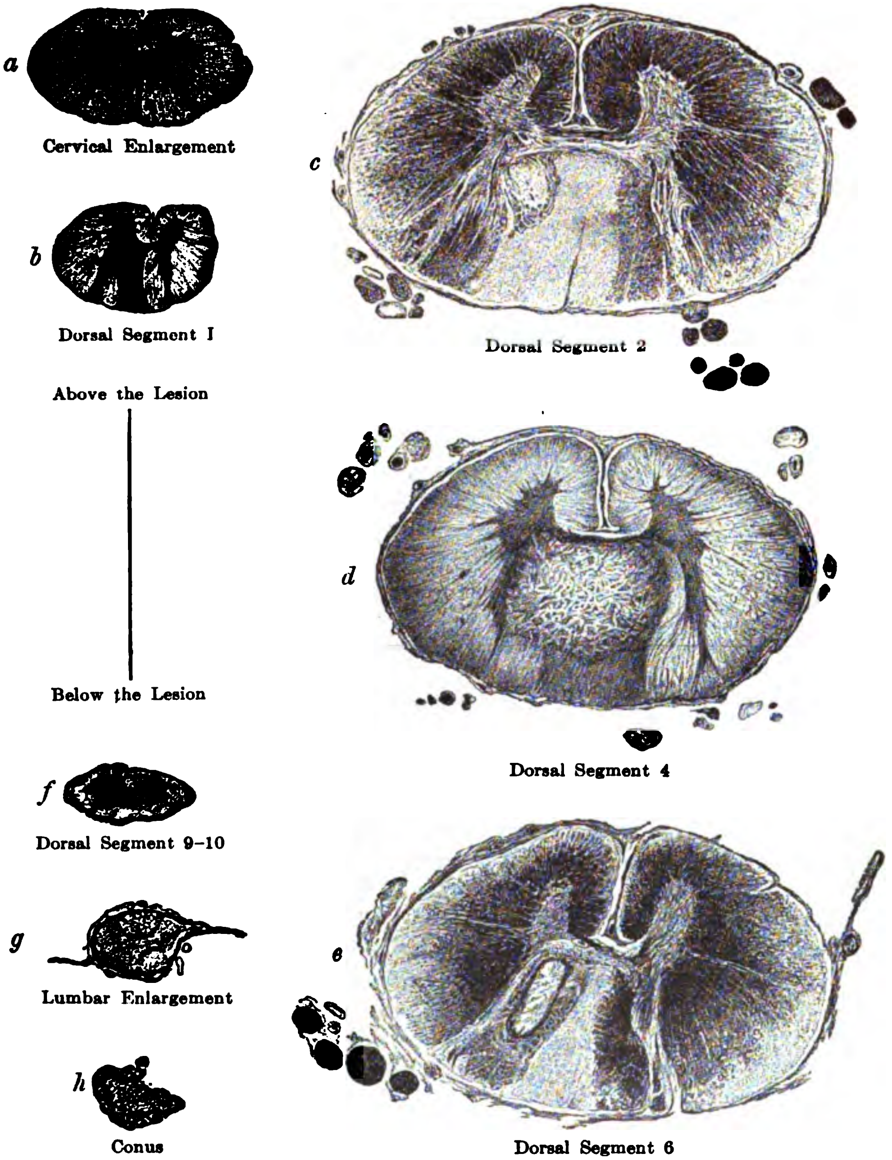

Fig. 167.—*Destruction of the cord* from the 8th dorsal segment downward, Fig. *f, g, h*. *Ascending degeneration* in the posterior and lateral columns, Fig. *a–e*. *Central tubular hemorrhage* in the left posterior column (near the posterior horn of the 6th dorsal segment, Fig. *e*), which in the 4th dorsal segment, Fig. *d*, distributes itself over both posterior columns. Fig. *c, d*, and *e* are enlarged five times, the remaining ones about twice.

striation had disappeared, the muscle fibers were narrowed and permeated by fat globules. Of the nerves which supply the muscle fibers and of the nerve terminations nothing could be seen; the interstitial tissue, and especially the nuclei were increased.

The remarkable point of this case consists in the *total destruction of the cord* from the 9th dorsal segment downward *without demonstrable compression*. The autopsy showed that the 11th thoracic vertebral body was compressed into a wedge shape, but it only protruded 2 mm. into the vertebral canal. As, however, immediately after the injury the signs of a separation of the cord appeared, a transitory compression of the cord must have taken place whereby the vertebra, either spontaneously or by extension, must have returned to its original position. As in the remaining vertebræ and arches no changes could be demonstrated, the view is justified that compression only occurred at the 11th thoracic vertebra. If nevertheless such a large portion of the cord was destroyed this can only be explained by a hemorrhage which permeated the substance of the cord and destroyed it. The elongated focus in the posterior column may also be referred to hemorrhage. Traces of blood were scant which, however, can easily be explained from the age of the focus (15 months).

A similar case was reported by A. Westphal; it was one of traumatic myelitis (paraplegia) after fracture of the bodies of the 1st and 2nd lumber vertebræ. At the autopsy (patient died 7 days after the trauma), it was shown that the vertebræ nowhere exerted pressure upon the cord; the same condition was also revealed by the membranes of the cord, the external configuration being normal throughout. The histologic examination showed the gray, and in part also the white, substance destroyed in the sacral cord while the remaining portions presented marked myelitic changes, which were regarded as the direct result of shock to the cord.

We must still devote a few words to the symptoms on the part of the brain, the eye, the heart and lungs which occasionally appear in myelitis.

The *brain*, apart from encephalomyelitis, the disseminated multiple form of myelitis, is only indirectly involved by embolism from the heart; or by septic and uremic intoxication. A mental depression must not be underestimated which affects the entire condition, the appetite and with it the nutrition and sleep thereby increasing the exhaustion and loss of strength.

In myelitis, symptoms on the part of the *optic nerve* have been observed (Erb, Bielschowsky). Optic neuritis with turbidity of the retina, injection of the vessels and even hemorrhage have been reported. These changes are usually not severe but are of great importance in the diagnosis of disseminated myelitis. According to Henneberg, the lesion of the optic nerve may arise from the direct ascent of the meningo-myelitic process to the base of the brain, or to the distribution of the toxic products in the cerebrospinal fluid, or by a rise of pressure in the interior of the skull in consequence of an increase of the cerebrospinal fluid (stasis inflammation).

The *ear* may be affected; otitis media may be the cause of the development of a descending meningomyelitis.

The *general circulation* and the *heart* do not suffer directly; but from insufficient nutrition, from mental depression, as well as by shock and by various infections, the heart may be damaged, apart from cardiac affections which may have existed previously and become aggravated in the course of the disease.

Pulmonary complications appear without direct connection with myelitis, from embolism or other causes, as bronchitis, not infrequently as hypostatic pneumonia or as metastatic abscesses originating from bed-sores or from cystopyelitis.

3. STAGE OF REGENERATION

The period of *regeneration* is introduced by the gradual return of spontaneous movement and by an improvement in sensation. The muscles become more tense and the previously absent reflexes gradually reappear. The ulcers take on a better appearance, the bed-sores heal, the urine clears; simultaneously the general condition improves; the mental condition, the ingestion of food and the appearance of the patient (tonus of the facial expression) become more favorable, the power of the heart increases. But the patient is still in danger, for relapses and complications cannot be excluded for some time. The anatomical faculty of regeneration of the spinal cord, according to recent investigations, appears to be possible.

The *duration* of the period of regeneration up to a permanent arrest of the symptoms of paralysis cannot be determined with certainty. The more severe the first depressive stage the longer are the advances of the second, the period of restitution. The time up to the arrest of the disease, in mild cases, must be calculated at a few months; in severe cases many months even 1 or 2 years.

4. TERMINATION OF MYELITIS

The *terminations* of myelitis are the following:

(1) *Complete resolution* of the pathologic lesion, as well as entire functional recovery of the patient is quite rare. When this does occur, the cases are very mild, the diagnosis not always positive; but restitution of slight anatomical lesions has been exceedingly likely judging from the results:

(*a*) in mild cases of serous myelitis (edema of the cord);

(*b*) in syphilitic myelitis;

(*c*) in myelitis after infectious diseases, especially the disseminated form;

(*d*) in traumatic and compression myelitis.

(2) *Partial recovery* is more common than complete. A decided improvement in the anatomic lesion cannot be doubted in these cases.

(3) *Termination in typical sclerosis.* This is especially seen in multiple sclerosis following infectious diseases. In connection with this are

(4) The conditions in which the *residual processes* in the spinal cord (cicatrization, degeneration, cysts, contraction) must be regarded as *incurable.* On the other hand the sequels which the patient presents (paralyses, contractures, muscular atrophy, pain, cystitis, bed-sores, etc.) are more liable to improvement from proper treatment, which we owe particularly to the advances of physical-orthopedic therapy.

(5) The termination of myelitis in *abscess* is very rare: observations of this kind have been published by Nothnagel. These were mostly metastatic medullary abscesses, occurring in the course of pulmonary abscess. The abscess, with the rapid development of paralytic symptoms, soon causes death.

(6) The *lethal termination* is relatively frequent in the severe cases of transverse myelitis, as well as in bulbar myelitis. The autopsy, according to the duration of the disease, reveals the lesions of the fresh process, or at the periphery contracting connective tissue strands (cicatricial tissue), perhaps with small cavities (cyst formation) are already present.

In connection with these lesions in the spinal cord, *those of other organs*

must be considered and present more or less dependent complications or sequels. We must mention briefly (*a*) disease of the vertebræ, intraspinal tumors, (*b*) cystitis with pyelitis and nephritis, bed-sores and foci of general septic infection in the lungs, heart, or skin, (*c*) more rarely cardiac and pulmonary disease (hypostatic pneumonia), (*d*) muscular atrophy, changes in the bones and joints, (*e*) syphilitic changes in the internal organs.

Before proceeding to the clinical description of the forms of cervical, lumbar and sacral myelitis, it is necessary to describe the regional diagnosis of diseases of the cord. For this purpose we must first explain:

THE SEGMENTARY LOCALIZATION OF THE FUNCTION OF MOTION

Myelitis according to its height and transverse diameter in the cord may present a manifold clinical picture. An understanding of this varying physiognomy of myelitis is gained from an appreciation of the *segmental arrangement* of the spinal functions, which we must discuss somewhat minutely as it forms the foundation of the topical diagnosis of disease of the cord. *Not only in the brain, but also in the spinal cord, are ganglion cells, which are functionally united for the motion, sensation and reflexes of the various members, grouped in definite parts, one above the other, that is in segments.* Of these there are 31 in the cord, corresponding to the nerve root pairs, 8 cervical, 12 thoracic, 5 lumbar and 5 sacral segments and one in the coccyx.

These segments do not correspond in location to the vertebræ of similar names, but are higher than the point of exit of the nerve roots belonging thereto. This difference increases caudally. It is the consequence of the relative shortness of the spinal cord, which is only 45 cm. (compared with the vertebral column which is 70 cm.), and therefore at the top of the first, occasionally of the second, lumbar vertebra (especially in women) finds its termination. In the embryo the cord fills the entire vertebral canal; but in the further course of fetal development the longitudinal growth of the cord no longer keeps pace with that of the vertebral column, especially its lumbosacral portion, so that finally in the new-born the 4 lower lumbar vertebræ and the sacrum contain no cord but only the cauda equina.

The knowledge of this *upward shifting of the cord segments* is of great importance in diagnostic and especially in surgical considerations. Thus a morbid focus located in the lumbar cord should not be sought for in the lumbar vertebral column, but in the lowest two thoracic vertebræ. On the other hand operations may be performed upon the lower lumbar vertebræ and upon the sacrum (resection, puncture) without danger of injuring the spinal cord. The cervical cord segments are situated about one vertebra higher than their corresponding vertebra. In the thoracic segments this difference amounts to from 2–3 vertebræ, so that finally the lumbar cord is at the height of the 2 lowest thoracic vertebræ. The sacral cord is situated behind the first lumbar vertebra and the conus medullaris behind the succeeding intervertebral disc, sometimes even behind the upper half of the second lumbar vertebra. It must, therefore, be remembered *that the cord focus is always higher than the vertebra which numerically corresponds to it.*

Prior to a consideration of the various clinical pictures presented by disease of the individual sections of the cord, we shall discuss the spinal cen-

tralization of motor and reflex functions in a tabular form. The credit of having constructed the laws of segmentary innervation is due to a number of physiologists, anatomists, and clinicians, but especially to surgeons. We refer to Bolk, Bramwell, Bruns, Edinger, Goldscheider, Gowers, Head, Kocher, Fr. Kraus, Lapinsky, Oppenheim, Ross, Seiffer, Sherrington, Allen Starr, Thornburn, Wagner and Stolper, Wichmann and others whose experiences we have utilized in this compilation.

We shall not, as has been customary in previous compilations, regard each segment as an elementary central organ, but on the contrary attempt to present the localization of the muscle groups which functionally act together. One segment never acts alone, never does a single muscle contract, but always several segments are innervated simultaneously. Even the simplest movements are not brought about by the contraction of an isolated muscle, but by the harmonious coördinated effect of several muscles. Thus a simple cortical impulse necessary to flex the elbow employs 4 segments, in corresponding to the localization of the flexors of the elbow joint (brachialis internus, biceps, brachioradialis, pronator teres, extensor carpi radialis longus) from the 4–7 cervical segments. No single one of these muscles can voluntarily be contracted by itself. Besides every single muscle, as well as one and the same cutaneous area, especially in the limbs, is in connection with at least 3 nerve roots, that is, segments (compare P. Lazarus, "Über die spinale Localisation der motorischen Functionen," *Zeitschr. f. klin. Med.*, Bd. 57, p. 99 et seq., 1905).

Thus the biceps is innervated by 4 segments (4, 5, 6, and 7th cervical segments). Usually only one or 2 segments contain the center for the main function of the muscle in question, while the neighboring segments are mainly concerned in coördination. As at the present time we possess no definite knowledge in regard to the localization of muscle nuclei, in the following table all segments are included, which may be in direct connection with the respective muscle groups. Subsequent investigation will be required to modify or correct our tabulation.

In connection with this multi-segmentary localization and innervation is the fact that one and the same muscle may be involved in various functional movements. Thus the biceps may elevate the arm anteriorly, flex the forearm and finally supinate it without it being possible for us at the present time to state definitely, which ganglion cells of the segments concerned supply one or the other function. Nor does the stimulation of a single anterior root of a multi-segmentary muscle lead to the contraction of the entire muscle, but only to a definite portion. Neither does section of a single root produce complete paralysis, but the muscle is rendered useless only for certain movements, in which it was correlated with other muscles (Sherrington, Russell and others).

In conformity with this we note in some spinal diseases, such as Duchenne-Aran's paralysis, that only some portions of the muscle atrophy, while others remain normal; consequently the muscle may be paralyzed only in one function, while in other respects it acts normally. The loss of function, therefore, of a single segment or root produces a partial functional disturbance, which may be compensated by the vicarious action of retained adjacent segments.

The joints are also supplied by several segments or nerves. Thus upon

the extensor surface, the knee joint is innervated by the crural, and upon the flexor side by the sciatic, nerves. Every cutaneous area beside being in connection with its own segment is to a certain extent also influenced by the adjacent segments, above and below. Only the loss of function of at least 3 segments produces complete paralysis. In contrast to this, paralysis produced by lesions of a peripheral nerve implicates the entire area of distribution of the nerve. For this reason segmental paralysis has a different limit than that brought about by disease of the peripheral nerve.

In the following tables the separate motor functions and the reflexes, with the segments that supply them, are placed together. This grouping we employ in the examination of spinal lesions. From a methodic and detailed investigation of the power of motion, of sensation, and of the reflexes, or from the absence of definite functions, we are enabled to conclude which components of the muscle and nerve are involved and thus determine the segment that is affected.

Further it may be noted from the table, that one and the same segment may contain the fibers for the antagonists as well as of the agonists (both acting together in every coördinate movement), and that the functional area of the segment does not coincide with the region of innervation of the peripheral nerve trunk. The spinal cord segment like the brain supplies no single muscle or nerve, but only power for a definite kind of motion.

This segmentation is even more marked in the lower animals than it is in man. Thus in the zebra, it is marked by the stripes of colored hair, in the ring snake by the roseate swelling of the spinal cord corresponding to the points of exit of the nerve roots (Lüderitz). In man the individual medullary segments are not so sharply separated from each other; they are united by numerous "intersegmental and conjunction tracts." The enlargement of the cervical and lumbar areas (spinal arm and leg centers) merely correspond to the complicated functions of the arms and legs.

Finally we must mention that up to this time we only know the projection of the spinal cord segments into 2 organ systems, into the skin and muscles, while in regard to the spinal innervation of the internal organs, especially of the heart, of the vessels, of the intestinal tract (with the exception of the sphincters) and even of the bones and joints, we possess no positive knowledge.

SPINAL LOCALIZATION OF THE MOTOR FUNCTIONS

Abbreviations: C., D., L., S. = Cervical, Dorsal, Lumbar, Sacral. S. = Segment. N. = Nerve. Plex. = Plexus.

Head Movements: I.–VIII. C. S.

Flexors of the head: I.–V. C. S. (Nervi cervical.–Nerv. accessorius.)

Extensors of the head: I.–VIII. C. S. (Nervi cervical.–N. suboccipital, N. accessorius, N. occipital. major.)

Rotators of the head and lateral movements: I.–VIII. C. S.

(N. suboccipital.–N. accessorius.–N. cervical.)

NECK MOVEMENTS: I.–VIII. C. S.

Flexors: II.–VIII. C. S.
(Plex. cervical. et. brach.)

Extensors: I.–VIII. C. S.
(Plex. cervical. et. brach.)

Rotators and lateral movements: I.–VIII. C. S.

(N. suboccipit.–N. cervical. I.–VIII, Plex. brach., N. dorsalis scapulæ.)

Movements of the hyoid bone: I.–III. C. S.

Elevators: I.–II. C. S.
(Ramus mylohyoideus N. trigemini ram. III., N. facialis et hypoglossus.)

Depressors: I.–III. C. S.
(Ram. descend. N. hypoglossi.)

BACK AND TRUNK MOVEMENTS: I. D. S.–I. S. S. (perhaps also with the action of the I.–VIII. C. S.)

Retracting flexors (*extensors*) I. C. S.–I. S. S.
(N. cervical.–thoracal.–lumbal–sacral, N. thoraco–dorsalis, N. glutæus inf.)

Erectors: V. D. S.–IV. L. S.
(N. intercostal. V.–XII., N. iliohypogastricus – N. ilioguinalis, plex lumbalis.)

Rotators and lateral movements: I. C. S.–I. S. S.

(N. cervical.–thorac.–lumbal.–sacral.)

ABDOMINAL MUSCLES: V. D. S.–I. L. S.

(N. thoracal. V.–XII. N. iliohypogastr.–N. iliolinginalis.)

RESPIRATORY MOVEMENTS: (Medulla oblong. up to XII. D. S.)

Inspiratory muscles: Med. oblong.–XII. D. S.
(N. facialis, N. vagus, N. phrenicus, N. accessorius, N. intercostal. I.–XII., Plex. cervic. et brachialis.)

Expiratory muscles: II. D. S.–I. L. S.
(N. intercostal. II.–XI.–N. iliohypogastr. et N. ilioinguinalis.)

Abdominal press: VII. D. S.–I. L. S.

(N. intercost. VII.–XII, N. iliohypogastric. et ilioinguinal.)

UPPER EXTREMITY

Shoulder girdle and also shoulder joint: II. C. S.–VIII. C. S.

Elevators: II. C. S.
(N. accessorius, Plex. cervical., N. dorsal. scapul., N. thoracic. long.)

Depressors: V. C. S.–VIII. C. S.
(N. subscapularis N. thoracal. anter.)

Adduction: II. C. S.–VIII. C. S.
(N. accessor.–N. subscapularis, N. dorsal. scapul.)

Abduction: V. C. S.–VII. C. S.
(N. thoracal. long.–N. subscapular.)

Shoulder joint: IV. C. S.–VIII. C. S.

Abduction: IV.–VI. C. S.
(N. axillaris, N. thoracic. long., N. suprascapular.)

Adduction: IV.–VIII. C. S.
(N. thoracal. ant., N. suprascapular.–N. subscapul., N. radialis.)

Forward: IV. C. S.–VII. C. S.
(N. axillaris, N. musculocutaneous, N. thoracic. long.–N. thorac. ant.)

Backward: IV. C. S.–VII. C. S.
(N. axillaris, N. subscapular.)

Inward rotator: V. C. S.–VII. C. S.
(N. subscapularis, N. thoracal. ant.)

Outward rotator: IV. C. S.–VI. C. S.
(N. suprascapular, N. axillaris.)

Elbow joint: IV.–VIII. C. S.

Extension: V. C. S.–VIII. C. S.
(N. radialis.)

Flexion: IV. C. S.–VII. C. S.
(N. musculocutan.–N. median.–N. axillaris.)

Supination: V. C. S.–VIII. C. S.
(N. radialis, N. musculocutaneous.)

Pronation: VI.–VIII. C. S.
(N. medianus.)

Wrist joint: VI. C. S.–I. D. S.

Extension: VI. C. S.–VIII. C. S.
(N. radialis.)

Flexion: VI. C. S.–I. D. S.
(N. median, ulnaris et radialis.)

Radial flexion: VI. C. S.–VIII. C. S.
(N. radial. et median.)

Ulnar flexion: VI. C. S.–I. D. S.
(N. radial. et ulnar.)

Finger movements: VI. C. S.–I. D. S.

Extension: VI. C. S.–VIII. C. S.
(N. radialis.)

Flexion: VII. C. S.–I. D. S.
(N. median. et N. ulnar.)

Abduction: VII. C. S.–I. D. S.
(N. ulnar.–N. radial.)

Adduction: VII. C. S.–I. D. S.
(N. ulnaris.)

Thumb movements: VI. C. S.–I. D. S.

Extension: VI.–VIII. C. S.
(N. radialis.)

Flexion: VI.–VII. C. S.
(N. ulnar. et median.)

Abduction: VI. C. S.–I. D. S.
(N. radialis.)

Adduction: VII. C. S.–I. D. S.
(N. ulnaris.)

Opposition: VI. C. S.–I. D. S.
(N. radial., median et ulnar.)

Movements of the little finger: VII. C. S.–I. D. S.

Extension: VII. C. S.–I. D. S.
(N. radial. et ulnar.)

Flexion: VII. C. S.–I. D. S.
(N. ulnar. et median.)

Abduction: VII. C. S.–I. D. S.
(N. ulnaris.)

Adduction: VIII. C. S.–I. D. S.
(N. ulnaris.)

Opposition: VII.–VIII. C. S.
(N. ulnaris.)

Lower Extremity

Hip joint: XII. D. S.–I. S. S.

Extension: IV. L. S.–I. S. S.
(N. glutæus inf.–N. peron. commun.–N. tibialis.–Plex. ischiadic.)
Abduction: IV. L. S.–I. S. S.
(N. glut. sup.)
Outward rotators: II. L. S.–I. S. S.
(N. tibial.–Plex. ischiadic.–N. obturat.–Plex. lumb.–N. femor.–N. glut. inf.)

Flexion: XII. D. S.–I. S. S.
(Plex. lumb.–N. femor.–N. glut. sup.–N. obturat.)
Adduction: III. L. S.–I. S. S.
(N. femor. N. obtur.–N. tibial.)
Inward rotators: II. L. S.–I. S. S.
(N. glut. sup.–N. obturat.)

Knee joint: II. L. S.–II. S. S.

Extension: II.–IV. L. S.
(N. femor.)

Flexion: III. L. S.–II. S. S.
(N. tibial.–N. peron. com.–N. femor.–N. obturat.)

Ankle joint: IV. L. S.–II. S. S.

Dorsal flexion: IV. L. S.–I. S. S.
(N. peron. prof.)
Adduction: IV. L. S.–II. S. S.
(N. tibial.–N. peron. prof.)
Pronation: IV. L. S.–I. S. S.
(N. peron. superfic. et prof.)

Plantar flexion: IV. L. S.–II. S. S.
(N. tibialis, N. peron. superfic.)
Abduction: IV. L. S.–I. S. S.
(N. peron. superfic. et prof.)
Supination: IV. L. S.–II. S. S.
(N. tibialis, N. peron. prof.)

Toe movements: IV. L. S.–II. S. S.

Dorsal flexion: IV. L. S.–I. S. S.
(N. peron. prof.)
Adduction: I. et II. S. S.
(N. plant. lateral.)

Plantar flexion: V. L. S.–II. S. S.
(N. tibial.–N. plantar. med. et lateral.)
Abduction: I. et II. S. S.
(N. plant. lateral.)

Great toe: IV. L. S.–II. S. S.

Extension: IV. L. S.–I. S. S.
(N. peron. prof.)

Flexion: V. L. S. I.–II. S. S.
(N. tibialis, N. plant. med. et later.)

SPINAL LOCALIZATION OF THE REFLEXES

I. *Tendon reflexes*:
Achilles tendon reflex: III. L. S.–I. S. S.
Patellar tendon reflex: II.–IV. L. S.
Extensor reflex in the upper and lower arm: VI. C. S.
Flexor reflex (closure of the fingers upon tapping the palm): VII. C. S.

II. *Cutaneous reflexes*:

Sole of the foot reflex: IV.–V. L. S. (perhaps also I. II. S. S.).
Babinski's reflex: dependent upon the intactness of the pyramidal-lateral column tract.
Cremaster reflex: I. II. L. S.
Glutæl reflex: IV.–V. L. S.
Abdominal wall reflex
epigastric: IV.–VII. D. S.;
meso- and hypogastric: VIII.–XI. D. S.
Interscapular reflex: V. C. S.–I. D. S.

III. *Reflexes of the smooth muscles*:

Cilio-spinal center: VI. C. S.–III. D. S.
Dilatation of the pupil (upon irritation of the nape): III.–VII. C. S.
Ejaculatory reflex center: III. S. S.
Erection reflex center: II–IV. S. S.
Bladder and rectal reflex centers: III.–V. S. S.
(The rectal reflex center is situated below the bladder reflex center.)

TRANSVERSE DIAGNOSIS

Myelitis may principally attack the central gray matter—*poliomyelitis* already mentioned—which appears to be the case especially in disease of the cervical and lumbar enlargements. It is, however, more common for the inflammation to affect the white substance more intensively: border myelitis, *leukomyelitis*. Ordinarily myelitis attacks the gray and white substance in an irregular arrangement. In the localization diagnosis of myelitis not only the height of the lesion but also the transverse distribution, and with this the complete topography of the focus, is to be determined. The topographical transverse diagnosis is only possible through a minute knowledge of the functions of each individual fiber system. For this purpose the adjoining figure, which presents a diagram of a transverse section of the spinal cord, containing the motor and sensory functional regions, should be consulted (Fig. 168).

A *lesion of the gray substance* leads, almost exclusively, to a loss of function in the area of innervation to which it belongs, while destruction of the white substance also interrupts those tracts of conduction which pass the segment in question on the road to and from the brain. In this manner a *remote action* is produced in all of the segments situated above and below the lesion, which are present in the direction of physiologic conduction (cellulifugal) leading to ascending and descending degeneration; this condition may be demonstrated during the second week, after the lesion has taken place.

The *anterior horn cells* represent the *motor, trophic and probably also the vasomotor centers*, and isolated disease of this structure reveals the picture of atrophic muscular paralysis, such as we see in *infantile spinal paralysis* or in *progressive spinal muscular atrophy*. In both, flaccid paralysis with rapidly progressing muscular atrophy, with abolished reflexes and with DeR, are characteristic. Frequently vasomotor disturbances and anomalies in the secretion of sweat are added. All sensory and motor functions of the segments, situated above and below the point of lesion, are intact as well as those of

the bladder and rectum. In case a few ganglion cells remain normal within the affected anterior horn then the muscle fibers supplied by them retain their function. Disease of the anterior root produces the same picture as a lesion of the anterior horn to which it belongs; both including the axis cylinder which originates there form an anatomical unit, the spino-muscular neuron.

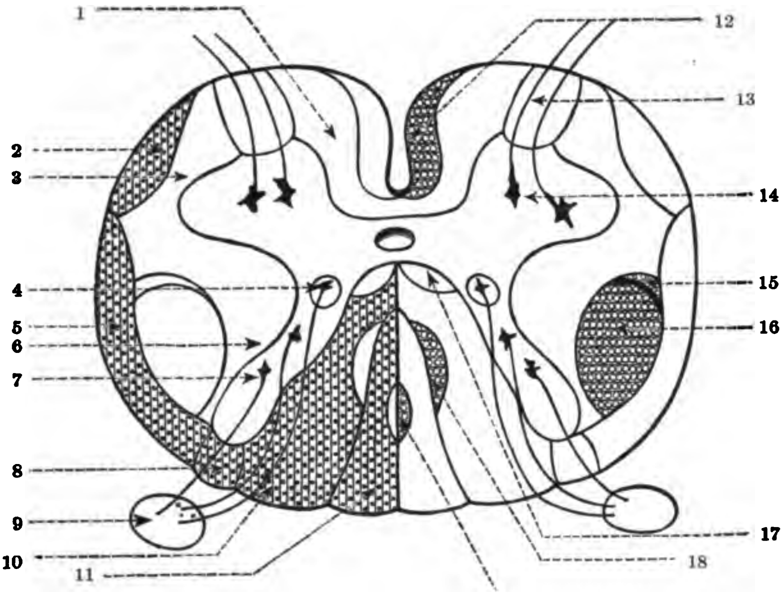

FIG. 168.—SCHEMATIC REPRESENTATION OF THE SENSORY AND MOTOR SPINAL CORD TRACTS. Left: *Ascending conduction tracts* (longitudinal shading and dotted). Right: *Descending tracts* (transverse shading with circles).

1, Anterior column-ground bundle; *2*, Gowers' column; *3*, Lateral column remains; *4*, Clarke's columns; *5*, Cerebellar lateral column tract; *6*, Lateral boundary layer of the gray substance; *7*, Posterior horn; *8*, Lissauer's border zone; *9*, Spinal ganglion with the posterior root fibers; *10*, Burdach's column; *11*, Goll's column; *12*, Pyramidal anterior column tract; *13*, Anterior root fibers; *14*, Motor anterior horn cells; *15*, Monakow's rubrospinal bundle; *16*, Pyramidal lateral column tract; *17*, Ventral posterior column field; *18*. Descending dorsal root fibers (Schultze's "comma field"); *19*, Oval field (Flechsig).

The shaded areas on the right characterize the most frequent distribution of descending, those on the left of ascending, degeneration after transverse lesions of the cord.

In contrast to this, disease of the cerebrospinal neuron, the *pyramidal tract*, gives rise to entirely different symptoms. The origin of the pyramidal tract is in the large pyramidal cells of the cerebral cortex, its termination in the crossed anterior horn. Lesion of the pyramidal tract not only produces an increase of tension of the muscle tonus (*hypertonia*) in the area of innervation of the destroyed segment, but also in those lying below; further there is an increase of the tendon reflexes, a positive Babinski toe reflex and some weakness of the muscles. Neither atrophy (apart from the atrophy of inactivity), nor complete paralysis occurs; automatic and reflex movements, as well as electric contractility are retained. A lesion of the pyramidal lateral column

tract may be compensated for, to a certain extent, by the vicarious action of the motor reserve tracts (pyramidal anterior column tract, the ganglion tracts), in contrast to the irreparable functional destruction after disease of the anterior horns. We must mention briefly that combined disease of the entire motor system (anterior horn and pyramidal tract) produces the well known picture of amyotrophic lateral sclerosis (paralysis with muscular atrophy and spasm). According to recent investigations it appears doubtful whether the spastic condition is due to a lesion of the pyramidal tract or that it is only the consequence of an irritation of the anterior horn cells.

The symptoms of absence of function in the other descending tracts, from the cerebellum, the optic thalami, the corpora quadrigemina, the red nucleus and the olives which pass in part into the lateral columns, partly into the anterior columns, are as yet not absolutely determined.

Sensory disturbances only appear in diseases of the posterior roots and in their area of distribution in the posterior horns and in the posterior columns. An affection of the posterior roots or of the posterior columns produces complete segmentary anesthesia and analgesia, with loss of the tendon reflexes. An irritative condition of the posterior roots gives rise to paresthesia and hyperesthesia, and also to lancinating pains and girdle sensations, from the position of which we may sometimes determine the height of the medullary focus.

A *partial sensory defect* points to the intramedullary seat of the spinal paralysis. Dissociated sensory paralysis is characteristic of an isolated disease of the posterior horn, i. e., the arrest of pain and temperature sense with retained tactile sensation. In unilateral disease of the cord this sensory paralysis occurs in the area of innervation of the lesion upon the same side, on the other hand, in the area of innervation of the regions of the cord situated below upon the opposite side, for the sensory tracts cross within the central gray matter (compare the clinical picture of Brown-Séquard's paralysis).

Disturbance of the muscle sense with decrease of the muscle tonus (hypotonia), and disturbance of the tactile and pressure senses with but slight loss of cutaneous sensation, are characteristic of an isolated disease of the *posterior column.* An affection of the posterior column produces the picture of tabetic ataxia, which at the present time is generally referred to a disturbance of sensation. The functional importance of the remaining ascending tracts, of the delicate sensory anterior column ground bundle, also of the cerebellar lateral column tracts and of Gowers' tract, have not yet been positively demonstrated; possibly the cerebellar lateral column tract, which arises in Clarke's cells, passing to the corpus restiforme and cerebellum, serves to regulate equilibrium; according to Gowers the tract named after him conducts the sensation of pain, while other authors include Gowers' bundle among the equilibrium tracts because it terminates in the cerebellum.

After having described the pathologic anatomy and clinical picture of myelitis, as well as the symptomatology of the motor, sensory, and reflex disturbances, according to the height and transverse distribution of the myelitic process, we shall present the *individual clinical forms of myelitis*; in this we shall include a description of the etiology, prognosis and treatment of the disease.

1. CERVICAL MYELITIS

Inflammation of the cervical cord is the rarest and simultaneously the severest form of myelitis; it develops as the consequence of compression of the cord and may produce paralysis, as well as anesthesia of the trunk and of all 4 members; further, a disturbance of the eyes and of the face; and on account of paralysis of the diaphragm, the respiration may be so influenced that death from suffocation sometimes takes place.

Complete transverse disease of the cervical cord is rarer than in the dorsal cord, as the former is larger than the latter; according to Stilling's measurements the transverse segment of the cervical cord varies between 44.7 and 62.4 cmm., in contrast to 28.6–36.2 cmm. in the dorsal cord (measured in the child).

Diseases of the cervical cord may be divided into 2 groups; in the first—*superior type*—are affections of the 4 upper cervical segments, while to the second group—*inferior type*—belong the maladies of the 4 lower cervical segments. The diseases of the upper dorsal segment may be included with those of the inferior type. It is located behind the seventh cervical vertebra and anatomically as well as functionally belongs to the cervical enlargement which reaches from the 2d dorsal segment to the 5th cervical segment inclusive.

Disease of the 4 upper cervical segments directly endangers life. Affections of the *phrenic center* which is mainly contained in the 4 cervical segment are particularly serious; this is situated behind the upper portion of the 4th cervical vertebral body. In a total transverse disease at this height (*typus quartus*) symptoms of paralysis of the diaphragm appear: the epigastrium retracts during inspiration, the respiratory auxiliary muscles act energetically, but nevertheless little air enters into the pulmonary alveoli; on the other hand considerable blood enters the alveolar vessels; the dangerous symptoms of air hunger and of asphyxia (dyspnea, cyanosis) appear. Added to this, from the insufficient action of the diaphram, abdominal symptoms arise (downward displacement of the liver, meteorism, *retentio alvi*, engorgement of the circulation). The ordinary outcome of *paralysis of the phrenic nerve is death from suffocation.* Only in unilateral disease of the phrenic center, life may exist so long as the opposite half of the diaphragm continues to function. Unilateral paralysis of the phrenic nerve produces paralysis of the same side of the diaphragm; this half is elevated (cadaver position) and the lung above it takes no part in respiration. In this portion of the pulmonary system hypostatic pneumonia or a purulent bronchitis may readily develop and frequently enough terminates fatally.

The *symptoms of absence of function in disease of the upper cervical segments* (the first cervical segment is situated in the arch of the atlas) have not been accurately determined; at all events life can exist only with a partial lesion or with a very slight compression of the upper cervical segments. In an implication of the upper cervical segments all four members may show spastic paralysis; muscular atrophy arises only when the lower cervical segments are involved (Kahler). Besides there is usually paralysis of the deep muscles of the neck and nape, of the sternocleidomastoid and of the trapezius. Further there may be difficulty in the production of the voice and disturbance in deglutition, as well as paralysis of the muscles of the palate and tongue;

the origin of the hypoglossal and spinal accessory nerves reach as low as the second and third cervical segment. These symptoms thus form a transitional stage to bulbar paralysis, which not infrequently is due to an inflammation ascending from the cervical cord to the *bulbus medullæ*; and gives rise to the clinical picture of atrophic glosso-pharyngo-labial paralysis (see Leyden, *Archiv für Psychiatr. u. Nervenkh.*, 1870, Bd. II u. III u. VII: acute bulbar paralysis).

Cerebral symptoms rarely arise in uncomplicated cervical cord inflammation. Only when the inflammation attains the medulla and the brain, or is propagated to the membranes of the cord and thence to the meninges, or in cerebral and spinal disseminated inflammation, do cerebral symptoms appear; such as headache, altered states of consciousness, paralyses of the cerebral nerves and not rarely optic neuritis (Erb, Bielschowsky).

In regard to sensory phenomena in cervical myelitis there may be anesthesia distributed over the entire body from the toes to the back of the head. The occiput is innervated from the first and second cervical segments; the third cervical segment supplies the anterior and lateral skin of the neck, the fourth supplies the skin of the nape, the shoulder and the region of the clavicle to a point near the second intercostal space. The skin of the trunk below this is not supplied by the 4 lower cervical segments but from the dorsal cord. The 4 lower cervical segments and the first dorsal segment have no part in the sensory innervation of the neck and trunk; they supply the skin of the arms in the form of longitudinal striæ, which, corresponding to the height of the segment, run upon the radial and ulnar sides.

Symptoms of paralysis of the arms occur not only in disease of the 4 lower cervical segments but also with involvement of the 2 upper dorsal segments. The latter are situated behind the 7th cervical vertebra and the intervertebral disc beneath. In an inflammation of the 2 lowest cervical segments and of the 2 upper dorsal segments there is paralysis from the elbow joint downward (*paralysis of the forearm*). The ulnar muscles as well as the small muscles of the hand and fingers are most markedly affected and there is often atrophy and DeR. Ulnar flexion of the hand, all of the movements of the little finger, of the interossei and lumbricales, as well as adduction of the thumb are usually decidedly paretic, while the other movements of the thumb as well as of the long flexors and extensors of the fingers and of the hand are only somewhat weakened. Extension of the elbow joint, pronation, adduction and the backward movement of the forearm may occasionally be paretic, while supination (brachioradialis) and flexion of the elbow joint are quite normal.

The muscular atrophy appearing in the paralyzed members is characteristic of a lesion in the lower cervical cord; this is absent when the interruption in conduction is situated higher up (Kahler, also F. Kraus, Die Bestimmung des betroffenen Rückenmarkssegmentes bei Erkrankungen der unteren Halswirbel, *Zeitschr. f. klin. Med.*, 1891, XVIII. Bd).

In disease of the first dorsal segment, but especially in lesions of the first anterior dorsal root, besides paralysis of the small muscles of the fingers and anesthesia in the ulnar distribution, the symptoms of *Klumpke's paralysis* are observed: *contraction of the pupil and of the palpebral fissure* (*miosis et ptosis spinalis paralytica*) with *softening and retention of the eyeball* (paral-

ysis of Müller's muscle). Sometimes there is vascular dilatation and wasting of the same side of the face (flattening of the cheek). These symptoms are referred to an implication of the sympathetic nerve. The sphincter iridis is supplied by the oculomotor nerve, and the dilator of the pupil by the cervical sympathetic. Its oculopupillary fibers arise in the oculopupillary center of the medulla; they pass down through the cervical cord and out in great part through the first pair of dorsal roots. Thence they pass through the communicating branches to the inferior cervical ganglion of the sympathetic, from here into the cavity of the skull and then along the sinus cavernosus to the orbit. Irritation of the first dorsal root dilates the pupil of the same side. This was experimentally proven by Oppenheim, in a case trephined by v. Bergmann, in which the cervicodorsal vertebræ were opened on account of a gun shot injury. Along with the dilator fibers, the nerve fibers for the smooth muscles of the orbits and lids pass from the medulla to the first anterior dorsal root; therefore an isolated lesion paralyzes nearly all sympathetic ocular fibers. But a lesion of the medullary segments above this, up to the medulla, may also produce pupillary disturbances, as all cervical segments are penetrated by the pupillary fibers. On the other hand, according to Klumpke's experience, oculopupillary symptoms never appear in plexus lesions.

If the inflammation also involves the 5th and 6th cervical segments, which like the two lower segments are about 2½ cm. in length, the paralysis is distributed to the *upper arms.* Most of the cell groups for the functions of the deltoid, brachioradialis, brachialis internus, coracobrachialis, teres minor, infraspinatus, supraspinatus and of the rhomboidei are contained in the 5th cervical segment. In disease of the 5th cervical segment the arm hangs flaccidly, as if dead; besides there may also be paresis of the scaleni, as well as of the deep muscles of the neck and nape. The consequence is difficulty in rotary and flexure movements and in fixation of the head; the respiration, as the result of paresis of the auxiliary muscles of respiration, may be of pure abdominal type. According to Thorburn and Kocher, an outward rotation and abducted position of the arm with a flexed elbow joint, is characteristic of disease of the 6th cervical segment, as the muscles centralized in the 5th cervical segment retain their function and produce this posture.

When the inflammation is exclusively limited to the anterior horns of the cervical enlargement, that is to the anterior roots, then a flaccid atrophic paralysis arises, restricted to the arms, if the inflammation is distributed to the long tracts in the white medullary substance, a spastic paralysis of the legs occurs in addition.

A complication of *myelitis with spinal meningitis* is not rare, particularly in the region of the cervical enlargement, as the membranes of the cord are in immediate proximity. Spinal meningitis is recognized from the severe pains in the nape, which radiate to the tips of the fingers, by contracture of the muscles at the back of the neck, by sensitiveness to pressure and rigidity of the vertebral column, and by Kernig's sign (incapability of extending the knee in the sitting posture on account of contracture of the flexors of the knee), as well as by hyperesthesia of the skin and muscles. The spinal symptoms, especially cervical neuralgia and sensory paralysis in caries of the cervical vertebra, may for a long time precede the direct vertebral ones (deformity, pain, gravitation abscess) (Kahler).

In transverse myelitis of the *7th and 8th cervical segments* and also in disease of the *first dorsal segment* (lower arm type) the anesthesia extends to the ulnar region, inner surface of the arm, the back of the hand, the ball of the little finger, the entire extent of the two last fingers and to the second finger (only upon the dorsal surface). Compare the diagram of sensory topography, Fig. 176. The other fingers and the region of the hand may present mild sensory disturbance (hypesthesia), the thumb being least affected. Only in destruction of the *5th and 6th cervical segments* is the anesthesia distributed to the radial side of the forearm which is characteristic of this condition; the anesthesia then involves the entire arm. The neck, nape and occiput, however, remain free as these structures are innervated by the four upper cervical segments. If the cervical cord is only partially affected in its transverse section, the sensory involvement will vary according to the position of the lesion; perhaps the legs may be unaffected and only the arms attacked. Sensation and motion cannot be so accurately limited in myelitis as in our physiologic diagrams, for myelitis does not present an anatomical sharply circumscribed, but a diffuse, inflammatory process. Only in complete transverse disease is it characteristic that the distribution of the sensory paralysis is present from the tips of the toes to the uppermost diseased segment area.

In *cervical cord myelitis* paresthesia and lancinating, radiating pain, from the nape to the arms and tips of the fingers, are not rare. On the other hand in disease of the lower cervical cord the girdle sensation, at the boundary of the thoracic anesthesia in the second intercostal space, is absent. The lower four cervical segments only supply the arms with sensation, so that the sensory area of the 2d dorsal segment immediately adjoins that of the 4th cervical segment at the top of the thorax. Only in disease of the latter are the supraclavicular nerves, which originate from it, involved.

Headache is common in cervical myelitis. One of our patients complained of a tormenting neuralgic occipital pain. The large occipital nerve originates from the upper cervical cord. Hemicrania is also observed in this form of the disease and may be referred to the fibers in the cervical sympathetic originating from the cervical cord.

In lesions of the lowest portions of the cervical cord and of the first dorsal segment a *fall in temperature* sometimes appears, while in disease of the upper cervical cord there is said to be a rise (Thorburn). The latter condition has been observed by Wagner and Stolper even after simple contusions of the nape of the neck. On the other hand Kocher, from his experience with compression of the cervical cord after injuries to the vertebræ, could not confirm this behavior of the temperature with the seat of the lesion in the cervical cord.

The behavior of the *arm reflexes*, in inflammation of the cervical cord, is very difficult to estimate, because they may be absent in normal persons or very difficult to demonstrate. In a unilateral inflammation differences in the condition of the arm reflexes may be of value in the diagnosis. The reflexes of the lower extremities and of the abdomen, in partial involvement of the cervical cord, are mostly increased; only in a complete transverse lesion are the reflexes of the legs absent. Wagner and Stolper even maintain that the presence of the tendon reflexes in the legs excludes a complete transverse lesion. The arm reflexes are usually absent in an inflammation of the cervical enlargement and in this condition all of the muscles supplied by the brachial

plexus are usually flaccid, paralyzed, and atrophic. Sometimes motion is retained in the legs. Such a condition permits us to conclude that the central portions of the cervical cord are especially implicated, while the long tracts in the white substance of the spinal cord are spared.

The condition of the bladder and rectum in cervical myelitis is the same as in dorsal myelitis. Occasionally the appearance of glycosuria has been reported in literature. Priapism has been repeatedly noted in cervical myelitis but may occur in other diseases of the spinal cord situated above the lumbar enlargement, in which the center of erection is located. Bramwell refers priapism to an irritation of the fibers which pass from the brain to the sexual reflex center. According to other authors it is to be regarded as a vasomotor disturbance.

Bulbar myelitis (acute bulbar paralysis) is usually not due to very large foci, since life is destroyed by very small ones. The paralysis is of the degenerative, atrophic form and implicates the region of the nerve nuclei situated in the medulla; it, therefore, attacks (completely or incompletely) the facial nerve, the hypoglossal, pneumogastric and spinal accessory nerves, also the respiratory muscles; especially significant is the paralysis of the lips, the tongue, the pharynx and the organs of speech (anarthria). The paralysis of the pharynx, the threatening involvement of the vital centers of respiration (*noeud vital*, Flourens, 1842) and of the disordered act of deglutition constantly endanger the life of the patient.

2. LUMBAR MYELITIS

The lumbar cord has a length of 5.13 cm. (Lüderitz). It begins at the top of the 10th thoracic vertebra and is about twice as long as the sacral cord. Distally the 5 lumbar segments decrease in size (15.6 mm., 13.0 mm., 10.25 mm., 7.0 mm., 5.5 mm., according to Lüderitz). *Myelitis of the lower half of the lumbar enlargement* (second lumbar segment to the second sacral segment) *paralyzes the motor and sensory area of innervation of the sciatic nerve,* especially all of the muscles from the knee downward (excepting the tibialis anticus). In diseases of the 4th and 5th lumbar segments, which are situated at the height of the 12th thoracic vertebra and the succeeding intervertebral disc, besides the symptoms of loss of function in sacral myelitis (foot and toe paralysis) the following conditions are noted: paralysis of motion in the hip joint in the sense of inward and outward rotation, backward movement of the thigh, and flexion at the knee joint. The antagonistic movements (extension of the knee and flexion of the hip) are retained in isolated disease of the lowest part of the dorsal cord. Peroneal paralysis is particularly severe and prolonged; the same is true of paralysis of the extensor digitorum communis and of the tibialis anticus. Even in the cases of lumbar myelitis terminating in recovery a flaccid paralysis of the foot remains; such patients cannot walk because they cannot lift the tip of the foot from the ground.

When the inflammation is distributed to the 3 upper lumbar segments there is complete paralysis of all of the muscles of the leg.

Myelitis of the lumbar enlargement preferably attacks the central gray substance; *the paralysis is,* accordingly, *flaccid with degenerative atrophy, electric reactions of degeneration, and loss of the reflexes.* The paralysis,

therefore, resembles the neuritic form; it differs, however, from the latter by the segmental arrangement of the motor and sensory symptoms, also by the early incontinence of the bladder and rectum, and the frequent presence of bed-sores.

Added to this flaccid, atrophic paralysis there is soon rapid emaciation of the legs, paralytic contractures, osteoporosis and rarefaction of the bones.

In *disease of the 3 lowest lumbar segments anesthesia attacks the feet and the legs in toto, also the gluteal region, the perineum and striaform zones of the skin upon the posterior surface of the thighs*, therefore, especially the sciatic region. Only in an involvement of the upper half of the lumbar enlargement will the area of the *crural* and *obturator nerves* be affected. The consequence is total motor and sensory paralysis of both legs; the anesthesia involves a line drawn through the symphysis, the crests of the ilia and the 5th lumbar vertebral process.

In complete *destruction of the lumbar cord* all the *tendon and cutaneous reflexes may be absent.* The patella tendon reflex can only be present when its reflex center in the 2d to the 4th lumbar segments is intact. The cremaster reflex, and sensation in the testicles are abolished in disease of the 2 upper lumbar segments. The Achilles and plantar reflexes can only be present when the lower lumbar and sacral cords are not affected. The functions of the bladder, rectum, and the sexual organs are for the most part paralyzed early in lumbar myelitis.

3. SACRAL MYELITIS

Myelitis of the sacral cord and of the conus terminalis (this reaches from the 3d sacral segment to the *filum terminale*) occurs particularly after trauma, such as falls upon the buttock (conus hemorrhage). All of the 5 sacral segments, according to Lüderitz, are only 2.11 cm. in length (measured in the cadaver of a female, aged 33); they are, therefore, shorter than any single one of the middle thoracic segments (5.–9.), their length varying between 2.25–2.4 cm., while the length of a single sacral segment, according to Lüderitz, is only from 0.35–0.42 cm. In the slight transverse and longitudinal extent of the sacral cord, which is included within the space of the first lumbar vertebra, there can hardly be question of an isolated disease of a single sacral segment from a clinical standpoint. We shall, therefore, consider together the motor, sensory, and reflex signs of loss of function, in discussing disease of the sacral cord.

Motor symptoms appear only in disease of the *second sacral segment and those above*, and especially involves the *ankle joints*, while the knee joints are usually but little or not at all affected. The free, hanging foot shows the picture of peroneal paralysis, but all the other motor functions, dorsal and plantar flexion, pronation and supination, as well as extension and flexion of the toes, are rendered very difficult. With an involvement of the lowest lumbar segment, the nerve root of which belongs to the sacral plexus, outward and inward rotation of the thigh, flexion of the knee joint (semi-muscles, biceps; their function should be tested in the lateral position or the abdominal posture) may be paretic. The extensor quadriceps, the sartorius, psoas, and the adductor group retain their function. With support such patients may still walk, but their feet are dragged along like a prothesis. Rising upon the tips of

the toes, especially raising the external border of the foot, is exceedingly difficult on account of peroneal paresis. The paralysis is flaccid, without or only with very slight muscle contractions; there is degenerative atrophy with abolished reflexes and DeR or even with entire loss of electric reactions.

The *sensory disturbance*, in case only the last two sacral segments are attacked, includes the anus and the anal region. In a *lesion of the 3d sacral segment* the "*riding breeches*" *form of anesthesia* is characteristic (buttocks, perineum, posterior surface of the thigh). The skin of the scrotum is also anesthetic (see Figs. 169 and 170). The testicles retain sensation within the anesthetic scrotum, as these organs are innervated by the upper lumbar segments. If the disease distributes itself to the 2 upper sacral segments anesthesia of the sole of the foot and of the posterior surface of the leg arises. In a complete lesion of the sacral cord the anesthesia implicates the entire region of the buttocks, coccyx, the anus, perineum, the external portions of the genitalia, the mucous membrane of the rectum, the bladder and urethra (absence of strangury, loss of sensation upon catheterization, and upon the passage of feces); besides the anesthesia is distributed to the posterior side of the thigh, to the external surface of the lower leg and to the lateral half of the foot. The medial half of the dorsum of the foot and of the sole may be hypesthetic in disease of the sacral cord so that the entire foot presents motor and sensory paralysis.

The *Achilles tendon reflex* and that of the *sole of the foot are abolished early* in sacral myelitis, while the patella and cremaster reflexes may be retained. Occasionally *trophic disturbances* develop very rapidly, especially *bedsores* upon the heel, the ankle, and the sacrum.

The *bladder* and *rectum*, as well as the *sexual power*, are early paralyzed in sacral myelitis. What is true of the sphincter ani is also true of the sphincter vaginæ, its center being situated in the 3d and 4th sacral segments. The centers for erection and ejaculation are situated in the 2d–4th sacral segments, usually above those for the bladder and rectum. Therefore in sacral cord disease priapism does not occur, as is sometimes noted in affections of the cervical cord.

A case reported by Neumann is remarkable (*Virchow's Archiv*, 1890, 122. Bd., p. 505), that of a man æt. 37 suffering from a stab wound in the left half of the sacral cord. The penis was paralyzed according to the Brown-Séquard type. Erection was unilateral occurring upon the uninjured side, but this side was less sensitive than the opposite.

The following case (III) is an example of *myelitis of the lumbar cord*: Karl J., æt. 31, married, well until 6 years previously. At that time (1896) he suffered from suppuration of the inguinal glands without there being disease of the genitalia. At about the same time—without the patient being able to assign a cause—an ulcer appeared upon the lower lip and later enlargement of the glands of the neck took place. There was no eruption; systematic inunction treatment.

His present illness began in December, 1900, with constipation and lancinating pains along the course of the sciatic nerve, combined with numbness in the right leg. At that time there were, as yet, no disturbances of motion or of the reflexes. The pain distributed itself to the right half of the sacrum and attained such a degree of intensity that it was relieved only by hypodermic injections of morphin and belladonna. In April, 1901, chills were frequent. In May, 1901, paralysis began, first in the toes of the right foot. In addition there was severe sciatica even upon the left side, as well as retention of urine and cystalgia. The paralysis in the succeeding 4 weeks involved both

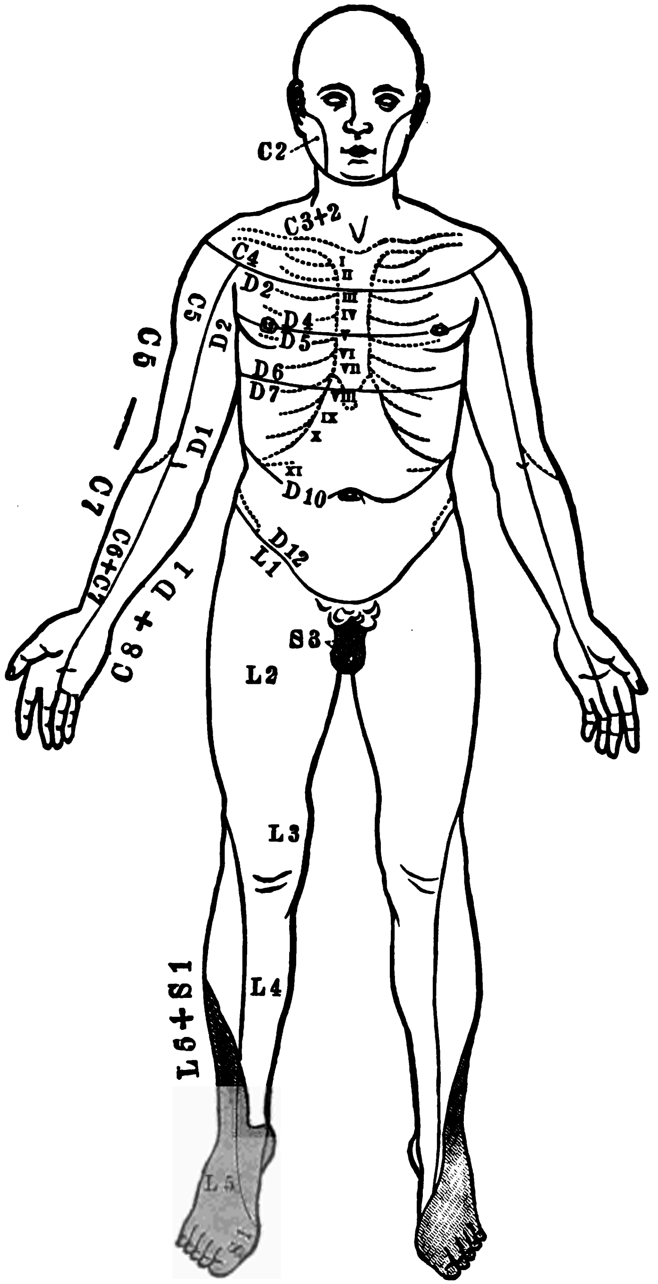

FIG. 169.—DISTRIBUTION OF TACTILE AND THERMIC ANESTHESIA AND ANALGESIA (SHADED LINES DRAWN IN SEIFFER'S DIAGRAM) IN A CASE OF LUMBROSACRAL MYELITIS.

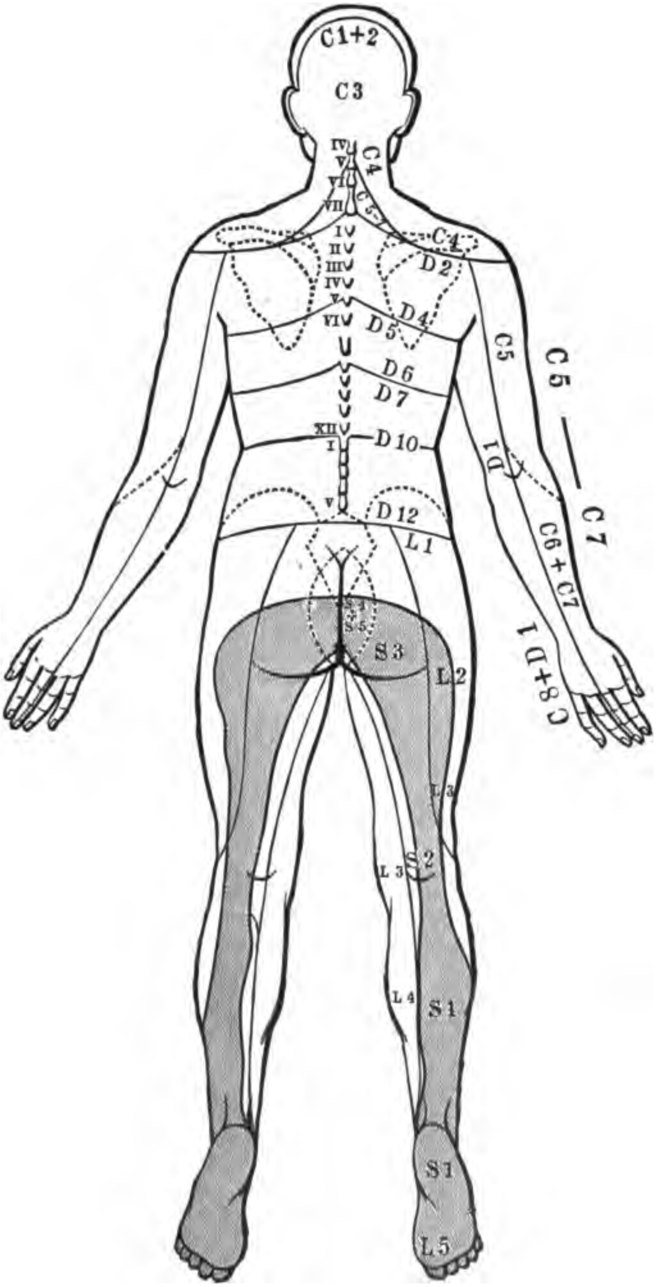

Fig. 170.—Posterior View of Fig. 169.
(For caption see page opposite.)

ankle and knee joints. Since that period walking was impossible. The paralysis was flaccid, and atrophy of the muscles of the leg soon appeared (June, 1901). The following 6 months the patient was in the hospital. Systematic use of the catheter, faradization, galvanism, massage, and another mercurial inunction treatment were without success. The pain increased in December, 1901, especially in the left leg, which became completely paralyzed. Spa treatment in Wiesbaden brought about no relief and for this reason the patient was admitted to the Charité upon July 21, 1902.

Status præsens. A powerfully developed man who can only move forward with great difficulty supported by two crutches. Internal organs normal as is also the temperature. *Complete flaccid paralysis of the entire musculature of the left leg and of the muscles of the knee and ankle joints upon the right side.* In the recumbent posture only slight flexion and adduction of the right hip joint are possible. The muscles of both legs, especially the left quadriceps and gastrocnemius, are very atrophic. The circumference of the thigh in the upper third upon the right side amounted to 46 cm., upon the left 39 cm.; in the lower third upon the right 37 cm., upon the left 35 cm. The circumference of the calf upon either side was 28 cm.

Faradic stimulation of the entire left lower extremity, even with maximum currents, produced no contraction neither of nerve nor muscle; only the adductor longus revealed a faint contraction. The faradic contractility of the muscles of the right thigh was quantitatively diminished but from the knee joint downward it was completely abolished.

There was no response to *galvanism* of the entire left limb, with indirect as well as with direct stimulation, even with very strong currents. In the right thigh only with the strongest currents (40–50 Ma.) with direct, as well as with indirect, applications could rapid contractions be obtained. The formula was normal. In the right leg only the extensors and the peronei muscles reacted to the same strength of current; the muscles of the calf would not contract.

Sensation in all of its qualities (tactile, temperature, electric, and pain) was totally abolished. The loss of sensation was distributed almost symmetrically to the buttocks, scrotum, the posterior surface of both legs and to the feet *in toto.* In the last named area the patient complained of spontaneous lancinating pain (*anæsthesia dolorosa*). The urethra was also anesthetic. *Retentio urinæ et alvi.* The left leg showed a slighter disposition to sweating than the right.

The reflexes of the sole of the foot as well as the cremaster, of the patella and of the Achilles tendon were abolished bilaterally; the arthritic sense was lost upon both sides. Lumbar puncture, under low pressure furnished about 10 c.c. of clear fluid; microscopic and bacteriologic examination was negative. Bed-sores upon the sacrum. The spinal column was not sensitive to pressure.

X-ray examination revealed 4 *osteoporotic* foci in the center of the 12th dorsal vertebra, and the 3 upper lumbar vertebræ, which could be recognized from the flaky, white clarification within the vertebral shadow. An X-ray photograph of the left hand and foot revealed a distinct difference in the structure of the bones. The bones of the hand were normal, while those of the foot showed osteoporotic changes in the bones of the paralyzed members.

The treatment consisted in systematic exercise in the bath, and galvanism with strong currents. In addition carbonated baths, exercise with apparatus, sand baths, bloodless nerve stretching, injections of corrosive sublimate and iodipin were employed. The severe paroxysms of pain in the regions of the sciatic and peronei nerves, and in the left tendo Achillis only yielded to hypodermics of morphin.

Under this treatment, to which the patient submitted for 6 months, the functional disturbances improved somewhat—although very slowly. The neuralgic attacks became rarer and less intense. The boundaries of the anesthetic zone narrowed somewhat, especially upon the buttock and right thigh; analgesia gave way to hypalgesia, the arthritic sense returned. The bed-sore healed. The movements of the hip joint (extension and flexion), especially of the right side, increased, and in the right knee joint slight active motion appeared. Galvanic contractility improved to the extent that all of the nerves and muscles—excepting the left crural and sciatic—reacted to strong currents (23 Ma.).

There remained a *flaccid, atrophic paralysis of the left knee and ankle muscles and of the muscles of the right ankle joint; complete anesthesia upon the posterior surface of both legs and also upon the [illegible] and sole of the foot.* Orthopedic apparatus (see Fig. 175) adjusted to the 3 joints enabled the patient to walk in a walking-frame, later

upon 2 canes, and finally when he was discharged from the hospital (March 24, 1903) with one cane, or with support upon one side—although the gait was dragging—but nevertheless with surety, even in ascending steps. He has resumed his former occupation and has been attending to his duty continuously for 2 years.

The typical, segmentary, symmetric distribution of sensory disturbance, the bladder and rectal symptoms, the absence of the reflexes and the bilateral, flaccid, atrophic paralyses in our case present the clinical picture of a subacute-chronic disease of the lumbosacral cord. In regard to the difference in the implication of the musculature—on the right side the region of the sciatic, on the left the sciatic and crural, nerves were involved—the focus was situated in the motor areas of the cord asymmetrically, and upon the left (to about the II lumbar segment) higher than upon the right side (to about the IV. L. S.). The distribution of the anesthesia corresponds to a lesion of the 4th lumbar segment downward. The history of the case just described, therefore, is that of a myelitic focal disease (of syphilitic origin), which had extended from the 2d lumbar segment to the conus medullaris.

4. DISEASES OF THE CAUDA EQUINA

may also give rise to flaccid paralysis of the legs, anesthesia in the distribution of the sacral, the pudendal, and coccygeal plexuses and in the areas of the sciatic, crural, and obturator nerves. The bladder and rectum may be incontinent and the sexual power may be lost. A focus that has involved all of the nerves of the cauda equina—which pass downward from the 2d lumbar segment—may, therefore, produce the same clinical picture as a lumbosacral medullary focus.

The differential diagnosis between a root and medullary lesion is, therefore, very difficult in this region. In favor of disease of the cauda equina is the unilateral appearance and the limitation of the symptoms of absence of function to a nerve or functional area. In the cauda the sensory roots are dorsally, and the motor, ventrally situated; only outside of the dura do they unite and form the mixed peripheral nerve. If the peripheral, caudal filaments are affected then there is paralysis of the crural and obturator nerves (paralysis of the adductors and quadriceps, extensor surface of the limb). If the deeper and more central fibers are attacked there is then functional disturbance in the distribution of the sciatic nerve (the foot and flexor surface of the knee joint).

Further, in favor of disease of the cauda—corresponding to the more marked development of the sensory caudal roots—is the marked alteration of sensation and particularly the predominance of sensory, irritative phenomena: paresthesia, radiating pains (root neuralgia). Even *analgesia hyperæsthetica* or *anæsthesia dolorosa* may appear, that is, pain in portions of the body that are objectively without sensation. In contrast to this, in medullary lesions, motion suffers sooner and to a greater extent than sensation. Only in lesions of the anterior root are there muscular twitchings, spasm and finally paralysis with rapid, progressive, degenerative atrophy. The destruction of a few roots may, however, occur without symptoms, as almost every muscle and every cutaneous region is supplied by at least 3 roots. While a lesion of the posterior cauda roots is distributed to the entire sensory functional area, in incomplete

medullary lesions, sensation may be retained in some or even in all of its qualities. *Dissociated sensory paralysis,* for example, analgesia and thermoanesthesia with retained tactile sense, and also the arrangement of the *sensory symptoms in symmetric, segmental zones* are in favor of the medullary seat of the disease.

Finally we must mention that in trauma the cord is sooner involved than the nerve roots which are more resistant and that in the central medullary portions, rich in vessels and poor in connective tissue, extravasation of blood is more likely to occur than in the white substance. *Central myelitis is, therefore, very often more intense than inflammation of the white substance of the cord.* Partial, transverse disease corresponds to an incomplete paralysis; sensation may be retained or only disturbed in part, while motion is completely abolished.

ETIOLOGY OF ACUTE MYELITIS

Spontaneous development of acute myelitis without a demonstrable etiology, as was formerly believed, is now generally rejected. According to recent investigations of the spinal cord, especially bacteriologic, and of the cerebrospinal fluid, *infections* and *intoxications* play the most important rôle among the etiologic factors in acute myelitis (compare the references of Strümpell and Redlich upon acute myelitis at the 19th Congress on Internal Medicine, Verh. d. Congr. 19. Band, 1901). In this disease accordingly there may be either a primary *entrance of bacteria into the cord,* or the lesion is due to the action of their toxins, or the effect of chemical or metallic poisons.

The paraplegias occurring in the course of infectious diseases belong to the first group in which the pathogenic agents are found in the myelitic foci. *Microörganisms* have been repeatedly demonstrated in the *inflamed spinal cord,* such as the staphylococcus albus et aureus, streptococci, pneumococci (Fürstner), anthrax bacilli (Marinesco), typhoid bacilli (Curschmann), and tubercle bacilli (in tuberculous myelitis or in tuberculosis of the spinal cord).

More often bacteria have been found in the *spinal fluid* especially pyogenic organisms and tubercle bacilli. In the spinal fluid of infectious poliomyelitis, Schultze found the Jäger-Weichselbaum meningococcus. Myelitis may arise after *whitlow,* as reported by Strümpell, and in this case the staphylococcus albus could be demonstrated in the cerebrospinal fluid.

The microörganisms find their way into the spinal cord partly from the central canal, partly from the subarachnoid space (Marinesco), that is, from the cerebrospinal fluid, by way of the blood and lymph (Babes). The capillary spinal cord emboli described by v. Leyden also belong to this category; these were noted in the course of ulcerative endocarditis. In acute marginal myelitis, in the course of epidemic cerebrospinal fever, Leyden was able to demonstrate the point of entrance of the cocci along the septa in the medullary substance.

We must also remark that in general septic processes, bacteria for example, streptococci have been found in the cerebrospinal fluid, without pathologic lesion being present in the cord or in the membranes. The bacteria are present in the circulation and find their way into the secretions and excretions, as into the urine (bacteriuria), without producing changes in the kidney through

which they pass. Only upon the *demonstration of products of inflammation* (increase of lymphocytes and leukocytes, intracellular bacteria) is the view justified that the spinal cord or its membranes are directly affected.

It has been possible *experimentally* by the introduction of bacteria (especially streptococci, staphylococci, pneumococci, typhoid bacilli and those of diphtheria), into the subarachnoid space of animals or into the circulation, to produce meningitic processes. The changes in the ganglion cells are worthy of note (clearing and swelling of the cells and their processes, as well as atrophy of Nissl's bodies) which were produced experimentally by Goldscheider and Flatau in superheated animals. F. Brasch was able to show the same lesions in man, with a pre-mortal continuous rise of temperature to over 105½° F. Langworth Nichols observed pathologic changes (central and peripheral chromatolysis) in the anterior horn cells, as well as in the spinal ganglion cells in cases of enteric fever and also in rabbits infected with typhoid bacilli; the chromatic substance in the diseased cells may return to the normal condition.

Myelitis may be a sequel of almost all infectious diseases and all three forms may appear: the *myelitic focus* (transverse paralysis), *poliomyelitis* and not rarely *disseminated myelitis,* i. e., *sclerosis.*

Among the most frequent *infectious diseases* in which during, or after the febrile course has passed, myelitic processes may be observed are variola (Westphal, Leyden and others), enteric fever (Salzwedel, Leyden, Ebstein, Curschmann, Schiff, Lépine) diphtheria, influenza, pneumonia, dysentery, sepsis and pyemia, puerperal fever, malaria, erysipelas, gonorrhea; these are mostly combined with meningitis (Leyden, Ullmann, Debove, Hayem, Spillmann, Herzog). *Angina tonsillaris* may, as in the case reported by Hoche, give rise to myelitis. As is well known the tonsils are a common port of entrance for different infections; we need only refer to suppurations and tuberculosis, acute articular rheumatism, peliosis rheumatica, plague, nephritis, endocarditis and influenza.

Among the infectious diseases just enumerated from which spinal cord disease originated, the pathogenic agent could very rarely be demonstrated. These negative findings render it very likely, that not alone the bacteria, but the toxins produced by them, are the agents which produce inflammation of the spinal cord. The spinal cord inflammations, evoked experimentally in animals, are in favor of this view. The forms of myelitis produced experimentally in animals by the introduction of bacteria into the subarachnoid space, upon examination often failed to reveal the presence of bacteria; they often disappeared in the course of a few days (Marinesco, Hoche); on the other hand, by the injection of toxins, such as diphtheria toxin (Henriquez et Hallion, Crocq), streptococci toxin (Homen), spinal cord inflammation was produced. Occasionally myelitis may appear as a primary disease and this is favored by the observations of Oppenheim and others.

Tuberculosis and *syphilis* (the hereditary and the acquired form) are relatively frequent causes of myelitis; these diseases being the most important ones among the chronic infections. Pochhammer calculates the frequency of syphilis in the history of myelitis at 64 per cent. Syphilitic myelitis occurs either as primary hyperplastic pachymeningitis or as a chronic cerebral or spinal cord disease (encephalomyelitis luetica). In the latter form there is

commonly spastic paraplegia, paralysis of the ocular muscles, and of the optic nerve, as well as other cerebral symptoms (aphasia). Syphilitic myelitis is not always of an inflammatory nature; often there is a gumma of the cord or its membranes; further a primary syphilitic endarteritis of the spinal vessels is also capable of giving rise to myelitis. Repeatedly the appearance of diffuse myelitis has been noted after an attack of acute poliomyelitis which had occurred years previously. This is due to a rekindling of the inflammation in the cicatrized anterior horns (W. Hirsch).

Next to bacterial, *chemical* and *metallic poisons* produce inflammatory changes in the cord. Thus circumscribed and disseminated, especially chronic, forms of myelitis have been noted after *lead, chloroform, carbonic oxid* and *illuminating gas poisonings,* the last especially in firemen. Here also myelitis, due to *pellagra,* must be included which occurs among the people of Italy and South Russia who eat much maize.

Of great importance, particularly in regard to accident cases, is myelitis occurring after *trauma.* All injuries and diseases of the vertebral column, especially *tuberculous spondylitis* and *carcinoma of the vertebral column,* as well as *scoliosis* and *kyphosis* with marked deformity, may give rise to lesions of the cord (compression, hemorrhage, laceration, inflammation). The micro-organisms present in the circulation may colonize upon the extravasated masses of blood. In addition to these traumatic disturbances, as well as to the ischemic necroses of the cord (after embolism, thrombosis, compression), reactive processes may occur (round cell infiltration, fatty granules), so that there is no difference between the lesions arising in *acute myelitis, traumatic myelitis* and *compression myelitis* (Fr. Kraus, *Zeitschr. f. klin. Med.,* Bd. XVIII, 1891, Case I, cervical focal myelitis after fracture of the 5th cervical vertebra).

It is important to know that hemorrhage, as well as acute degeneration, and inflammation, may arise from shock to the vertebral column without injury to the vertebræ (*contusion myelitis*). The post traumatic development of progressive degeneration, such as *chronic myelitis, syringomyelia, multiple sclerosis,* has been repeatedly reported after blows, railroad accidents (railway-spine), without there being external signs of injury. Changes develop in the cord after some time, months or even years, similar to the condition in late cerebral apoplexies (compare the reports of Stadelmann, Langerhans) which must be referred to the progress of the original shock which produced the spinal lesion. The investigations of Minor also teach that contusion of the spinal cord may result in anatomical lesions in the form of small hemorrhages. If shock is capable of tearing a blood vessel, the more sensitive nervous tissue will suffer much more readily.

This is also proven by Schmaus's experiments. He placed a hard rubber plate, which he struck repeatedly with a hammer, upon the vertebral column of rabbits; finally paresis of the hind legs appeared after which the animals were killed and their spinal cords examined. There were found, partly diffuse, partly system-like degenerations, myelitic softening, hydromyelia and gliosis. Schmaus, quite correctly, emphasizes that *contusion of the cord not only produces lesions in a few vessels, nerve fibers, and ganglion cells, but that finer changes may be the result which become obvious only later on, as post-traumatic pathologic conditions.* Shock of the spinal cord, therefore, according to

Schmaus, may give rise to traumatic necrosis of the axis cylinders and of the cells with arrest of their function, either by "decomposition" of the molecule, i. e., a change of position of the individual atoms, or, that from a definite strength of the irritation of the mechanical nerve stimulation the nerve fibers are not only paralyzed, but from the excess of the stimulation they are destroyed. This necrosis may, according to Schmaus, produce secondarily, from destruction and reactive hyperplastic processes, distinct column degeneration, softening, cavity formation and gliosis. Therefore, according to experimental and clinico-anatomical facts at hand, the *possibility of development of myelitis after shock of the vertebral column* must be recognized.

Caisson paralysis, observed in tunnel workers, which may be assigned to "internal trauma," may be included with traumatic myelitis. In 1879, v. Leyden had an opportunity of observing and reporting such paralyses; in the dorsal portion of the cord, especially in the posterior columns and posterior lateral columns, he found numerous small foci of softening and referred these to tears in the medullary substance, which had arisen from the escape of gas bubbles, in consequence of rapid decompression. Recently v. Schrötter, Mager and Heller, have published a voluminous book upon caisson paralysis and have reported quite a number of such air-pressure paralyses; they refer them to ischemic necroses, especially of the white medullary substance. With rapid decompression, engorgement of nitrogen gas and extravasation into the blood vessels occur, because the lungs are incapable of giving off the nitrogen rapidly enough. The extravasation of gas into the blood vessels produces *gas embolism* which has been repeatedly demonstrated in the mesentery, the heart, and the central nervous system. This *gas embolism of the spinal cord arterioles*, according to the previously mentioned investigators, produces multiple focal necroses symptomatically most often indicated by paraplegia.

In connection with trauma are those forms of myelitis due to *inflammatory processes which ascend from the periphery* along the cellular tissue or the nerve trunks. The development of myelitis from *ascending neuritis* and *polyneuritis* has been proven repeatedly, both experimentally and clinically, for example, after trauma and erosion of the peripheral nerve trunks.

Exertion is also noted among the etiologic factors of myelitis; as a rule exertion is only a secondary cause as with a preexisting syphilis. Fürstner was able, however, by his "rotary movements" to produce degeneration in both lateral and posterior columns in dogs. According to Edinger's ingenious "function theory" as well as from his experiments on rats, an immoderate employment ("over-exertion") of the nerve tracts may produce such damage that atrophy of the nerve substance results and the glia tissue is destroyed. He produced experimentally by over-exertion in rats, degeneration of the posterior columns, such as are characteristic of tabes.

The etiology of those cases of myelitis is less clear in which *refrigeration, sexual excess,* and *great mental emotion* are mentioned as causes. Myelitis after severe exposure to cold (wetting, effects of snow, etc.) has been demonstrated clinically and experimentally in rabbits by forced application of cold to the vertebral column. Not infrequently the actions of cold are combined with those of exertion or infection.

Finally myelitis has been reported after *psychical effects*, especially *fright.* Such cases have been described by v. Leyden in his "*Klinik der Rückenmarks-*

krankheiten," and later in the *Zeitschrift für klinische Medicin*, Bd. I, 1879. In the latter, there are illustrations of the histologic changes in the cord in a case of *myelitis due to fright*; the patient was a servant girl, æt. 21, who was frightened by a fire occurring during the night. A few days later a paralysis of the legs developed, which gradually increased in severity, and finally after several months, terminated fatally. The histologic examination of the cord revealed the picture of chronic myelitis in the middle thoracic cord, especially involving the posterior and lateral columns, as well as the posterior horns. Similar cases have been reported by Köhts and Brieger.

The pathogenetic connection between *psychical shock and myelitis* is not easy to explain. Great emotion may produce such an acute vascular contraction (vasomotor reflex), that nutritive disturbances arise in the delicate structure of the cord. It is further possible that psychical shock causes an intense increase of blood pressure, which, with a co-existing arterial disease analogous to the condition in the brain, causes a spinal cord apoplexy. Quite often in addition to "psychical trauma" other etiologic or predisposing factors can be discovered.

It must still be mentioned that many conditions which debilitate the organism predispose to myelitis. Among these are the paraplegias in *pernicious anemia* (Minnich), of *leukemia,* of *carcinomatous cachexia,* and of the *climacterium.* Myelitis has been observed several times during *pregnancy* and in the *puerperium*; possibly infectious processes also are here concerned.

Myelitis occurs in *both sexes to the same extent.* In regard to *age,* acute inflammation of the anterior horns is much more common in children, while typical, acute myelitis is more usual during middle age. In the aged, paraplegia sometimes appears in connection with incontinence of the bladder. These are sometimes due to *endarteritic processes of the arterioles of the spinal cord,* occasionally there may be compression, the result of a kyphoscoliosis or of a deforming, anchylosed spondylitis.

DIFFERENTIAL DIAGNOSIS OF MYELITIS

We must propound 3 questions in paraplegia:

(1) What is the *etiology and the pathologico-anatomical nature* of the disease, especially is there an *extramedullary process* present (tumor, lesion of the bone)? This is the *etiologic* or *pathologic diagnosis.*

(2) Which segments are affected (*segmental diagnosis*)?

(3) Which parts of the segment are involved—poliomyelitis, myelitis transversa vel disseminata, leukomyelitis—(*transverse section diagnosis*)?

Topographical diagnosis has been described under segmental localization and also under the special forms of myelitis; we shall, therefore, devote ourselves to the *pathological diagnosis.* The clinical picture of myelitis may be produced by any transverse lesion of the cord, such as *hemorrhage, compression, tumor or trauma.* Later the differential diagnosis from meningitis, neuritic and psychical paralyses must be considered. In regard to the differentiation of myelitis from spastic spinal paralysis and from multiple sclerosis, we may refer to the chapters upon these diseases in this volume by Professor Redlich.

Hematomyelia—hemorrhages into the pia, into the arachnoid, *apoplexia canalis spinalis,* rupture of a cerebral hemorrhage into the ventricles or of

an aneurysm into the spinal canal—may have an apoplectiform onset in common with acute myelitis. Usually prodromes precede the onset of myelitis for some time, which are absent in primary spinal cord hemorrhage. In favor of the latter affection are a preceding injury, as well as diseases of the blood and of the vessels. The result of lumbar puncture may be of decisive importance as when the cerebrospinal fluid is admixed with blood; recent hemorrhages, due to lesion of a dural vessel consequent upon injury during puncture, must not be confounded with these. In favor of the clinical picture of hematomyelia, accurately described by L. Minor, in a unilateral affection, is the Brown-Séquard type of anesthesia; when both halves of the gray substance, as in the lower cervical cord, are implicated, atrophy appears in the arms; in the trunk, and in the legs; simple pareses without atrophy, and dissociated sensory paralysis are noted. The pathologic picture of hematomyelia may, therefore, resemble central gliomatosis (syringomyelia).

Paraplegia from embolism of the aorta also appears suddenly; but in this condition the vascular disturbances are most prominent—cessation of femoral pulsation, ischemic tissue necroses, etc.

In *spinal meningitis* the severe pain, the stiffness of the vertebral column, tonic tension of the muscles of the trunk and extremities, Kernig's symptom, and the fever are prominent in the clinical picture, while the signs of paralysis, at least at the onset of the disease, are ill defined. The discovery of an increased number of *lymphocytes* and *leukocytes* and especially of *intracellular bacteria* in the cerebrospinal fluid is decisive.

A *unilateral paralysis* (Brown-Séquard) in consequence of partial medullary disease, can hardly be confounded with cerebral hemiplegia; in the latter the characteristic bladder, rectal, and trophic disturbances of the cord lesion are absent, and, on the other hand, the typical, segmentary, sensory disturbances only occur in Brown-Séquard's paralysis.

The differential diagnosis between *myelitis* and *compression of the spinal cord* may be exceedingly difficult, if, externally neither by inspection, palpation nor percussion (local pain, stiffness, deformity), a disease of the vertebra can be demonstrated. In *disease of the vertebra* the history is most important. Thus in young persons who present signs of scrophulosis or tuberculosis in other organs, a tuberculous spondylitis is thought of while in a paraplegic woman, in whom a brief time previously a carcinomatous breast has been removed, a vertebral metastasis should be thought of, especially if intense pains are present. Sometimes the vibration of the tuning fork is experienced as pain over the diseased vertebra. In caries of the vertebra in children, according to Finkler, in about one-half the cases the local pressure pain is absent; the lumbar vertebræ are most often affected by caries. The first symptoms of medullary compression are often pareses and root neuralgias. The sensitiveness to pressure of the sacral vertebræ upon palpation through the rectum is also in favor of an extramedullary seat of the disease, particularly if a mass can be felt (tumor, callus).

Of great significance is the *X-ray finding* in regard to differential diagnosis. Leyden and Grunmach [1] several years ago published a number of *radio-*

[1] "Die Röntgenstrahlen im Dienste der Rückenmarkskrankheiten." *Arch. f. Psychiatrie*, Bd. XXXVII, Heft 1 (1903).

scopic examinations in disease of the spinal cord. These proved that even slight changes in the bones and displacements of the vertebræ, which were impossible to demonstrate by means of former methods, are revealed with surprising clearness by radioscopy. On the other hand primary medullary diseases could not be recognized by the X-ray with sufficient clearness. But the negative X-ray finding is also of value, as it renders it extremely likely that the myelitic disease is independent of an affection of the vertebræ. Grunmach observed, in affections of the transverse section of the cord, an osteoporosis of the vertebræ and of the members downward from the focus, corresponding to the distribution of the lesion; he believed, therefore, that it was possible to determine, from the distribution of the osteoporosis, the height of the lesion in the cord. Radioscopy of the paralyzed limbs is also important in so far as the abnormal shadow of the bone indicates *osteoporosis,* that is, the anatomical nature of the malady, and indicates also the danger of a spontaneous fracture. Osteoporotic bones are often anesthetic (test by means of vibration of the tuning fork).

If there be suspicion of medullary compression, in consequence of *tuberculous spondylitis,* when pain upon pressure, stiffness, and deformity of the vertebral column are absent (Pott's curvature), the diagnosis may be made by the demonstration of tuberculosis in other organs (lungs, lymph glands), further by the presence of tubercle bacilli in the cerebrospinal fluid (*pachymeningitis tuberculosa*), or by the positive result of the tuberculin reaction, provided there is no other tuberculous focus present.

Difficult and even impossible at times is the differentiation of myelitis from an *intraspinal tumor.* In favor of an *extramedullary* seat of a tumor are the signs of compression of the nerve roots; these consist of intense *neuralgic pains* (intercostal neuralgia, sciatica), which are commonly unilateral and only later, when the tumor presses upon the other half of the cord, also implicate the other side. At the onset *motor,* usually *spastic paralysis is limited to one side of the body.* The objective sensory disturbances are not so well defined in compression of the cord as in a primary transverse disease. In addition there is sometimes *sensitiveness of the vertebral column to palpation and vibration.* The slow progression of the paralysis to the opposite side while the localization in height remains the same, except when there are multiple tumors, which may then produce the clinical picture of multiple sclerosis, is characteristic of an *extramedullary* tumor.

An *intramedullary* seat of the tumor, according to its extent, is favored by the symptom-picture of a unilateral lesion or complete transverse paralysis; further its location in the cervical cord where primary myelitis is much rarer. With an intramedullary seat of the tumor pain may be entirely absent (compare the article by Professor Schultze in this volume).

The presence of *particles of the tumor in the cerebrospinal fluid* will render the diagnosis positive. Also in meningomyelitis of syphilitic origin, as well as in *pachymeningitis gummosa* the results of lumbar puncture may be of importance. The presence of *numerous lymphocytes* in the cerebrospinal fluid is greatly in favor of the syphilitic nature of the disease.

Landry's paralysis may give rise to confusion. This affection may appear in the form of an acute polyneuritis or as an acute degeneration of the motor cells in the anterior horns and medulla oblongata (bulbar variety). An *acute*

ascending, occasionally also *descending paralysis*, of all nerve tracts is characteristic. This commonly develops with fever, beginning with complete, flaccid paralysis of the legs, which, in a few hours or days, involves the muscles of the trunk, the arms, the nape, the face, and finally the muscles of speech, of deglutition and of respiration. The paralysis is flaccid often combined with DeR; the tendon reflexes are abolished. Pain and sensory disturbances may be absent; the functions of the bladder and rectum are normal. Sometimes the symptoms of a general infection are still present—fever, enlargement of the spleen, albuminuria. Death results in a few days or weeks from paralysis of respiration (Cheyne-Stokes' type), or the paralysis improves; recovery has several times been reported.

In the differential diagnosis the decision of the question is very important, in the given case, as to whether the paraplegia is due to a disease of the cord (myelitis) or to a peripheral affection (multiple neuritis, polyneuritis). The latter malady was formerly included with spinal paralysis. In my (Leyden) various publications (*Zeitschr. f. klin. Med.*, Bd. I, 1880, and also in the *Deutsche militärärztliche Zeitschrift*, 1888: Die Entzündung der peripheren Nerven: Polyneuritis—Neuritis multiplex) I described the clinical differences of neuritic and spinal paralyses and the clinical picture of polyneuritis.

In favor of a disease of the *peripheral nerve trunk* or of the *root* within or without the dura, are *marked sensory symptoms* with decided irritative phenomena, paresthesia, pressure points, lightning pains, root neuralgias. Typical of a lesion of the posterior roots is *analgesia hyperæsthetica*, that is, *anæsthesia dolorosa*, with absent motor disturbances. The interruption of the posterior roots on the one hand causes an arrest in the conduction from the periphery to the center, and on the other hand the irritation of the lesion in the center of the roots causes pain, which, in regard to its development, may be compared to the pain projection in amputated members. In *medullary disease*, however, *motion suffers sooner and to a greater extent* and it may even be involved alone, with sensation completely retained. Further, in a medullary disease, the paralysis is distributed to the entire area below the point of lesion; thus in an affection of the cervical cord to all four members, the trunk, the bladder and the rectum. Paralysis below the lesion is commonly rigid with increased tendon reflexes and contractures.

In contrast to this, a *disease of the roots*, for example, those passing out of the cervical enlargement, gives rise to disturbances exclusively within the region of distribution of the affected roots, such as a flaccid paralysis and neuralgia in the arm, while the trunk and the legs are not involved. In an incomplete medullary disease, only a portion of the member may be attacked. In this condition, however, in an anterior horn affection, motion is lost and sensation retained or in an incomplete affection of the posterior column there is *dissociated sensory paralysis*, as the spinal cord fibers which supply the various qualities of sensation run in tracts separated from one another. For this reason *dissociated sensory paralysis*, for example, *analgesia* and *thermoanesthesia* with retained tactile sense, as well as the presence of sensory disturbances in definite zones, is characteristic of an *incomplete medullary disease*. In contrast to this the sensory disturbances in *peripheral disease* involve all *sensory qualities* and extend to a definite cutaneous area. As is well known, in disease of a peripheral nerve, for example, in alcoholic neuritis, degenerative

atrophy, motor paralysis, DeR, and the abolished reflexes are limited to the distribution of the affected nerve trunk, while myelitic paralysis does not confine itself to the area of innervation of the peripheral nerve.

In a complete transverse disease of the cord the reflexes may also be absent; but then the other symptoms of transverse disease are unmistakable, especially the bladder and rectal disturbances which are only exceptionally affected in a pure neuritic or polyneuritic lesion. The arrangement of unilateral motor, and contralateral sensory, paralysis (Brown-Séquard), further the appearance of priapism and the demonstration of extramedullary diseases (spondylitis, tumor) also favor the central seat of the paralysis. The differential diagnosis between myelitis and disease of the cauda equina has been detailed elsewhere (see page 423). We must also refer to the article in this volume by Professor Bernhardt upon paralysis of the peripheral nerves.

Confusion of myelitic, with *hysterical,* paralysis has occurred several times. *In favor of the psychic nature* of the paralysis is the *sudden appearance* of the paralysis after emotion, the presence of other *hysterical stigmata,* the typical distribution of the *hysterical sensory alterations,* the *absence of incontinence of the sphincters* as well as of other objective paralytic phenomena (*degenerative atrophy, DeR*), the *relatively rapid change* in the *intensity of the paralysis,* the *disappearance of the paralysis during sleep* and under anesthetics, *negative Babinski reflex* and the *normal composition of the cerebrospinal fluid.*

PROGNOSIS OF MYELITIS

The function necessary to maintain life (respiration, heart, deglutition) are usually not involved in myelitis. Nor is the pain so intense that it exhausts the strength of the patient. On the other hand, in the course of the first or second week, *complications* on the part of the bladder and rectum may arise, which from their sequels, cystopyelitis and bed-sores, seriously endanger life. To this there is often added psychical depression and physical exhaustion (insomnia, deficient ingestion of food, cardiac asthenia), which according to their intensity increase the anxiety of the physician. The prognosis of myelitis, therefore, *quoad vitam et sanationem* is always serious.

Prognosis depends particularly upon (1) the *etiology,* (2) the *course,* (3) the *distribution* and *intensity* of the *myelitic paralysis,* that is, upon the size and seat of the focus, (4) upon *complications.*

In an *etiologic respect compression myelitis* in *spondylitis tuberculosa,* in caisson paralysis and in myelitis after syphilis or acute infectious diseases, offers a more favorable prognosis than other varieties.

In regard to the *course* the prognosis is naturally more serious in a rapid, progressive paralysis. The more rapid the progress the slower is the stage of regression. The conditions are more favorable as to prognosis when the signs of paralysis are incomplete, *bladder and rectal symptoms are absent* and no *serious complications* (*bed-sores, cystitis*) are present..

In the severe cases of myelitis, only when regeneration begins, is there a hope of improvement, i. e., only when the paralytic phenomena improve, the abolished reflexes return, the flaccid paralysis assumes a spastic character, the bladder and rectal symptoms ameliorate, and the bed-sore shows a tendency to heal. The muscular regeneration is shown by an improvement in the

electric reaction and only in this stage of regeneration is the danger of life-threatening complications less and the hope of a more or less complete recovery established.

In the stage of restitution we must remember that in organic disease of the cord a *restitutio ad integrum* does not occur and that improvement is but very gradual. In the mild cases it takes many weeks even in the favorable ones, and in severe cases many months. It must also be remembered that just as in subacute and chronic inflammations of other organs so also in myelitis there are temporary stages of arrest and even apparent improvement, until spontaneously or from some special cause (refrigeration, exertion, febrile infection) the apparently dormant inflammatory process flares up again; it then destroys the effects of improvement and brings about even a more severe paralysis. This *relapsing form of the inflammation* may produce several *acute relapses* (*myélite à rechutes*).

In regard to the *localization of the focus, cervical myelitis presents the most serious prognosis*, especially when the process ascends to the medulla (*bulbar paralysis*). The larger the focus the more serious the prognosis. The *involvement of the sensory qualities* favors a more *intense disease of the cord.* Small foci may heal without producing marked functional disturbance. For this reason disseminated myelitis, which usually presents small foci, and unilateral lesions, offers a more favorable prognosis than transverse myelitis which is associated with large foci. Extensive disease of the anterior horns, that is, degenerative muscular atrophy with electric DeR due to it, as well as complete, flaccid paralysis or serious contractures, are of unfavorable import. The prognosis of myelitis is rendered more grave by general complications (old age, debilitated constitution, diseases of the heart or lungs, cachexia, etc.), further by unfavorable external conditions of life.

When the symptoms of paralysis have reached a *definite stage of arrest* (stationary stage) then with suitable care the patient may live for many years. It may be stated that in general the prognosis of myelitis, thanks to the advance in early diagnosis and the recognition of the advantages of physical treatment including proper nursing, has improved very much. In the course of the last few years we have repeatedly succeeded in putting many a myelitic in the position of earning his livelihood.

THERAPY OF MYELITIS

(1) PROPHYLAXIS

The prophylaxis of myelitis consists in the avoidance of the etiologic factors. *Immunization* acts in this sense as in the case of diphtheria, hydrophobia (protective inoculations) and in malaria (the administration of quinin). *Caisson paralysis* may be prevented by prophylaxis and therapeutic measures, by sufficient supply of air, and ventilation of the working chambers, as well as by other means. Upon the first signs of caisson paralysis the patient is put back into the chamber, where, by the introduction of compressed air, the pressure of the air is increased to the degree in which the patient had labored in the caisson. The introduction of the compressed air, according to v. Schrötter, should be gradual, two minutes for every fifteen pounds

of atmospheric pressure; besides, oxygen is to be administered. *Inhalation of oxygen* often relieves the disorder of the pulmonary circulation and of respiration; it indirectly increases the excretion of free nitrogen from the blood vessels. The patient remains in the "artificial caisson" until the serious symptoms have disappeared, after which the atmospheric pressure is gradually decreased. Since the introduction of State regulations (in Germany) in caisson working (tunnels, bridge building) and of careful observation of the prophylactic measures, caisson paralysis has become exceedingly rare.

First aid, in *injuries to the vertebra,* is most important in a prophylactic respect, such as *expert ambulance service* and *proper surgical treatment* (*extension*). In a similar manner an early rational treatment of spondylitis is effective, that is, before pressure symptoms on the part of the cord appear.

The *avoidance of the predisposing causes of myelitis* acts in the same sense, especially in neuropathically predisposed persons; also in syphilitics or alcoholics (avoidance of immoderate indulgence in various sports, wetting, intoxications, such as lead, alcohol, etc.).

It may, therefore, be observed that the physician can do much in the prevention of myelitis.

(2) TREATMENT

In attempting the treatment of myelitis it is well to remember and also to inform the patient that the disease is a very protracted one, lasting months and not infrequently years.

The anatomical process can rarely be influenced directly, for example, by *specific treatment* in syphilitic myelitis, or by relieving the compression, as in pressure myelitis. In most of the cases our aid is limited to improving the functional disturbances, in facilitating the efforts of healing by proper and careful nursing, which will maintain the strength of the patient and *prevent serious complications.* The last named, the psychical and physical exhaustion, bed-sores and cystitis, and not the disease of the cord itself, are the cause of the fatal issue in myelitis. For this reason the main object of the physician consists in a *prophylactic treatment which will prevent these serious complications,* by a proper control of the *mental state of the patient* and *by careful nursing.* Leyden has made the apparently paradoxical statement that the recovery of the patient often depends upon the fact that we keep him alive, i. e., so that he does not perish from complications. Among these, which may often be avoided by proper treatment, is *inanition*; this is especially true of such a conspicuously chronic disease as myelitis. In a considerable number of paraplegias, who terminate fatally, hyponutrition is the cause of exhaustion and such patients then succumb to complications or intercurrent febrile affections.

The *food* of the patient may be adjusted to his wishes, in so far as this is possible, for, except in the case of intestinal crises, atony of the intestine, and incontinence of the sphincters (limitation of fluid) there are no special indications. The daily amount of food should not be below 2,000 calories, especially during convalescence, for during this time treatment by systematic exercise demands increased exertion on the part of the patient. It is serviceable to vary the diet as much as possible and to increase the ingestion of food by the addition of substances rich in calories, such as honey, malt extract,

or cream (1 liter = 1,920 calories). Fats which are very rich in calories are advantageously given with vegetables (mashed potatoes), milk and rice, or bread. Alcohol had better be limited especially during the acute stage. Naturally an immoderate gain in weight, which renders the restitution of motion difficult, should be prevented.

A difficult and exceedingly important object in the treatment of myelitis is the *patient's posture in bed* especially in regard to the *prevention of bed-sores.* This develops upon those portions of the body that are exposed to prolonged pressure, which in combination with paralysis of motion, of sensation, and of the vessels (vasoplegia) leads to disturbances of the cutaneous circulation; these may be readily recognized from the bluish-red discoloration. Further there is transudation, formation of vesicles, hemorrhage, and even necrosis. The patient is greatly debilitated by the bed-sore; often enough in consequence of complications arising from it (erysipelas, sepsis, embolism, gangrene, rapidly progressing phlegmons) it is the cause of death. *The prevention of the bed-sore is, therefore, a vital question to the patient, and in treatment it is a matter of conscience to the physician;* it is true when there is incontinence of the bladder and rectum this is one of the most difficult problems of nursing.

Bed-sores are prevented by a suitable position in bed and cleanliness, provided that the strength of the body is maintained by suitable nourishment. It is all the more necessary to maintain the power of the heart, for cyanosis, that is, stasis hyperemia, favors the development of the decubitus.

The bed of the myelitic, which, often for months and years, forms his place of rest, should be placed in a room in which there is sunshine, and at the same time is sufficiently large and airy. The patient should rest comfortably in bed and all circumscribed, constant pressure must be avoided. As flat a position as possible, with a slightly raised position for the head, distributes the pressure over a large surface and thereby prevents circumscribed pressure points. The bed may be either an air, or water bed; this should only be one-half filled or at most two-thirds, so as to render it more pliable; the bed sheets must be dry and without folds. Patients with incontinence should be placed upon some impermeable material. The pressure of the bed clothes upon the dorsum of the foot, and the tendency to the formation of pes equinus is prevented by a loop arrangement over the feet upon which the bed clothes rest; a cushion filled with chaff may be placed at the foot of the bed to support the feet. A change of position in bed or even removal to another bed is a pleasant change for the patient whose mood is liable to be exceedingly depressed by being compelled to assume the dorsal decubitus for a long time.

When cystitis has appeared it is of advantage *to raise the upper end of the bed.* The inclined position favors the off-flow of urine and diminishes the danger of an ascent of the cystitis to the ureters. For this purpose some of the special invalid's beds may be used or the head of the bed may be raised about 15 cm. by wooden blocks. The patient soon accustoms himself to the change in position.

The endangered areas of the body are placed upon air pillows (the center being hollow so that there is no pressure) and dusted with a zinc powder. Placing the patient upon elastic felt, in which the pressure points are cut out, has been advised. By means of gentle massage and the inunction of alcohol,

wine of camphor, or lemon juice, an attempt may be made to improve the circulation and the tonus of the skin; painting with ichthyol-collodium (five per cent.) prevents infection of the skin. After the voidance of urine and feces special cleanliness is necessary. Washing with an antiseptic soap is advisable. The bed-sore may be best avoided by systematic sitz- or full baths with subsequent sponging of the endangered areas with alcohol.

Much more difficult than the prevention is the *treatment of a bed-sore that has already formed.* It should not have plasters applied as the secretions are retained by this means. Among local applications hydrogen dioxid has proven most useful in our hands. The demarcation of the ulcer and the desquamation of the necrotic eschar proceed rapidly upon the daily use of this agent. When the borders of the ulcer are inflamed a moist dressing of aluminum acetate or 70 per cent. alcohol should be used, otherwise a dry dressing of xeroform or iodoform gauze; when there is much secretion, bismuth gauze should be employed. Undermined margins, as well as fistulæ, are to be divided early to prevent the retention of secretions. Gangrenous bed-sores are best treated with warm sitz- or full baths (35° C.) lasting several hours; in the severest cases the permanent water-bed, advised by Hebra, may be employed. Putrid ulcers are to be deodorized with iodoform and charcoal in equal parts. Indolent granulations are treated with hydrogen dioxid or with a 1 per cent. caustic salve, but the newly formed margin of epidermis, at the border of the ulcer, should be avoided. The clean granulating base of the ulcer should have an indifferent salve applied (zinc salve, or boric acid and lanolin). Lately sunlight, electricity and the X-ray have been used in the treatment of ulcer.

No less difficult is the *treatment of the cystitis.* In spite of the strictest antisepsis in catheterization, especially in women, cystitis is a very common complication of paralysis of the bladder. Proper evacuation of the bladder, catheterization with all antiseptic precautions prevents retention of urine. Bladder washings and urinary antiseptics prevent decomposition of the urine; a high position of the upper end of the bed stops the ascent of the catarrh of the bladder to the renal pelvis. Double tube catheters are employed for these washings and warm solutions at 30° C. (2 per cent. boric acid, rose red potassium permanganate solution, ½ per cent. ichthyol, 1–1,000 silver nitrate); in putrid cystitis solutions of ½–1 per cent. hydrogen dioxid; finally permanent bladder drainage with the Nélaton catheter. If the urine is alkaline we administer benzoic acid by the mouth, or salol, urotropin, helmitol; further, teas of herba herniariæ or folia uvæ ursi, or mineral waters of various kinds (Wildungen, Biliner, etc.). On account of the prolonged character of the disease we must constantly change these remedies.

When there is *incontinence of the bladder* a urinal is placed in front of the patient or a permanent catheter is introduced into the bladder. Further, in this condition massage of the bladder, by means of suitable appliances with the aid of electricity (vibration apparatus), has been employed with success. This bladder vibration is to be practised with the bladder empty; the semi-globular electrode is pressed above the symphysis or upon the perineum, toward the bladder. In women the bladder may be caused to vibrate from the anterior vaginal wall. The duration of the vibration treatment, which is practiced daily once or twice, should be about 5 minutes. In nearly all cases this method produces a favorable influence upon the incontinence. In the same manner

faradization of the bladder, that is, of the sphincter urethræ may be attempted (with a sound electrode), the patient being encouraged to contract the bladder with the entrance of the current.

The method of nursing, including the patient's position in bed, and the diet, are to be maintained during the entire acute course. Only some weeks after this stage has run its course may an attempt be made, with great caution, to place the patient in an easy chair, provided that this does not exhaust him too much. Change of position often immediately alters the mental attitude of the patient and thereby favorably influences the other functions of the body; appetite, ingestion of food, sleep, and digestion.

It is the duty of the physician to strive in elevating the psychical condition of the myelitic, especially in desperate cases (*elever le physique et le moral*).

If the question of *posture* is so extremely important in uncomplicated myelitis, its solution represents the chief indication in the treatment of *compression of the cord,* as for example in caries of the vertebra. The vertebral column, on the one hand, must be kept at rest and, on the other hand, must be relieved of weight. For this purpose, formerly circular plaster casts were employed, which were applied to the patient while in an extended position, and, in caries of one of the upper thoracic vertebra, reached from the head to the pelvis. This process kept the vertebral column quiet, but it had great disadvantages, for often pressure areas and eczema were produced and the sensation of constriction, particularly in the hot seasons, was almost unbearable. Bonnet's wire basket or Rauchfuss's suspensory apparatus, with an accurately adjusted posterior piece, is much more suitable.

We prefer *extension* and *suspension treatment* upon an inclined bed, that is, in the *extension bed.* The avoidance of pressure is brought about with Glisson's head apparatus and the oblique position of the body aids in extension. It is well to begin with mild extension (twice daily for 10 minutes) which may be increased gradually to several hours daily. By this means the vertebral column may be lengthened 2–4 cm.

After the severe symptoms of compression have been relieved, we employ *supportive* and *extension corsets* (of celluloid, leather, some strong material or sailcloth with steel braces (Hessing) or of light and flexible felt) which may be modelled upon the trunk of the patient. If the focus is situated in the cervical vertebræ, an apparatus constructed of pasteboard is sufficient; an appliance which extends the neck between the occiput and chin on the one hand, and, the clavicle and the shoulder on the other hand, keeping the parts at rest. In caries of the thoracic and lumbar vertebræ extension is obtained by a jury mast and a few steel braces, which reach from the axilla to the crest of the ilium.

We must mention that in *tuberculous spondylitis, sapo kalinus venalis transparens,* in inunction, has been in use for a long time. Two or three times weekly about 30 grams are rubbed in similar to the inunction treatment in syphilis, the patient having a bath one hour later.

In paralysis due to pressure, operations have been attempted to remove the cause—exudates, tumor, fungoid proliferations, caries of the bones. It may be stated that in general the operative treatment of pressure myelitis has not been satisfactory, particularly as the pathologic focus is usually situated

in the body of the vertebra. It is, therefore, hardly possible completely to remove the affected parts, without risking a secondary fracture of the vertebræ with compression of the cord. Only in disease of the posterior portions of the vertebral arches, or of the transverse arches does laminectomy come into question, with a possibility of covering the defect by a celluloid plate to close the cavity in the vertebral column. "Redressement" of the spondylitic deformity, described 10 years ago by Calot, has been generally abandoned, particularly in the original manner, for injuries to the cord and even fatalities have resulted from this treatment.

We must now discuss the *treatment of the irritative symptoms* which probably no myelitic escapes; these represent the most distressing features of the disease. This is especially true of the *sensory irritative* symptoms, the paresthesia, the neuralgic or continuous pains in the vertebral column, or in the nerve trunks. The anatomical cause of the pain is due to an inflammation of the membranes of the cord (intradural exudate, meningitic induration) or to an inflammatory irritation of the posterior roots (*rhizitis posterior*). With the exception of gummatous meningomyelitis, it is impossible to influence the local lesion; we must, therefore, aim to diminish the pain by decreasing irritability with the aid of local or internal remedies. In acute meningomyelitis an ice bag, rubber or metal tubes (Chapmann's tubes) are applied to the spinal column; cold water is permitted to circulate through these. They may remain *in situ* until the pain has subsided. Former treatment by means of the red hot iron has quite properly been abandoned. Milder cutaneous irritants have been substituted, which often have an anodyne effect: faradization at the point of pain, sinapisms, liniments, blisters, massage, applications of hot air, etc. Painting with tincture of iodin or dry cups along the vertebral column, as well as the administration of laxatives, often decrease the neuralgic pains of the myelitic.

Among *drugs* the various antineuralgics and sedatives are useful: aspirin, phenacetin, the salicylates, quinin, caffein, pyramidon, antipyrin (also 0.1–0.2 subcutaneously), the bromids and veronal. The sovereign remedy, however, is morphin hypodermically; but caution must be observed so that the patient does not become addicted to its use. Injections of cocain, eucain, antipyrin, stovain, Schleich's solution, and iodipin may be used in the vicinity of the painful nerve trunk. In desperate cases the painful nerve trunks are exposed and stretched or anesthetized (touched with osmic- or carbolic acid), even neurotomized.

The *motor irritative symptoms*—hypertonia, muscular contractions, spasms, and contractures—are no less difficult to manage. The best remedy to combat spasm is absolute rest and the avoidance of all reflex irritation. To avoid pressure on the part of the bed clothes, the spasmodic members have a rest placed over them, the soles of the feet are supported by a soft pillow (filled with any soft material). Protracted luke-warm baths (33° C.) or luke-warm sand baths, or enveloping the patient in hot compresses and sometimes splinting the extremity are of service.

Stabile anodal galvanization has a sedative effect ascribed to it. In this treatment a large anodal plate is placed over the vertebral column or better upon the irritative points of the muscles and nerves, and only weak currents (1–2 Ma. are used; these are gradually increased and decreased. This ano-

dyne effect, which also decreases irritation, is not to be obtained by Volta's alternatives or any other method; it may be continued for one hour. For this purpose the electrodes are attached to the parts.

With greater certainty and more rapidly than galvanization with the anode, is the action of drug sedatives, which in general decrease reflex irritability and irritation transmitted from the cerebral cortex. Among these are chiefly the bromids, chloral hydrate, and atropin; the last named also being valuable in intestinal colic and spasm of the bladder; also curare which paralyzes the terminal nerve filaments. It is best administered subcutaneously for the painful clonic contractions (1 c.c.); nitroglycerin (2–3 tablets gr. $\frac{1}{100}$ daily) which is of use in angiospastic conditions, is useful for the muscle spasms.

In desperate cases we have employed *lumbar infusions* of cocain, eucain, or stovain-adrenalin (Bier), and have obtained analgesia and flaccidity of the muscles lasting several hours. In general we must advise against repeated infusions into the cerebrospinal canal for they sometimes give rise to very disagreeable reactive phenomena (fever, chills, severe headache), and especially as the relief of pain lasts but a few hours. Besides the introduction of drugs into the spinal canal, when the cord is already in the state of irritative inflammation, appears to us to be a more serious measure than when the parts are normal.

In general the drug treatment of myelitis does not play a great rôle apart from the specific treatment in syphilitic disease of the cord. Under these circumstances we prefer the hypodermic employment of iodin and mercury, the former given in the form of iodopin (25 per cent.) 5–25 c.c. every other day. Mercury we administer as corrosive sublimate injected into the muscles or as hydrarg. succinimid., eucain, āā .01 for an injection (this is painless and may be given every second day). In this manner 20–30 injections should be given. Occasionally, however, even energetic antisyphilitic treatment is without effect in luetic spinal disease; even dural injections of sodium iodid we have found to be ineffective.

Etiologic treatment, serum therapy, has up to this time shown no direct results, even in the case of post diphtheritic myelitis. In post infectious myelitis, Oppenheim advises diaphoretic treatment combined with the administration of the salicylates.

The use of silver nitrate or strychnin has rarely been followed by good results. Strychnin is employed in paralysis of the sphincters, in anesthesia, and in flaccid paralysis. By stimulation of the reflex centers it increases the excitability of the motor nerves in the paralyzed muscles and in combination with electric and gymnastic measures it favors the restitution of the muscles. Strychnin is best employed in solution 1–1,000, best subcutaneously 1–5 mgms. daily.

Other indications, in the course of myelitis, sometimes require the employment of drugs, such as analeptics, tonics, stomachics, laxatives, etc.; these are prescribed according to well established therapeutic principles. In general the value of drugs, even in the most favorable case, is purely symptomatic; physical curative methods are quite correctly substituted for them.

The physico-mechanical treatment of myelitic paraplegia must fulfill the following objects:

Prevention or relief of muscle atrophy and contractures; the most thor-

ough restitution of motion by the compensatory education of retained muscle functions (exercise, etc.) or by apparatus, that is orthopedic-surgical measures.

Before proceeding to employ these methods one question must be answered which is of fundamental importance in the physical therapy of myelitis: In how far is there an anatomical possibility of restitution in myelitis? If the inflammation has destroyed the entire transverse section of the cord naturally there can be no question of a restitution of motion. On the other hand in partial, insular foci a certain functional rehabilitation may take place. The unaffected half of the cord may, to a certain degree, function vicariously for the diseased portion. Turner showed experimentally (*Brain*, 1891), that even with a unilateral section of the cord almost complete functional activity may occur. Kocher has reported almost complete recovery in patients suffering from unilateral lesions of the cord. In our case of Brown-Séquard's paralysis, following a stab in the 2d dorsal segment, sensation was abolished upon the contralateral side, but motion returned to the extent that the patient was enabled to walk without aid, almost like a normal person.

Regeneration does not take place at the point of lesion, on the contrary, ascending and descending degeneration here occurs, but in the surrounding of the pathologic focus the compensatory function of the retained tracts of conduction substitute for those that have been destroyed. If the main tract of motion is destroyed, the pyramidal lateral column tract, upon one side, the contralateral pyramidal tract and also the extra-pyramidal motor tracts (v. Monakow's rubrospinal tract, the corpora quadrigemina anterior column tract) may act alone, and by suitable exercise may become substitution tracts (compare also P. Lazarus, Die Bahnungstherapie der Hemiplegie, *Zeitschr. f. klin. Med.*, 45. Band, 1902).

Less clear is the special localization of the sensory spinal cord tracts; some qualities of sensation, such as deep sensation, are innervated from both halves of the cord, so that the normal side of the cord may act for the diseased portion to a certain extent. The bladder and rectal tracts are also to be regarded as bilateral, so that incontinence of the sphincters indicates, as a rule, a bilateral disease of the spinal cord. In a unilateral affection, as in Brown-Séquard's paralysis, sphincter disturbance, as a rule, is lacking. Further the symptoms at the onset of myelitis point to an extensive lesion, for in the surrounding of the pathologic focus, similar to the condition in inflammation of the brain or apoplexy, reactive disturbances appear (alterations in the circulation, edema, small extravasations of blood). Only with retrogression or absorption of these secondary phenomena is the loss of function limited to its anatomical minimum. The compressed ganglion cells and nerve fibers then have an opportunity to recuperate. This is especially true of pressure paralysis and of multiple sclerosis, in which the medullary sheath is preeminently involved, while the axis cylinder is retained for a long time. The problem of physical therapy consists in educating the nerve tracts which remain to perform compensatory functions by means of methodic exercise.

The first object of physical therapy in myelitic paralysis is a prophylactic one and consists in the prevention of muscular atrophy and of loose joints; of no less importance is the avoidance of spastic and paralytic contractures. It is very necessary to meet this indication because contracture deprives a paretic muscle of all possibility of motion, while if the tonus be at all normal

it may still be capable of function. A number of measures serve this purpose which should be utilized as early as possible.

We begin with a passive change in position of the paralyzed members. According to a definite formula of motion, the paralyzed extremities are placed in different positions several times daily, no joint, no muscle group being neglected. Primarily those positions are preferred which antagonize the tendency to contracture. Thus the ankle joint is flexed dorsally and pronated; the foot, in consequence of its weight and on account of pressure of the bed clothes, shows a tendency to plantar flexion and in consequence to pes equinus; this is prevented by using a pillow which will maintain the position of dorsal flexion and by a loop appliance to prevent pressure of the bed clothes.

These passive movements are gently and gradually extended to the normal limit. Rough handling only increases the contracture or produces it. Passive exercises are practised in one joint or in several at the same time, for example, flexion in the hip, knee, and ankle joints or abduction of both limbs. The joints may be held in position temporarily by sand bags, bandages or braces. We endeavor to prevent the very frequent adduction contracture of the legs by placing a pillow, containing some soft material or air, between the knees, or placing the patient for from 1–2 hours upon "straddle-board" (Mikulicz, Hoffa).

Relaxation exercises may also be employed to prevent contractures. For this purpose the patient is encouraged to let the hypertonic members hang flaccidly and as limp as possible. By this means many patients succeed in relieving the tendency to contracture at least temporarily. (Muscular relaxation, according to experiments in apes performed by E. Hering and Sherrington, is due to a feeble stimulation of the cortex.) These atomic gymnastic exercises are even more effective in a warm, prolonged bath (general or local). Other forms of application of heat, packing the rigid limbs in hot cloths, surrounding them with rubber tubes in which hot water is caused to circulate, the application of hot bottles or thermophores, compresses, and finally hot air baths. The use of heat also improves the cutaneous circulation; the cool cyanotic extremities becoming warm and red. Warm sand baths (caution must be exercised so as not to produce burns) have an influence in producing relaxation.

In addition to the "antagonistic change of position," the "exercises in relaxation" and the application of heat, massage, and electricity may be employed; those muscle groups should be especially treated which act against the tendency to contracture. Vibratory and stroking massage is used for the muscles and joints and for the nerve trunks, in the form of friction and vibration. Percussion massage of the contracted tendons, movement of the joints which are in positions due to contracture, have a transitory effect in relaxing the muscles and improving motion.

These methods of treatment are also opposed to the development of *inactivity atrophy* of the muscles. The muscle fibers whose trophic anterior horn centers are completely destroyed cannot be protected from atrophy. In this case and also in paraplegia after complete transverse myelitis, the baths, massage, change of posture and the passive movements have the purpose of stimulating the circulation in the paralyzed members, of preventing venous and lymph stasis, of opposing the development of inactivity atrophy and of

vasomotor and trophic disturbances and thus improve the entire metabolism of the organism. In this sense we employ massage and gymnastic exercises in our paraplegics twice daily (morning and evening).

The *treatment of contracture*, that has already developed, is much more difficult. We endeavor to combat it by extension and redressment, particularly with the aid of hot air or the warm water bath, or by fixation by means of steel braces in a position of over-correction. For this purpose we employ, in contracture of the knee joint, screw extension or our own redressment apparatus, in which the principle of extension is combined with direct redressment of the joint (compare P. Lazarus, *Charité-Annalen*, 27. Jahrg., 5. Jahresbericht, Ueber die Anwendung der physikalischen Heilmethoden in der I. medic. Klink und Poliklinik). Passive pendular or swinging exercises, by manual aid or special apparatus (pendular apparatus of Krukenberg, Zander) are of value. In advanced contracture, where the joints in consequence of atrophy of the contracted tendons and fascia, are very difficult to move, surgical measures or apparatus come into consideration in which elastic opposition maintains the equilbrium against the contracted muscles. Old, rigid, especially painful, contractures can be relieved only by cutting the tendons.

In connection with the treatment of contracture and atrophy is "*exercise*" therapy. This consists in the use of *passive, autopassive* and *active methodic gymnastics.*

Passive motion, either by the hand of the physician, or by the electric current, or by any apparatus, conducts sensation from the altered tension of the nerve terminations in the muscles, tendons, fascia and joints, from the parts set in motion to their cortical centers. The central convolutions, according to the celebrated experiments of Hitzig and Fritsch, as well as those of H. Munk, are not only the motor, but also the sensory centers for the extremities, so that a sensory stimulation of the muscle sense produces a simultaneous stimulation of the motor tract. Passive motion, therefore, stimulates motor innervation, for an irritation of the muscle and joint sense stimulates the center of the affected extremity from which then the motor impulse may descend to the periphery.

In an interruption of the pyramidal tract, either in the internal capsule, or in the spinal cord, the motor impulses cease above the lesion. In spite of all endeavor on the part of the patient there is no motion or at most the merest trace; only after repeated attempts, according to the law of summation of stimulation, is there a distinct, although incoördinate movement. This fact, that the paralyzed patient is enabled to exert his will, even though in an incoördinate manner, proves, that a new communication has taken place between the sensory and motor tracts at a point outside the lesion.

Our object now consists in cultivating this tract by systematic exercise. The repetition of the impulse of the will prepares the way for movement at the command of the will. By this we are to understand those movements of the paralyzed members which the patient is able to carry out himself with the aid of the normal members. The active muscles guide the paralyzed ones; thus the legs, with the aid of the arms directly or by means of apparatus, are set into motion; in the same manner the leg that has more movement may be fixed to the paralyzed one, so that the latter is caused to partake of the same action. By these simple "auto-exercises" the path is prepared for the

motor impulse from the cortex of the brain to the muscle; the patient, whose voluntary motion has suffered from the prolonged inactivity, once more gains the sensation and conception of motion; he learns to reinnervate, until, finally, he is enabled to perform simple, active movements.

In this stage the patient must practice walking movements, first in the recumbent posture; similar to the method that we employ in the physical treatment of hemiplegic contracture (see P. Lazarus, *Zeitschr. f. diät. u. phys. Ther.*, 1901–1902, B. V, Heft 7). The patient at the word of command is ordered to raise and lower the leg, or to extend it in 4 quarter time; at the knee joint to raise it, bend it, extend it and again lower the leg. The ankle joint is also exercised actively in stepping movements. These should first be practiced with the heel upon a base of support, such as sliding movements followed by exercises with the leg raised. In this manner the movements of walking are practiced in bed. In case the patient is able to sit up he is put either at the edge of the bed or upon a high chair and encouraged to perform extension and flexion of the knee and ankle joints with the legs hanging free. These exercises must always be of brief duration, but may be repeated frequently during the day. If contraction or fatigue are noted, a pause should be made. It is serviceable to note in writing the nature and manner of the different movements, especially in regard to the active movement of the individual joint. Patient and physician gain a better insight by this means of the technic and progress of the treatment.

We advance gradually with our exercises and permit the patient to attempt methodic standing and walking exercises in the erect posture. For this purpose we employ crutches or walking frames. Our latest model of a walking frame consists of a horse-shoe shaped iron frame; this is propelled upon 3 wheels. The closed portion of the loop behind has a convenient chair attached. The front, free ends of the frame, have movable arm and axillary supports to which a scale is attached. This construction has the following advantages: the patient is able to walk unencumbered, without constriction about the chest or abdomen, his movements become more independent, especially as he need not depend upon a second person to control the chair as in most apparatus of this kind. The paraplegic may also practice walking in this apparatus while in the sitting posture; the greater exertion of the erect posture is lacking, the legs are relieved of the weight of the body and the movements of walking are readily instituted.

For walking we have had an exercise-shoe constructed which in shape resembles a skee. At the anterior and posterior end of a thick oak wood block, 80 cm. long 4 cm. broad and about 0.8 cm. thick, there are sandals in which the feet are strapped. Those in front are used by the patient, who is manually supported or placed in the walking frame, those behind are occupied by the physician. The latter is enabled, by the movements of his own leg, to produce the corresponding motion in the paralyzed lower extremity of the patient and thus to regulate the step-movement or to improve it. Finally the physician may institute resistance gymnastics, permitting the patient to use active motion with the leg, while the physician opposes it.

We gradually pass on to *free exercises*; the attendant steps behind the patient, holding him either by a girdle or by the clothing or passing his arms under the axillæ of the paraplegic. Both step out simultaneously the physi-

cian with the tip of his toes directing the movements of the patient or having some one else place the patient's feet in the correct position; or the patient is encouraged to walk in foot marks that are drawn upon the floor. In this stage walking exercises in the parallel bars or along banisters, passive and active exercise in swinging apparatus and the like are also useful. Later by locking arms with another person or holding hands; or walking with 2 sticks then with one stick, and finally entirely alone locomotion is reestablished. A result of this kind will naturally only be obtained in favorable cases; in the severe ones we cannot attain our purpose without orthopedic or surgical measures. The mechanical treatment of paralysis is greatly facilitated by *wave-baths* and *electrotherapy.*

In the warm bath muscular rigidity and even recent contractures are relaxed. The first traces of returning motion are often then observed. Leyden and Goldscheider have called attention to the great value of "kinetotherapeutic baths" in the treatment of paralysis. In the bath the weight of the member that is to be moved is compensated for by the buoyancy of the water. The specific gravity of the water may be increased by the addition of salt or brine. Maintaining the paralyzed members in equilibrium, combined with the relaxation of the hypertonia, prepares the road for active motion. This is particularly of importance in those patients who must learn to overcome the actual weight of their paralyzed parts in the water-bath. The impulse of motion, in these patients, receives a mighty stimulus by the returning power of motion obtained in the bath. The apparently dormant motor impulses awaken in the bath and after a long period of inactivity again reach the muscles. If the tract has once been established, between the impulse to motion and the muscles, our further object consists in cultivating this tract by methodic exercises until the patient is capable of sending definite impulses of motion precisely into those muscle groups to which they belong.

For this purpose all muscles and joints of the paralyzed members are moved actively and passively in the kinetotherapeutic bath. Standing and walking exercises, in high bath tubs, are especially useful (Goldscheider's standing bath). The same author praises, quite properly, the favorable psychical influence (*plaisir de mouvement*) which these movements in the bath exert upon the motor impulses (see Goldscheider und P. Jacob, Handbuch der physikalischen Therapie, 1902).

We must now turn to the electric treatment in myelitic paralysis. The curative effect of the electric current was formerly greatly exaggerated. There was an idea that the electric current, employed therapeutically, was able to penetrate to the spinal cord substance and there unfold its curative action. On the other hand we may assume that electric irritation, in consequence of the artificially stimulating muscular contractions, excites the dormant sensation of motion in the paralytic; to this may be added the passive motion which is produced by the electricity; this stimulates regeneration of the paralyzed muscles and nerves in causing them to assume activity. The sensory stimulation produced by the electric current in the periphery has an inhibitive action upon the cortical motor impulse. This has been experimentally proven by Exner, Bubnow and Haidenhein, who showed that it requires a weaker electric irritation of the cerebral cortex, when the member, which is supplied by it, has previously been treated by electricity at the periphery.

Spastic paralyses should have sedative treatment; active electrical contractions should be avoided. For this purpose we apply a weak constant current which is permitted to act upon the motor nerves, motor points, and muscles. The strength of the current is gradually increased to from 2–5 Ma. and this is again gradually decreased. The anode is placed either stabile upon the motor point or is run slowly 20–30 times over the muscle, longitudinally and transversely. When marked reflex irritability is present and also in the acute stage, electric treatment is not employed.

Flaccid paralysis and *atrophy* are treated by stimulation either with the galvanic, faradic, or the combined, currents. When there is DeR we only employ galvanism; the anode, as the stimulating electrode, in case, as is usual, it is the more active. Strong contractions may be produced by Volta's alternatives. To increase the stimulation, especially in the deeper muscular areas, the combined galvano-faradic current is applied. The combination of both currents, according to de Watteville, is said to unite the stimulating influence of the intermittent faradic current with the "interstitial electrolysis" of the uniform galvanic current.

The faradic battery is most often employed in flaccid paralysis, in muscular atrophy, in paresthesia, and in anesthesia. It is serviceable to have the muscular contraction produced by faradism occur simultaneously with the intent of the patient; in this manner the motor impulse to the muscle is reinforced. In the same way, by increasing or decreasing the strength of the current, we are enabled to aid the motor impulse more or less. By gradually decreasing the electric current we may burden the will more and more, until, finally, the patient is able to produce the movement without our aid.

Cutaneous anesthesia is treated with the faradic brush or with franklinization (by drawing sparks). Stabile or labile cathodal galvanization (2–6 Ma. for 5 minutes) is said to be effective in anesthesia and paresthesia. Some other mechanical and thermal cutaneous stimulants (massage, carbonic acid or oxygen baths) further, contrast irritation (alternating applications of heat and cold) restore sensibility to the skin.

In the management of *spinal irritation* and the *girdle pains*, the current may be sent through the thorax (cathode upon the vertebral column and anode upon the sternum) or in a longitudinal direction (anode above the supposed focus, cathode below or labile upon the peripheral nerves).

For some years we have combined *electricity with bath treatment.* In the warm bath (full or local) the motor impulse is aided by the decrease of muscular rigidity especially if this be combined with electric stimulation in the manner described above.

Recently paralysis and anesthesia have been treated with the electric 4-cell bath according to Dr. C. Schnee. By means of the 4-cell bath electricity may be employed in many different forms—galvanism, faradism or combined, as a pulsating direct current or as a sinusoidal alternating current; finally also for cataphoresis. By means of apparatus the current may be sent through the body in any desirable strength or direction.

To attain deep action, as in incontinence of the bladder, we employ the combination of faradization with electric vibration-massage. For this purpose the negative pole of the opening induction current is united with the wave of the vibration apparatus, which has a metal electrode at its end covered with

flannel. This forms the common stimulation electrode of the faradic and vibration apparatus. By the wave-like propagated vibration the faradic current penetrates deeply and this increases the stimulating action of even weak faradic currents.[1]

If these methods of treatment bring about the coarse power of motion then a further object arises: the improvement of the coördination. For this purpose complicated movements are divided into their essential components and these are practised just as untiringly as the tactile exercises of the blind, until they are performed satisfactorily. In these coördination exercises, which are often very difficult, the delicacy of the sensation of motion is most important; often the paraplegic loses his motor experiences, his memory of motion, from prolonged inactivity. By methodic exercise, passive or active, with closed eyes, the movements being carefully adjusted, just as in ataxia, we occasionally succeed in improving the deeper disturbances of the muscle and joint senses.

The same principles are also operative in paralysis of the upper extremities; but here the hand requires especial consideration. All muscles of the forearm, also the interossei and lumbricales, as well as all of the muscles of the thumb, should be subjected to careful electric and mechanical treatment. The treatment of the nerve is particularly important in those cases in which the same nerve supplies muscles that act synergistically.

Spa treatment can only be considered after the acute stage has passed, the patient no longer has fever and there is no danger in transportation. In general it is not good practice to send a myelitic away for mineral spring treatment sooner than 6 months after the inflammatory symptoms have passed away. Extreme temperatures, hot as well as cold, and particularly steam baths are to be avoided.

On the other hand, moderate thermal baths, as in all other hydrotherapeutic procedures, at a temperature of about 37° C., are useful. They have a sedative effect upon the nerves of the skin and therefore reflexly upon the nerve centers; they decrease muscular rigidity and increase metabolism as well as the cutaneous and general circulation. The duration of the bath, at the onset, should be about 30 minutes, but in spastic paralysis, in hyperesthesia and hyperkinesia the time may be extended to several hours. This prolonged bath often has a very quieting effect.

Among the indifferent thermal baths are Ragaz-Pfäffers in Switzerland (35° C.), Badenweiler (26° C.), Römerbad (37½° C.), Schlangenbad (18–32½° C.), Wildbad (26½°–32° C.), etc.

The thermal, salt and chalybeate springs rich in gas are of service, such as Wiesbaden, Baden-Baden, Oeynhausen-Rehme, Nauheim and Münster. The carbonated thermal baths have a sedative action in diseases of the spinal cord and at the same time stimulate motility and sensibility. The favorable action of carbonic acid baths has gained for them many adherents among the laity, a proof of this being the many apparatus and preparations for the artificial development of carbonic acid baths. In our Clinic we employ the v. Orth system. With this system the compressed carbonic acid gas (or

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[1] In regard to the diagnostic and therapeutic indications of immediate electrization of the spinal cord and the cauda, see P. Lazarus, "Ueber die spinale Localisation der motorischen Functionen." *Zeitschr. f. klin. Med.*, Bd. LVII, p. 99 (1905).

oxygen) is led from the steel cylinder into porous tubes which enter the bath tub and from which the gas emerges in small bubbles. The amount of gas may be accurately controlled by a manometer and stopped at any time. This system is characterized by simplicity, accuracy, lack of destruction of the bath tub, and by the low cost; it being possible to prepare a carbonic acid bath at a cost of about 2½ cents. The duration of the bath should be 10 minutes, at most 20 minutes, the temperature 36° C.–31° C. The mechanical effect may be increased by undulations or shower baths. In paralysis combined with anemia, iron-peat baths, such as Franzensbad, Marienbad, Elster, Cudova, are valuable. Peat baths stimulate the skin and have a great absorbent effect. The hot sulphur springs are very useful in syphilitic myelitis (Aix la Chapelle, Baden near Vienna, Pistyan). When arthropathies are present, peat, mud and fango packs may be employed.

The *physico-mechanical methods* of treatment (*massage, gymnastic exercises, electricity, hydrotherapy* and *balneotherapy*) which have been described are not sufficient when rigid contractures or grave, flaccid, atrophic paralyses have appeared. Under these circumstances apparatus and surgical measures are necessary.

The *treatment by apparatus* has for its purpose the relief of the contracture, the substitution of function for the paralyzed muscles, and the strengthening of the flaccid joints. The contractures are managed by redression apparatus, best adjusted after a previous tenotomy, or by a plastic operation of the contracted tendon. We attempt to supply the function of the paralyzed muscles by elastic bandages, which, in their action, imitate the physiologic mechanism of movement and by their tension maintain in equilibrium the power of the antagonists. Thus in paralysis of the quadriceps or of the peronei muscles an apparatus may be applied which has a hinge for the knee or ankle joints, and elastic bands corresponding to the insufficient muscle groups; this apparatus, to a certain extent, converting the displacement of the muscle power to the outside of the leg.

Apparatus may also be applied to support loose or deformed joints. By this means we have often succeeded in again putting a paraplegic "upon his legs." We shall report a case of myelomeningitis lumbalis in a man aged 31.

This patient came to us with atrophy and flaccid paralysis of the muscles of both legs; those of the hip joint being markedly paretic, of the knee and ankle joints being immotile to active impulses. The left leg, nerve and muscles, could not be stimulated by galvanism or faradism; the right leg hardly reacted to faradism and only responded to maximal galvanic currents and then only the quadriceps, crural, and peroneal muscles. The muscles of the right calf did not react to the strongest currents. Upon the posterior surface of both legs as well as upon the dorsum of the feet there was anesthesia and analgesia; the scrotum was also anesthetic. Cutaneous and tendon reflexes were abolished in both legs. It was, therefore, a case of transverse disease reaching to about the second lumbar segment.

The treatment consisted in hydrotherapy and electro-gymnastics, sand and water baths, bloodless nerve stretching, this having an especially favorable effect upon the severe sciatica; further, injections of iodopin and corrosive sublimate. The result consisted in increased motion of both hip joints and of the right knee joint. The galvanic contractility of the muscles of the right thigh returned. The patient, however, was unable to walk on account of the residual, complete, flaccid-atrophic paralysis of both ankle and knee joints. Only by means of the apparatus here illustrated and with the aid of 2 canes could the patient walk with some degree of certainty; he was able to resume his occupation, and can now descend from a carriage and ascend stairs.

If the paresis involves the hip joints, the patient may be aided by an apparatus consisting of a corset to which 2 splints are attached for the legs. Such an apparatus is shown by Fig. 172. This illustration was taken from a patient aged 50, suffering from high grade ataxia of the arms, legs, and trunk, combined with pareses, muscular atrophy and arthropathies in the ankle, knee and shoulder joints. Even with support the patient could neither stand nor walk, and upon attempts to assume the erect posture she collapsed at the hip and knee joints. In consequence of ataxia of the arms she could not even hold a cane. The supportive apparatus consisted of a corset with steel braces, with supports to the hips and axillæ; this was united by means of rubber to a brace for the legs. Abduction and flexion of the hip joints was somewhat hindered but not enough to allow forward movements. The illustrations, Fig. 172, a, b, c, show in detail the structure of the apparatus; this permitted the patient to stand unsupported and even to walk with slight assistance.

Fig. 172.

In complete paralysis, when the antagonists and the agonists are both involved, another principle may be applied, namely, the transmission of the normal muscle power to those involved. Thus the shoulder or the arm may be connected with the paralyzed leg by inextensible bands, so that the leg is moved by the arm.

But even the best apparatus is [illegible], especially if it must be worn day and night; [illegible] these must be com- [illegible] the mechani- [illegible] hope of success [illegible] hyper-

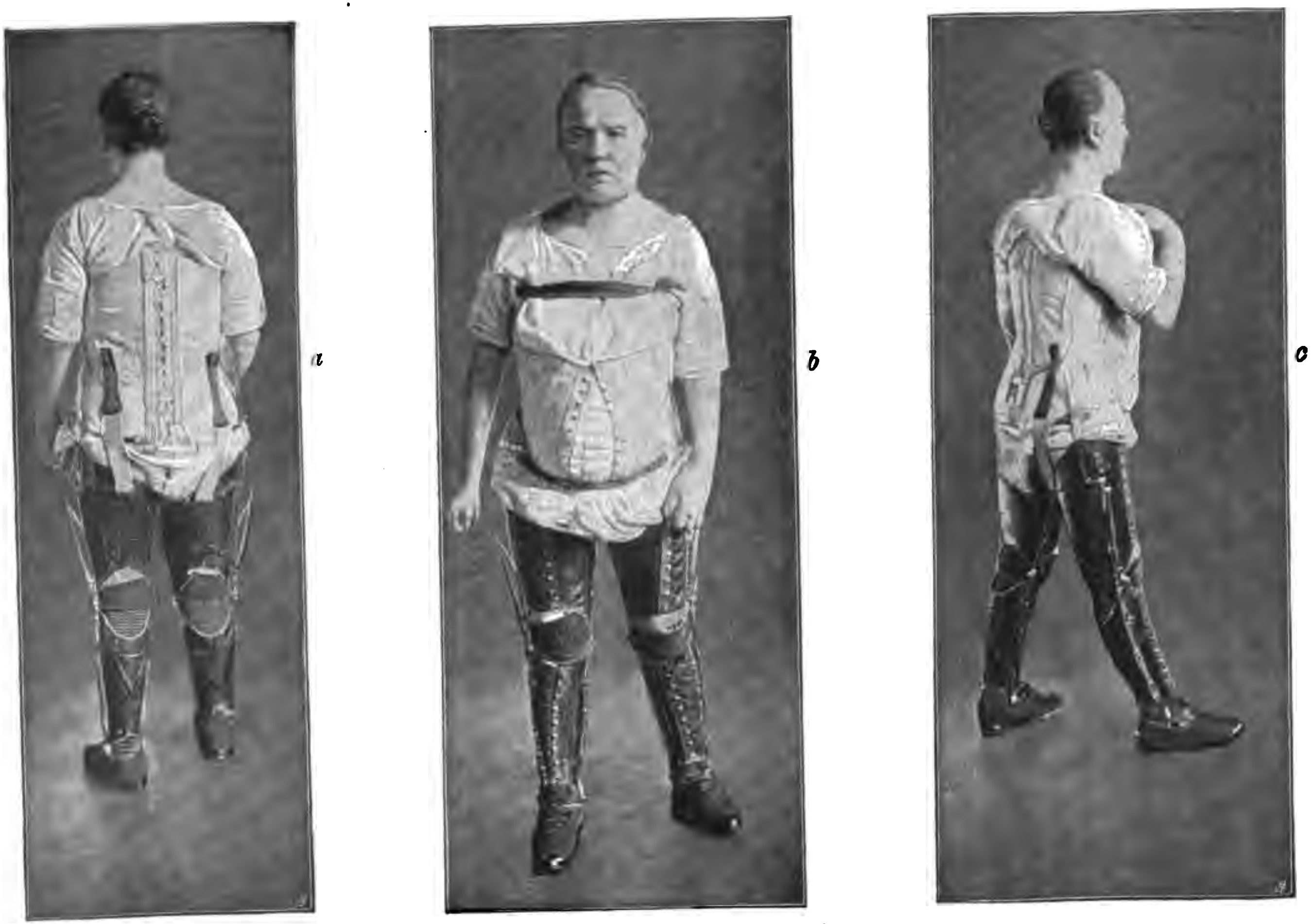


FIG. 172.—APPARATUS FOR THE TRUNK AND LEGS.

distension of the flaccid muscle, as the peronei or the dorsal flexors of the foot in paralytic pes equinus; further in loose joints, arthrodesis either by uniting the tendon to the capsule of the joint or by freshening and suture of the arthritic surfaces. Arthrodesis should be performed only when all other methods of treatment have failed and when there is paralysis of all peri-articular muscles.

In regard to the operative treatment of the spondylitic gibbus, see Hoffa "Die moderne Behandlung der Spondylitis, 1900."

Transplantation of tendons, proposed and performed by Nicoladoni in 1881, has assumed great importance; this is most successful in dissociated muscular paralyses. In this condition only one muscle group is affected, while the antagonists are preserved and are usually strongly contracted. In these paralyses only surgical and orthopedic measures present hopes of success, and either redression apparatus or operative transplantation of the functionating muscles to the insufficient ones. The latter is accomplished by tendon transplantation, by which the normal muscle is implanted upon the affected one; thus in paralysis of the dorsal flexors of the foot and of the peronei, the Achilles tendon may be divided in 3 parts and 2 of these may be attached to the muscles that do not function. Prior to the operation we must determine by careful and accurate functional and electrical tests that the muscle to be used is actually normal. During the operation we may form an opinion of the condition of the muscle by electric stimulation and by examination; normal muscles have a fresh, red appearance, those presenting fatty degeneration are of a yellowish color and are flaccid.

With the transplantation of the normal tendon into the affected one, compensation is as yet not brought about; in the cortex of the brain a change must also take place. When, as was done successfully by F. Krause in a case of infantile paralysis, the quadriceps femoris is replaced by the normal flexors of the knee, this alone does not transmit the function. The patient must first learn to extend the knee with the center for the flexors of the knee, he must learn to innervate in a diametrically opposed manner, and this is one of the objects of our treatment. This function is usually attained rapidly and the patient, especially if he be young, soon learns to contract the transplanted muscle portion. According to these principles, in the last few years, numerous operations of this kind have been performed with marked excellent functional success.

Many patients owe their power of earning their living and their freedom from the rolling chair, to these physical-orthopedic methods. The common labors of the internist and the surgeon have had a beneficent effect in the "border land" of myelitic paralysis and many paraplegics have been saved from the almshouse and home for cripples. The prognosis of myelitis has improved vastly owing to the progress of physico-orthopedic therapy.

Even the apparently hopeless cases of myelitic paralysis should be treated by the physician, free from nihilistic scepticism and just as free from uncritical polypragmaty. Full of hope, he should prepare a plan of treatment which will meet all of the indications in a harmonious manner and this should contain all the well tried pharmacologic and physical methods of cure sanctioned by our science and our experience.

EXPLANATION OF THE COLORED PLATE

FIG. 1.—Influenza myelitis. Hemorrhagic focus of softening (4); extensive hemorrhagic infiltrations (2, 3, 5, 6), ascending degeneration of Goll's columns (1).

FIG. 2.—Acute myelitis in the puerperium. Spinal cord hemorrhage with secondary myelitis (ascending and descending degeneration).

FIG. 3. Hematomyelia in the puerperium. Description in the text.

PLATE I

Fig. 1.—Influenza Myelitis.

Fig. 2.—Acute Myelitis in the Puerperium.

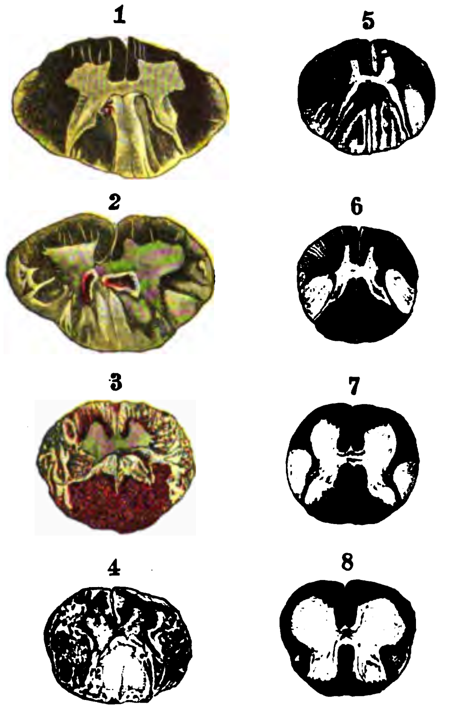
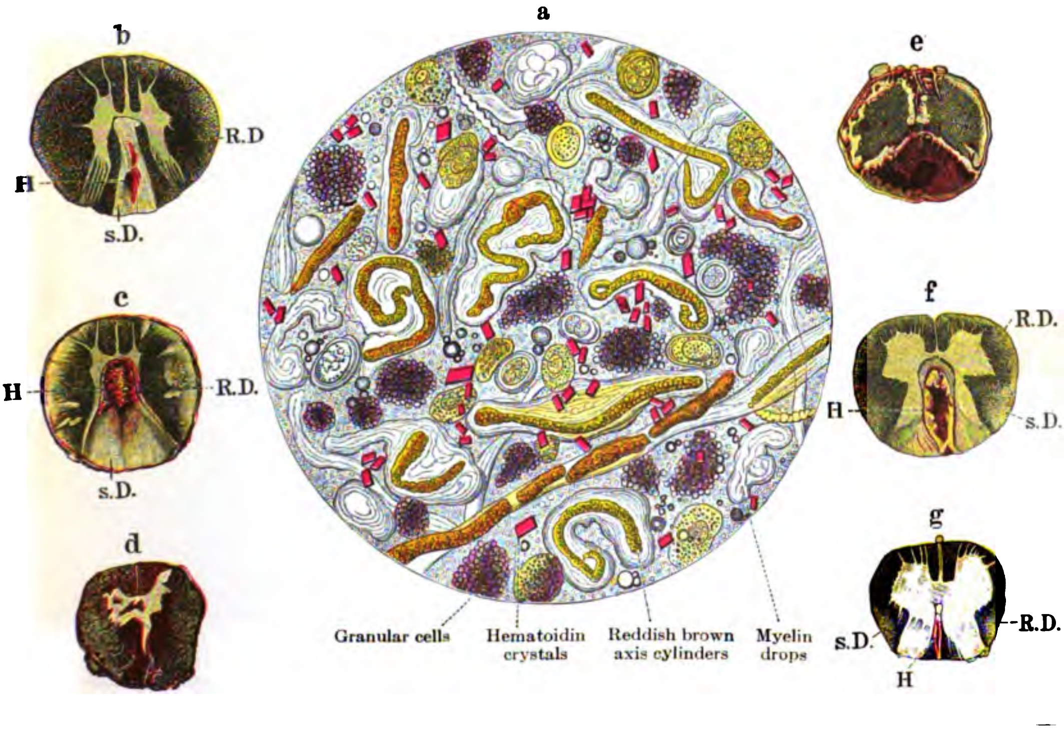


Fig. 3.—Hematomyelia in the Puerperium.



TABES DORSALIS

(GRAY DEGENERATION OF THE POSTERIOR COLUMNS OF THE SPINAL CORD; ATAXIE LOCOMOTRICE PROGRESSIVE; LOCOMOTOR ATAXIA)

By WILHELM ERB, Heidelberg

I. INTRODUCTION AND HISTORY

In the points of frequency and importance, tabes [1] is unequalled by any other chronic disease of the spinal cord. It is the most widely distributed, the most thoroughly studied, the richest in symptoms, and in the number of scientific problems it presents, and is unexcelled by any in the number of opinions to which it constantly gives rise.

Clinically as well as anatomically it is sharply characterized, and is one of the forms of spinal disease which have a most sure foundation. **Clinically** —in spite of the unending variety of symptoms which it produces—it is marked by phenomena which recur with the greatest regularity, and which form the true foundation of the pathologic picture, around which at times many other symptoms crystallize. In the *first stage of the disease* we chiefly note disturbances of the sensory sphere (pain, paresthesia, hyperesthesia, anesthesia of typical form and localization, and sensations of fatigue) combined with disturbances of certain cranial nerves (the optic, the nerves of the muscles of the eye) and of the bladder and sexual apparatus; in the *second stage*, by the addition of a new and very characteristic motor disturbance, tabetic ataxia and disturbance of coördination are produced, and dominate the clinical picture. **Anatomically**, it is mainly marked by a gray degeneration of the spinal posterior columns and the posterior roots, as well as by other invariable or accidental lesions.

A comprehensive knowledge of this remarkable and many-sided disease which affects so severely the life of the individual and society in general is one of the achievements of the nineteenth century. In the writings of the ancients from the time of Hippocrates all search for a description of tabes had been fruitless; it is hardly conceivable that the typical peculiarities of the disease could have escaped the attention of the great investigators in all former ages, or that its description in their works should not be recognizable. Tabes does not appear to have existed in antiquity, nor for many centuries after. Since we know to-day that the most essential, the almost exclusive, requisite

[1] We agree with Moebius that the term "tabes" now everywhere accepted is a suitable designation for the affection under consideration.

for the development of tabes is syphilis, this appears reasonable, for the most recent investigations indicate with increasing positiveness that syphilis was transported to Europe only after the discovery of America and toward the end of the fifteenth century.

It is not our purpose here to enter upon a discussion of the history of tabes. It is sufficient to state that these views concerning it date from about the middle of the preceding century; there were reports of this affection by Horn (1827), Stanley (1840), Steinthal (1844), and others, but it was the comprehensive descriptions of Todd (1847), of Romberg (1851), and of Duchenne (1858–9) which acquainted the professional world with the disease; the classical description of Romberg and the masterly clinical presentation of Duchenne, the latter of whom first characterized it as "ataxie locomotrice progressive," have established the clinical picture and have inspired numberless investigations and much labor concerning the apparently "new" disease. The period from 1860 to 1870 witnessed a flood of important and comprehensive clinical and pathologico-anatomical investigations, especially in France (Trousseau, Charcot, Vulpian, Dujardin-Beaumetz, Mar. Carré, Topinard, Jaccoud, and many others) and less so in England; in Germany, however, in the year 1863 three reports appeared by Eisenmann, Leyden, and Friedreich, and these formed a landmark in the study of tabes, even in a pathologico-anatomic respect. Many other reports by German physicians followed, clinical and anatomical investigations increased in number, until it appeared from the explicit presentations of the subject published about 1865 by Leyden and Erb as if our knowledge were complete.

But not so! The subject proved to be inexhaustible, and in all civilized countries the investigation was continued until a true high water mark of reports upon tabes seemed to be reached, thousands being poured out, in an endless, overwhelming stream! [1]

This led to a comprehensive consideration and specialization of the *symptomatology*: The recognition of reflex pupillary rigidity, the absence of the tendon reflexes, the hypotonia of the muscles, the special form, localization, and topography of the sensory disturbances connected with the old Romberg's symptom, the determination of an enormous number of so-called visceral symptoms (gastric and intestinal crises, laryngeal, renal, bladder, genital, and cardiac crises, etc.), the very important trophic disturbances (tabetic arthropathy, loss of teeth, atrophy of bones, etc.), the description of nuclear and bulbar cranial nerve disturbances, of peripheral, neuritic, muscular atrophies, of paralysis of the recurrent laryngeal nerve, and many other conditions filled literature, and revealed a most unexpected richness of individual symptoms.

The improved methods of our time have given us comprehensive *pathologico-anatomical* reports which furnished new and varying conceptions, and led to most surprising conclusions; unfortunately, much in this realm still remains obscure, and contradictory views have not yet been reconciled.

In the study of the *etiology* of tabes the conditions have been different. A debate which has continued to the present time was started about 1870 when Fournier and Vulpian maintained that the great majority of tabetics had a syphilitic history, that tabes, therefore, was to be regarded as a "para-

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[1] See the report by Möbius, 1–14.

syphilitic," or according to other authors, a meta-syphilitic affection due to syphilis. I investigated these reports, at once confirmed them, and took my stand on the side of the French authors; Gowers, Strümpell, Möbius, and others followed, but there was opposition on the part of nearly all the other German authors. Thus a lively partisanship arose. Full statistics and casuistry were compiled; reasons and opposing reasons were advanced, pathological anatomy and treatment were invoked to decide the issue. The result of the dispute is as follows: The assumption of a connection between tabes and preceding syphilis is sustained, although the exact relations and nature of this connection are still in doubt.

Nearly all of the prominent neurologists and internists, almost all of the recent text-books, recognize this connection, not only in Germany but elsewhere—in France, England, America, Russia, and Italy. Only a few eminent authorities (v. Leyden, Goldscheider, Moczutkowski, and others) are opposed to this and deny the relation. It is to be hoped that the overwhelming mass of statistical, clinical, and anatomical facts in proof thereof will overcome this opposition.

Much time has been consumed in discussing *general pathologic questions*, questions of the *physiology* and *pathology* of the spinal cord which naturally arise in the consideration of tabes: The position of the sensory tracts, the conduction of various sensory impressions in the spinal cord, the various reflexes, the location of their tracts, the innervation of the pupils, above all, the typical disturbance of coördination; briefly, the ataxia and much besides without coming to any conclusion.

The actual *nature and origin of the pathologico-anatomical changes*, their intimate relation to syphilis, the pathogenesis of the individual disturbances, their exact cause and peculiar localization, etc., have, of course, been much debated.

Finally, in the last few years we have undertaken the finer *investigation of the various forms* of the affection, we have learned to recognize the "formes frustes," to discriminate between the varieties of incomplete tabes, their combinations, and their widely different types; we have elaborated a much more comprehensive picture of this many-sided disease than would have been thought possible from the earlier, pure, and typical cases; but this is usually the case in studying the pathology of the nervous system. At first typical forms only are observed, and from these the clinical picture is constructed; by and by we recognize that this extends beyond the limits drawn, that other varieties, transitional forms, and related processes exist which not infrequently are exceedingly perplexing to the practitioner.

All these factors, however, have made the *diagnosis* of tabes more exact and more minute; we now fully recognize the gravity of the disease, even in its early stages, and the dawn of this century has just furnished us an important and most valuable aid to diagnosis in lumbar puncture and cytodiagnosis of the lumbar fluid.

That the *prognosis* of the disease is to-day vastly different from what it was fifty years ago when Romberg gave expression to his celebrated and hopeless view, will later be evident.

As a matter of course, from our modern etiologic knowledge new indications and broader methods of *treatment* have arisen; these are still being

developed and amplified, but so far have yielded no decided results; new methods of treatment, such as nerve stretching, suspension, and orthopedic treatment, have been introduced into practice, and have in great part again fallen into disuse; much more important, however, is the "compensatory practical treatment" (by movements, the "rééducation des mouvements" of the French) introduced by Frenkel, and especially advocated and practised by v. Leyden and his adherents; this, naturally, is only adapted to a late symptom of tabes, the ataxia, for which it is often beneficial.

Briefly reviewing the advance of the last thirty years in the teaching of tabes, and with what possessions it enters into the new century, we may say that in all probability the etiology of the disease has undergone the greatest change, that it has been worked out conclusively by demonstrating the intimate relation to syphilis; yet, in spite of this, much remains obscure—the auxiliary causes, the minute pathogenesis, etc.

On the other hand, the *symptomatology* of tabes, the recognition of different forms, of transitional stages, their *diagnosis* and *prognosis*, have all been greatly amplified.

In spite of this close investigation, the *pathological anatomy* and the pathogenesis of the disease are still doubtful; much which relates to the general pathology is still theoretical; the present *treatment* of tabes is still imperfect and unsatisfactory.

Therefore, notwithstanding our great advance, the enormous amount of labor expended, and the voluminous literature which has appeared, the twentieth century has before it a vast field in elucidating the teaching of tabes.

Let us hope that we shall get to the very depths of this problem!

II. ETIOLOGY. (THE CAUSES OF TABES)

It is obvious that with the introduction of tabes into pathology as a typical form of disease, the question of its cause immediately arose. Little care and acuity were shown in the answer. Authors were content to designate two etiologic factors, *sexual excesses* and *exposure to cold*, and even these were subject to doubt; in addition, over-exertion, profound emotion, nervous predisposition, rheumatic processes, and syphilis were mentioned, the latter of which even Duchenne did not greatly emphasize. These etiologic views convinced no one.

When Fournier reported (1871) the frequency of syphilis in the history of tabes, and this was soon after confirmed by Vulpian, Grasset, Gowers, and myself, a new factor appeared which formed the basis for numerous and thorough investigations. The number of those who adhered to Fournier's teaching steadily increased, for by compiling full statistics, by emphasizing the value of individual clinical observations and general considerations, they attempted to make the connection between syphilis and tabes more clear and more positive; the number of opponents who at first combated this view with other statistics and much reasoning, who attempted to make other etiologic factors prominent, and to emphasize their importance and frequency, constantly decreased; many an energetic opponent was convinced of the etiologic relation of syphilis, and only a few still cling tenaciously to the opposite view.

In the following presentation I propose to state facts which embody the

results of the investigations of the last 25 years; the reader will, however, be referred to special treatises for all details. A comprehensive and critical review is beyond the scope of this article.

We shall first consider the general circumstances under which tabes appears, and shall then discuss the special factors which are directly causative.

Tabes, upon the whole, is not a rare affection, and among the chronic diseases of the spinal cord it may even be regarded as common; whether it has become more frequent in our time, as some assume, is not certainly known, although it does not seem unlikely, especially when we consider its close relationship to a disease of the brain (progressive paralysis), in which this condition has been almost positively proven.

It is a disease of *adult* and *middle life*, and in the majority of cases appears between the ages of 25 and 50; it is rare in the aged, but cases have been observed which began after 60, and even later than 70; it is almost as rare in children, but recently cases of tabes in children as well as during puberty (infantile and juvenile tabes) have increased.

In regard to sex, tabes *predominantly* affects *males*; in my own large experience, which has been principally among the better classes, there were 1,258 tabetic men and only 63 women (therefore a ratio of 20 to 1). Other observers have found a much larger percentage of women, so that proportions of 10 to 1, 7 to 1, 5 to 1, and even 2.5 to 1, have been given. A very decided disproportion has been noted between the higher and lower classes, as well as between the different localities in which the affection occurred. Among women of the higher classes tabes is exceedingly rare; on the other hand, it is relatively more frequent in men of the higher than of the lower classes, and is much more common in women of the lower than in women of the higher classes, so that the number of tabetic women among the lower classes corresponds to that of tabetic men in the same class of society. This proportion is noted, as the rule, in large cities.

In fact tabes is much *more common* in the *city* than in the *country*; the larger the city the more numerous the cases of tabes, especially in women of the lower classes.

The disease prevails more extensively in *civilized countries* than in noncivilized; "civilization with its many deleterious factors appears to wield a predominant influence."

The social position seems to be of considerable importance in the etiology; the educated classes are much more frequently attacked than the uneducated, particularly merchants, travelling salesmen, and soldiers; among theologians it is rare; those who live much in the open air and are inured to cold and privation are not attacked to any special extent.

These peculiarities of the affection point to peculiar etiologic factors, which cannot be explained as the ordinary effects of cold, sexual excesses, trauma, etc.

We shall now consider what has been learned in the past 25 years concerning the *causes* of tabes.

Among these, according to my experience, *syphilis* plays by far the most prominent rôle. Statistics compiled from only the most carefully selected material, accurate histories, and exact clinical data prove its importance.

In illustration of this I shall first quote my own statistics which—begun with the view that tabes and syphilis had nothing in common—had been col-

lected in the past twenty-five years. I can guarantee their thoroughness and correctness, and the results which have never varied from the first hour (1879) to the present time seem to favor their reliability.

I shall not discuss the individual difficulties and the sources of error in these investigations; they have often been described, and with some practice and care in detail may in great part be overcome.

The best subjects for statistics are *male tabetics of the better classes*; from them alone are accurate and reliable reports to be obtained, far more so than from males of the lower classes, or even from women in whom, as is well known, the detection of preceding syphilis is often very difficult, and can be arrived at only by a roundabout process (through the husband, the physician, or the family, the prior social history, reports from hospitals, minute investigation for the sequelæ of syphilis, abortion, etc.). Nonne has published an especially valuable report on this matter.

My compilation, recently published,[1] of 1,100 cases of *tabes among the better classes*, shows that of this number 89.45 per cent. had previously been infected by venereal disease; among these 62.9 per cent. gave positive evidence of secondary syphilis, and 26.54 per cent. reported merely a chancre; doubtless many of the latter should also be regarded as positively syphilitic (because of their designating the chancre as a hard sore, of their having been treated with mercury and iodin, of leukoplakia in the mouth, of abortion in the wife, etc.).

Of the remaining 10.55 per cent. who denied infection, there were many cases which were more or less suspicious (because of repeated attacks of gonorrhea, of buboes, of questionable ulcers, of profuse leukoplakia of the mouth, of abortions and premature births in the wives, etc.) and which could not be regarded as positively free from infection. In this respect minute compilations of the last 900 cases showed that in at least 3 per cent. (more accurately 2.8 per cent.) of the cases the most carefully elicited history and closest investigation failed to reveal preceding infection. Any one familiar with the history of occult syphilis, "syphilis ignorée," syphilis insontium, will appreciate the importance of this fact.

Hence it appears from these voluminous statistics *that among 100 cases of tabes in men of the better classes about 90 had formerly suffered from syphilis or chancre, and only 10 were apparently free from infection.*

A striking and almost overwhelming demonstration! And yet these figures in themselves would not be conclusive (for with like statistics we might demonstrate that 80, 90, or a still greater percentage of tabetics had formerly suffered from measles, coryza, angina tonsillaris, influenza, and the like) provided it had not been simultaneously proven that in non-tabetics of the same class of society quite different relations exist. Therefore a control test must be made, i. e., we must determine what percentage of infected and non-infected are found in the same category, that is, among non-tabetic men of the higher classes over 25 years of age. I made this control test and, that it might not be partial, included even the paralytics and other patients with cerebral syphilis, etc.; this test now includes records of more than 10,000 men of the higher classes, and shows that of these only 21.5 per cent. had

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[1] Berliner klin. Wochenschr., 1904, Nr. 1-4.

been *previously infected* and 78.5 per cent. were *uninfected*; 9.8 per cent. of the former apparently had merely a chancre; and 11.7 per cent. had secondary syphilis.

The result is conclusive: Of those *uninfected* 10.5 per cent. had tabes and 78.5 per cent. were non-tabetic; of those *infected* 89.5 per cent. had tabes, and 21.5 per cent. were non-tabetics. Therefore, *among the tabetics there were almost 4 and ½ times as many infections as among the non-tabetic men* in the same class of society![1]

I believe these statistics to be most valuable and decisive, because they were based on very reliable data; but my statistics of men in the lower classes, and of women with tabes give almost the same results. I shall briefly quote these.

Among 158 tabetics in the *lower classes* 77.2 per cent. were infected, and 22.8 per cent. were uninfected with syphilis; therefore a smaller ratio than in the higher classes, yet, considering the difficulties in eliciting the history, a very high percentage. But the *control test* made in patients in my Clinic showed that among 1,300 cases (males over 25 years of age) only 6.54 per cent. were infected, and 93.46 per cent. were non-infected. This surprisingly low number of infections is probably accounted for by the circumstance that my clinical material was largely composed of country people. But other investigators also (Kuhn, Sarbó, Collins, and others) failed to find essentially higher figures in the lower classes.

Finally, we come to *tabes in women*. To simplify matters, I shall consider the higher and lower classes together, but I must remark that in the former infection could be much more positively determined (86.7 per cent. in a series of 16 cases) than in the latter. Combined, therefore, in a total of 63 cases, 44.44 per cent. showed *positive*, 36.5 per cent., *probable* infection, and in 19.05 per cent. *no infection could be demonstrated*. This result sufficiently agrees with the results in men.

So much for my own statistics! Far be it from me to regard these alone as decisive; but a brief review of the numerous cases in literature—and it is impossible here to enter into all the details—at once shows that many, and in fact most, other observers found similar or even higher percentages; I shall quote a few of these, and will state in this connection that earlier statistics—from about 1880—are not all reliable, as is shown by the fact that at first some physicians found only a small percentage, but, with more experience, this constantly increased. (See table on page 458.)

The statistics of *tabes in women* vary much more, and this is readily explained. In 13 cases Moebius found syphilitic infection in 80 per cent., and in 5 cases in 93 per cent.; Minor (8 cases) in 100 per cent.; Redlich (30 cases among the lower classes), positive in 23.4 per cent.; Kuhn of Berlin (78 cases), positive or suspicious in 64.1 per cent.; Kron (Berlin, 41 cases), positive in 44 per cent., most likely in 12 per cent. = 56 per cent.; Nonne (22 cases) in 77.3 per cent.; Silex (8 cases) in 87 per cent.; Fehre (Gerhardt's Clinic, 41 cases) in 66 per cent.; Collins (16 cases) in 70 per cent.;

[1] In pathologic anatomy it has finally been decided to adopt the control test. Fritz Lesser reports that in performing autopsies on 96 tabetics he demonstrated syphilis in 28 per cent., but in all other cases over 35 years of age which came to autopsy he found it in only 9.5 per cent.; therefore, it was thrice as common among tabetics!

	Infected
From France:[1]	
Fournier (last compilation of 1,000 cases)	93 per cent.
Labbé	93 " "
Martineau	95 " "
Déjérine	97 " "
Ferras	91 " "
Belugou and Faure (1,903–1,960 cases)	77 " "
From Germany and Austria:	
Voigt, first (1881), 67 per cent.; later (1885)	82 " "
Rumpf (1883), 66 per cent.; later (1887)	85 " "
Gg. Fischer, positive, 72 per cent.; probable	90 " "
Seeligmann (G. Fischer)	86 " "
Eisenlohr	60 " "
Bernhardt, first, 40 per cent., 60 per cent., 58 per cent.; finally	83 " "
Remak (1884)	63.5 " "
Strümpell	90 " "
Hirt (319 cases)	92 " "
Gerlach (Hitzig)	About 78 " "
Fr. Schultze	66–71 " "
Kuhn (Jolly's clinic, lower classes, 240 cases)	60.3–81.6 " "
Silex (Berlin)	81.5 " "
Dinkler (37 cases)	93 " "
Schittenhelm (Kast's clinic, lower classes)	67.8 " "
Sarbó (Budapest), higher classes	86.6 " "
" " lower "	74.6 " "
Redlich (lower classes)	65.2 " "
v. Halban (v. Krafft-Ebing's clinic, 236 cases)	13.5 + 73 " "
Coester (Wiesbaden, 93 cases)	82.8 " "
Determann (132 cases), positive syphilis	(7.2+) 72 " "
From England:	
Gowers (1889), 170 cases	75–80 " "
Althaus (1881), 90.6 per cent.; later (1884)	86.5 " "
Byrom Bramwell	76 " "
Mott (1903)	Positive, over 70 " "
From America:	
Seguin	72 " "
Spitzka	80 " "
Bonar (286 cases)	69 " "
Dana (50 cases)	68 " "
Collins (140 cases)	80 " "
From Italy:	
Negro (232 males, 1901)	89 " "
From Russia:	
Minor	60–70 " "
Gajkiewiez	90 " "
Anfimow (322 cases)	83 " "
Tumpowski (Warsaw, 257 cases)	64 " "
Homén (Helsingfors)	83 " "
Moczutkowski, positive, 36 per cent.; probable	72.2 " "

[1] Very few statistics from France have come to my knowledge in the last few years; as I know from verbal communications, the opinion is general in that country that tabes is in almost all cases a syphilogenous disease (compare Déjérine and Thomas, "Malad. d. la moëlle épinière," Paris, 1902).

Kojewnikoff in 63 per cent.; Friederichsen in 39.3 per cent.; Negro (25 cases) in 68 per cent.; Schittenhelm in 54 per cent., etc.

The total number of cases in these statistics of tabes certainly amounts to many thousands, and they all reveal the same conditions as my own.

But I cannot deny that there are compilations of cases which give a decidedly lower percentage; for example, that of A. Eulenburg, showing 36.8 per cent., of Hofmann 37 per cent., of Petrone 48 per cent., and others. Similar ones have been published, even up to most recent times, and usually based on a small number of cases, so that they may be added to the above statistics without decidedly modifying the result. Two of these, because emanating from very prominent authors, demand notice. They were published by Storbeck and Gutmann of v. Leyden's Clinic. These compilations are based on statistics of both men and women of the higher and lower classes of society. In his 108 cases Storbeck calculates 20.4 per cent., or, at most, 30.6 per cent. of positive syphilis (in the 75 men comprising his cases I calculated 60 per cent. as being previously infected although not positive syphilitics!), and in 111 cases Gutmann found only 35 per cent. with preceding syphilis; even on adding 20 cases from the records of a life insurance company, he found only 28.6 per cent.!

In the light of my own experience and that of other observers, I must admit that these low figures, which agree with a few other statistics, are absolutely incomprehensible to me, the more so because simultaneously, from other sources, in Berlin (Jolly's and Gerhardt's Clinics, Silex, Kron and others), figures much higher, even twice as high, have been obtained. It is not worth while to discuss the probable cause of these differences; an accidental distribution of the cases may play a rôle in these relatively low figures, or they may be due to a lack of thoroughness in the investigation, to an admixture of unreliable material, as, for example, of women from the lower classes (Storbeck reported 111 cases, among them 33 women, only 2 [!!] of whom were said to have been positively syphilitic), or to placing too great confidence in unreliable reports such as those obtained from soldiers who have become tabetic, or from life insurance companies whose statistics concerning preceding syphilis are unquestionably most unreliable—these may be the causes of the low figures, and time will reveal the true state of affairs. In any case I believe I may state that these statistics which give relatively low figures, when contrasted with the overwhelming majority of cases with high percentage, are not very important, and that even when added to the other figures they do not essentially change the result.

And this result is that from 70 to 90 per cent. *of all the cases of tabes are preceded by a syphilitic infection.* Experienced syphilographers who are familiar with syphilis occulta and syphilis ignorée are of the opinion that it is wise to recognize syphilis in 100 per cent. of the cases!

The question *by what length of time syphilitic infection precedes the appearance of tabes* is answered by the fact that in the overwhelming majority of cases (70 to 80 per cent.) tabes appears from 5 to 20 years after infection, quite frequently even in the first five years (certainly after the third year), more rarely from the twentieth to the thirtieth year; but there are reports according to which tabes appeared 34 and even 38 years after infection (the last case was confirmed by the positive cytodiagnosis of the cerebro-

spinal fluid!). Tabes therefore generally follows a preceding syphilitic infection after the same interval of time as do the other forms of tertiary, visceral, cutaneous, and mucous syphilis, and is therefore absolutely identical with so-called "*tertiarism*" in point of time.

The results of these statistics may be briefly summed up in the statement, previously made, that *nearly all tabetics have previously been syphilitic,* hence no one is likely to become tabetic unless previously infected by syphilis.[1]

Obviously the next conclusion is that *this preceding syphilitic infection is the most invariable, essential, and, we may even say, absolute prerequisite for the development of tabes.*

Another deduction is that *in the overwhelming majority of cases* (although not in all) *tabes is the direct sequel of syphilis,* is a *post-syphilitic* or a *positively syphilogenous disease,* also that it may represent a *late form of syphilis of the nervous system.*

This opinion is held by most authors, by some only in a modified form, by a few it is directly denied; these latter will admit no connection between tabes and syphilis.

It must be conceded that—in spite of striking facts—statistics alone do not justify this conclusion, neither do they *positively* prove the relation between tabes and preceding syphilis.

Searching for other facts and experiences to confirm this conclusion, we find many clinical observations of the last few decades which positively sustain the opinion that tabes is actually a syphilogenous disease, even though it be a peculiar manifestation of syphilis. I shall quote some of these.

1. In *regular, periodic sequence tabes follows syphilis* in from 3 to 10, or, at most, 20 years, in rare cases somewhat later. This is based on the fact that if *syphilis is acquired very late tabes will appear late*; there is a series of observations which show that after a recent syphilitic infection in the 48th, 54th, 55th, 57th and 68th years, tabes appeared in the 58th, 59th, 66th, 68th and 70th years respectively; the mere fact of the sequence of two conditions, in themselves rare, warrants the conclusion of an intimate connection between them.

In the same sense the unusually early appearance of tabes after early acquired syphilis may be mentioned (infection at 19 years—tabes at 22!); especially noteworthy is the frequent *occurrence of tabes in children* (*infantile and juvenile tabes*) which may almost invariably be attributed to hereditary syphilis or that acquired during infancy. The observations of Rumpf, Fournier, Dydinski, Brasch, Kron, Gumpertz, Babinski, Souques, myself, Linser, Brookshank-James, v. Rad, Nonne, and others permit not the slightest doubt of this. The fact is interesting that infantile tabes appears in both sexes with almost equal frequency, as does hereditary syphilis.

Tabes occurring in syphilis which is still florid, as well as in the presence of various tertiary symptoms in the skin, mucous membranes, bones, etc., *in tabes of prolonged existence,* favors the intimate connection of both affections; no less so does the appearance of other *phenomena of nervous syphilis,* such

[1] This expression "syphilitically infected" is used here in its broadest sense, that the mere existence of a chancre is presumptive evidence of an actual syphilitic infection. (Compare Erb, Aetiol. der Tabes. *Volkmann'sche Samml. klin. Vortr.*, N. F., Nr. 53, p. 521, 1892.)

as hemiplegia, paraplegia, headache with nocturnal exacerbations, etc.; all these things are occasionally, and by no means rarely, observed. (The pathologico-anatomical facts which may be utilized in the same sense will be discussed later.)

The *parallel frequency of cases of syphilis and tabes* in men and women of the higher and lower classes is important; both are much more common in men of the higher classes than in women, while in the lower classes the proportion of women to men is very much greater; Minor has shown that syphilis and tabes are much rarer among the Jews in Russia than among the Russians; the same is also true of the *different classes of society.* Those classes which are most often exposed to syphilitic infection, such as merchants, commercial travellers, officers, etc., also furnish the largest contingent of tabetics; among clergymen, syphilis and tabes are hardly ever observed. The few tabetic clergymen that I had an opportunity of observing had all had syphilis.

The appearance of *tabes in man and wife* is almost decisive; not seldom they are affected by tabes one after the other (in a similar way tabes may be combined with paresis); in almost all such cases syphilis was demonstrated (Raecke found it in 90 per cent., Hudovernig in 96.3 per cent., and Pourreyron even in 98 per cent. of the cases!). It is clear that this cannot be due merely to living together, to deleterious effects, to excesses, etc., which cause tabes, otherwise among the hundreds of thousands of married couples in which this occurs many would be affected who are not syphilitic; but the factor of syphilis alone is common to all, and is etiologically of the utmost significance.

This significance is even increased when tabes or paralysis (infantile or juvenile) or cerebral syphilis (so-called *family tabes*) is found in the *children* of such tabetic parents.

The extreme *rarity of tabes in virgins* need hardly be mentioned; but as a matter of fact, tabes has been observed among "virgines." Syphilis was almost invariably demonstrated in these cases, but this had been acquired either hereditarily, in childhood, or subsequently in an extragenital manner.

I must devote a few words to the remarkable statement which, however, is not yet sufficiently corroborated, that *tabetics appear to be immune from syphilis.* In the early stages of tabes many patients are exposed to infection, but I have never heard of recently acquired syphilis in a patient with severe tabes; even if such a case should be observed, the strong probability of its being a reinfection would decidedly weaken this proof in a negative sense.[1]

Among the few symptoms of the pathologic picture of tabes which indicate a syphilitic origin we must first mention paralysis of the muscles of the eye, which is so commonly observed, especially in the early stages of tabes, and is a forerunner of this affection; to every experienced eye specialist—provided no other deleterious factors, such as intoxication, cerebral affection, etc., can be detected—these suggest syphilis, and to this they are almost exclusively due. But another most invariable and significant symptom of tabes is

[1] It might be possible to solve this question by the "experimental" test reported by v. Krafft-Ebing at the Moscow Congress as having been made in the case of paresis. But in the present state of affairs this is hardly practicable.

of greater importance: *Spinal myosis with reflex rigidity of the pupil* (Argyll-Robertson). Babinski and some French authors succeeded in proving that this condition is invariably due to syphilis, and they regard it as almost pathognomonic of syphilitic infection; it is interesting to know that this view has been confirmed by cytodiagnosis of the cerebrospinal fluid.

The *lymphocytosis* of this fluid, which is almost invariably found in tabes, and even now may be regarded as a valuable clinical symptom, will be discussed later; here it need only be stated that it decidedly points to the syphilitic nature of tabes. The condition, however, has been so recently observed that no conclusive opinion is as yet justified.

Every unprejudiced observer who considers these facts, and keeps statistics in mind, must admit that they decidedly bear out the statement expressed above that "tabes is positively a syphilogenous disease"; each of these facts, while not in itself absolutely decisive, strengthens the value of the others, and their sum makes a convincing "indicational proof" of the relation of tabes and syphilis.

Nevertheless even to-day this connection is not generally recognized, and some prominent opponents absolutely deny it for the following reasons:

Statistics were rejected in this investigation. The data upon which they were based were declared to be incorrect, and their importance was minimized; to-day there can be no serious difference of opinion on this point; statistics must undoubtedly be recognized as a valuable aid in investigation, and in regard to number and quality their results are so uniform and convincing that no objection can now be made to them.

It was hoped that pathological anatomists would decide these questions, it was believed that the tabetic changes in the spinal cord were *not* specific, and did not conform to the recognized type of syphilitic alterations (gummatous or cellular infiltration, inflammation, proliferation, etc.), therefore could not be of syphilitic nature. It was left to these pathological anatomists to decide what processes were or were not syphilitic, but quite incorrectly! In the present state of pathologic anatomy, this will by no means prove which of the lesions are syphilitic, and still less so which are non-syphilitic. If, notwithstanding this, it has become customary—and quite properly so!—to regard certain changes like small-cell infiltration, the "gummatous" products in the vessels, meninges, and central nervous system, as undoubtedly syphilitic, this view is sanctioned by custom, but pathologic anatomy can by no means furnish convincing proofs. It has chiefly been justified by clinical observation, and even to-day it is clinical observation that usually decides these questions, and if we clinicians say "Here are spinal forms of disease (for example, tabes) in which in 90 per cent. of the cases a preceding syphilis may be demonstrated," pathological anatomy, whether it desires to do so or not, must admit the alterations found to be syphilitic or of syphilogenous origin. If doubts arise, it is impossible to prove that these conditions, especially the gray degenerations, are not of syphilitic origin. So far as I can see, pathologic anatomists with increasing unanimity admit that these gray degenerations, the primary atrophy of the nerve tracts and nerve-cells, as well as "specific" cellular infiltrations, may be produced by syphilis although they do not have its characteristic appearance.

I have investigated this question repeatedly, first in the year 1892, and

again in 1902,[1] and from weighty reasons I have demonstrated that these gray parenchymatous degenerations and atrophies may be considered and designated as syphilogenous with as much justice as the so-called "specific," gummatous, late products of syphilis. I must refer the reader to these investigations, and will not further discuss the subject at this point.

But in tabes, besides the typical gray degeneration of the posterior horns, other conspicuous and "specific" changes are demonstrated with increasing frequency in the meninges, in the vessels, etc. I have compiled these cases in the article last referred to. Prominent French observers (Nageotte, for example) have lately stated that "specific" changes, such as small cell infiltration, had invariably been found in the meninges, especially in the small veins of this membrane, and have declared them to be typical of syphilis.

These authors (Pierre Marie, Nageotte, and others) with increasing certainty ascribe the pathologico-anatomical origin of tabes to a "syphilosis" of the meninges, and look upon the lymphocytosis of the cerebrospinal fluid which is almost invariably found in tabes as confirming this opinion. Therefore, this pathologico-anatomical finding decidedly points to the syphilogenous nature of the tabetic changes.

Some of these facts still require confirmation and call for further research, but, when all is considered, the objections raised to the views of pathological anatomists which have been regarded as decisive cannot in my opinion be sustained.

This is also true of the criticism frequently made and regarded as important, that tabes can have nothing in common with syphilis because specific antisyphilitic treatment is fruitless. Aside from the fact that this demurrer is objectively incorrect, since there are undoubtedly many cases in which specific treatment has been shown to be beneficial, bringing about an almost complete recovery, the fact that it cannot be cured by mercury and iodin is no proof of the non-syphilitic nature of the affection. Who in the world dare maintain that in persons previously syphilitic all forms of disease which cannot be cured by mercury and iodin are therefore of non-syphilitic nature? Why do we find upon the autopsy table so many typical cases of "specific" meningitis, of meningomyelitis and encephalitis, with gummata, with specific endocarditis, with hepatitis and aortitis, even after energetic treatment with mercury and quantities of potassium iodid? This argument had better be dropped!

At the onset of this discussion it was remarked that specific treatment had only the slightest effect upon the tabetic changes in primary or secondary degeneration of the nerve elements, as well as upon the insidious development, the long duration, and the progressive character of the malady. If specific treatment often fails to remove specific products and cellular infiltrations, how much less can we expect it to act upon the severe tissue disturbances which affect the nervous elements themselves! Therefore this objection, too, is not sustained by the facts.

Another criticism has been made with some degree of justice, namely, that compared with syphilis tabes is relatively rare! It has been deemed expedient

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[1] Erb, "Die Aetiologie der Tabes." *Volkmann's Sammlung klinischer Vorträge*, Neue Folge, 1892, Nr. 53, p. 529.—Erb, "Bemerkungen zur pathologischen Anatomie des Centralnervensystems." *Deutsche Zeitschr. f. Nervenheilkunde*, 1902, XXII.

to determine statistically how many syphilitics become tabetic, and how many tabetics were previously syphilitic. And as it is notorious of the syphilitics that but few become tabetic, it was triumphantly declared that, *ergo*, tabes could not be the result of syphilis. Every one familiar with the pathology of syphilis will at once admit that this reasoning is erroneous, and that the form in which the question has been put is entirely wrong. Syphilis is a disease with numberless early and late symptoms; a few of these are common and almost invariable, many are rare, a few exceedingly rare, but all are signs of syphilis! Shall hepatic syphilis, syphilitic aortitis, cardiac gummata, syphilitic meningitis and meningomyelitis, syphilitic spinal paralysis, gummata of the brain and spinal cord, etc., be cast out from the pathology of syphilis and be declared to be non-syphilitic merely because they are rare sequelæ of syphilis? Certainly not! I believe that no one will demand this. *Now all of these conditions are more rare than tabes, some of them decidedly so.* Therefore tabes may still be considered as belonging among post-syphilitic diseases. And this is much more likely than in the previously mentioned forms of disease. It has never been stated that most syphilitics become tabetic, nor that "where there is much syphilis there is also much tabes." But we maintain that nearly all tabetics have previously been syphilitic, and this is the important point, for among 100 tabetics at least 80 to 90 (perhaps even more) were previously infected, and this is the main question, not, however, how many of 100 syphilitics will become tabetic, whether one, or 2, or 5, or 10. It is very interesting to determine this ratio in order to appreciate correctly the dangers of syphilis, but not to determine the relation of this affection to tabes.[1]

Such a question concerning other conditions would lead to remarkable etiologic views: There are thousands of alcoholics, yet how few are attacked by polyneuritis or hepatic cirrhosis! Innumerable workers in lead show the signs of lead intoxication, yet how few suffer from lead paralysis or contracted kidney! Incalculable cases of diphtheria, yet how rare is post-diphtheritic paralysis! There are thousands of tuberculous patients, yet how few develop Addison's disease! Etiology is not thus investigated!

Another erroneous statement frequently made and regarded as a forcible objection is this—that, in certain countries in which syphilis (as well as paresis) is exceedingly prevalent, tabes is unknown or exceedingly rare. These are countries with both a wholly uncivilized and also a very highly civilized population, such as Bosnia, Herzegovina, and Abyssinia; it is also true of the Kirghiz, the negroes of East Africa and America, and also of the Japanese. At first this appears to be quite remarkable; but, even though the main facts are correct, they must chiefly be considered from the same standpoint as the frequency of tabes in syphilitics in general. Such persons need not become tabetic, and there may be factors in the mode of life, in the greater or less development of these countries, to prevent it; perhaps there is a peculiar, modified, and attenuated form of syphilis such as is so frequently observed in countries where syphilis is endemic.

[1] The assumption that tabes is comparatively rare among prostitutes was found upon minute investigation to amount to nothing: Among 36 over 25 years of age Kron found 5 cases of tabes (but none among 148 younger ones), and Jadassohn declared in 1896 that tabes was quite common in prostitutes.

But these assertions are not even true, as is proven by many recent investigations; I have lately discussed this question minutely in two different articles,[1] and have found that the actual conditions are quite different from what has been stated: In Japan there are many tabetics (Nose) giving a high percentage of syphilitics; tabes is not rare in Abyssinia (not more so than are nervous affections in Vienna, v. Halban); among the negroes of America it is always associated with syphilis, and it has been observed in Bosnia and Herzegovina. The Transactions of the Eighth German Dermatological Congress at Sarajevo (September, 1903) furnished an exceedingly full report on this subject, but it can be critically discussed only after the full report of the Congress, which it is hoped will bring to light some very interesting facts and considerations.

I shall not here enter into details, but, leaving this exotic tabes to the critics, I shall limit myself to the consideration of tabes as it exists in Europe and America, particularly among men in the better walks of life; what has been shown to be true and invariable among these will probably also prove true of the lower classes and the opposite sex.

The compilation of the foregoing leads me to the conclusion that for the previously mentioned portion of the population tabes is undoubtedly a syphilogenous affection.

But this does not clear up the whole question; the etiology of tabes is not yet exhausted. Are there not other causes or may these be excluded? These matters must be investigated.

It is no doubt true that but relatively few syphilitics later become tabetic; how many we do not as yet know; perhaps one or two, possibly three to five per cent. or even more; this proportion varies according to the different regions and the different classes of society.

Why do so few become tabetic?

This brings us to the question of the *etiological factors* and the *auxiliary causes* of tabes in syphilitics. Only a few new ones can be added to the causes which have been known since 1880, and the importance of which will now be inquired into. In 1892 I collected reports of nearly 300 cases of tabes which I attempted accurately to classify, and made an investigation [2] to which I here refer. Recently Schittenhelm [3] published a similar investigation with corresponding results. I may, therefore, be brief.

(1) *Direct heredity* plays but a slight rôle in tabes. This is not remarkable, since tabes now appears to be an "exogenous" disease; otherwise, considering the great number of tabetics who for a long time are capable of rearing children, tabes would be more frequently transmitted from father to son or from mother to children. In the quite isolated cases in which this has been observed, syphilis was demonstrated in both generations, therefore nothing was proven as to the heredity of *tabes*. It also leads us to form an opinion regarding so-called "family tabes"; that is, the occurrence of tabes among children of the same family, or in parents and children, etc. In the not infre-

[1] Erb, "Syphilis und Tabes." Offener Brief an Prof. v. Krafft-Ebing, *Jahrbuch f. Psych. u. Neurologie*, 1902.—Erb, "Syphilis und Tabes." *Berliner klin. Wochenschr.*, 1904, Nr. 1–4.

[2] *L. c. Volkmann'sche Sammlung*, 1892.

[3] Schittenhelm, *Deutsche Zeitschr. f. Nervenhk.*, 1903, XXIV.

quent cases of tabes in brothers, syphilis was almost invariably demonstrated (I published a number of such cases); in cases in which both parents and children suffered from tabes (such cases are described in my last report—1904), syphilis of the parents and hereditary syphilis of the children was always demonstrated. But although this transmission may perhaps be explained by the peculiarities of the syphilitic poison, nevertheless we cannot deny the fact that in some families there is a certain predisposition to tabes when syphilis already exists.

(2) Such cases should be classified as due to *neurotic predisposition,* the importance of which in the development of tabes was greatly exaggerated by Charcot and his pupils. If *every* nervous affection, even in distant relatives, is to be regarded as the sign of a neuropathic predisposition, in the case of tabes these will be found to be very common—just as in most other diseases. I found this condition in scarcely one-third of the cases (28 per cent.), while Fournier found it in only 25 per cent., Möbius, Oppenheim, Voigt, and Rehlen only in 9 to 14 per cent. This indicates no noteworthy influence. I observed a *nervous predisposition* in the patient in 42 per cent. of the cases, but even this proportion is unreliable, and is only slightly in excess of what every nervous specialist nowadays finds among his male patients over 25 years of age. No doubt, however, the growing nervous predisposition and nervousness of our times may have some effect by diminishing the resistance of the nervous system, and thereby increasing its susceptibility to the syphilitic poison.

It is certainly not permissible for us to confine a congenital endogenous predisposition to tabes alone, which view culminates in Benedikt's statement, "Tabicus non fit, sed nascitur." We need merely to consider the effect of this statement upon the etiology and pathology of tabes, as we know them to-day, to appreciate its rashness. Are persons perhaps born with a predisposition to syphilis?

(3) *Chilling of the body,* by some authors considered the chief deleterious agent, is unquestionably a cause of tabes. Even when we consider how incidentally cold is often mentioned by the patients as a cause, and is so accepted by physicians, when we consider how many patients for years regard their pains to be "rheumatic," and are of the firm opinion that they must have taken cold, even though they cannot accurately state when—nevertheless a certain number remain in whom exposure to cold immediately preceded the first symptoms of tabes, and the conjoint and prolonged effect of a damp and cold work-room, living-room, or sleeping-room, of laboring in a swamp, or in water, or snow, of cold journeys during the winter, and the like, may be properly regarded as predisposing to the affection; but all this is observed almost exclusively in those previously syphilitic. Moreover, in my statistics a short interval is mentioned only in 34.5 per cent. of the cases, and for reasons above stated this figure is probably too high. In any case we cannot maintain that taking cold is the most important or the sole cause of tabes.

(4) The consensus of opinion among recent authors is that *sexual excesses,* which were at first regarded as almost exclusively the cause of tabes, and which have again been brought forward prominently by Moczutkowski, apparently play but a slight rôle. I found this cause mentioned only in 15.8 per cent., but this proportion is perhaps somewhat too low because the facts are so diffi-

cult to determine. Undoubtedly this cause is operative in individuals who commit sexual excesses, as in young married people who return from their honeymoon with the first tabetic symptoms, and especially, as I have repeatedly noticed, in elderly men who have married young wives.

(5) Similar conditions are noted after *bodily over-exertion and long marches*, for example, in the army, while hunting, upon fatiguing journeys, during military maneuvers, in Alpine climbing, bicycling, etc. Also from excessive exercise among so-called leg-workers, those who must stand or walk the entire day; for example, waiters, policemen, postmen, etc. I found this was supposed to be the cause in 27 per cent. of my cases; this, however, was not wholly correct, as is borne out by the investigations of Kron in sewing machine workers. But the view is quite justified that all of these occupations debilitate the nervous system, and lessen its power to resist the syphilitic poison.

Here we will briefly consider Edinger's *substitution hypothesis*, according to which the increased combustion of material by work, function, and exertion, etc., is not fully compensated and thereby leads to defective nutrition and to degeneration of the functioning elements. Under some circumstances, especially when syphilis, anemia, impoverished nutrition, or constitutional affections, etc., coexist, this lack of restitution produces characteristic tabetic degenerations in the central nervous system. This was Edinger's ingenious and convincing hypothesis for a number of functions in tabes as well as for their disturbance. But in my opinion a much better basis of support is necessary; for thousands and thousands of persons daily experience these effects, yet do not become tabetic, unless they are simultaneously syphilitic. And innumerable persons are debilitated by anemia, tuberculosis, digestive diseases, anxiety, and sorrow, yet do not become tabetic from these same deleterious influences unless they are also syphilitic. Edinger, as I know personally, is in entire accord with the view that syphilis is the most essential and perhaps the sole cause of tabes, but he thinks his theory explains the fact that definite areas of the spinal cord and nerve tracts become susceptible to the attacks of the syphilitic poison, and from this cause the typical picture of tabes, sometimes in isolated cases also the atypical picture, appears. Of course, this needs to be definitely proven; at present, it seems to depend much more upon the specific action of the syphilitic virus than upon the nature and localization of the functions which are overtaxed.

(6) *Mental exertion, over-exertion in any occupation, excitement, and psychical emotions*, which are often mentioned by patients as the causal factors, appear to act in the same way; they have a depressing effect, damaging the entire nervous system, and thus reducing its power of resistance to syphilitic intoxication. I can give no accurate figures concerning this point.

(7) *Traumata* of various kinds, a fall from a horse or from a great height, severe shock to the body, the fracture of a leg, continuous shock for years in certain occupations, accidents upon the railway, or in some occupation, etc., are frequently mentioned, and the recent laws concerning accidents have an added importance thereto. Several authors (E. Schulze, Spillmann and Parisot, Trömmer, Klemperer, and others, and finally v. Leyden) favor this view. Others (Hitzig, Mendel, Collins, Windscheid, and Schittenhelm) have sharply criticized it. Even the circumstance that trauma precedes less than 5 per cent. of all cases of tabes assigns to this factor a subordinate rôle in its

etiology. Cases in which syphilis has certainly not preceded, and in which before the trauma tabetic symptoms were positively absent—in which, therefore, the genetic connection could not be proven—are exceedingly rare; the majority will not withstand strict investigation. Taking into consideration the cases of chronic progressive diseases of the central nervous system, such as progressive muscular atrophy, amyotropic lateral sclerosis, chronic anterior poliomyelitis, multiple sclerosis, syringomyelia, and the like, which occur in connection with *trauma,* we must admit at least the possibility of a pure traumatic tabes; but it is certainly extremely rare. It appears, however, to be proven that when syphilis exists such a trauma may occasionally lead to the development of tabes, that the first tabetic symptoms are apt to follow it, and that very often, if tabes is beginning to develop, such a trauma decidedly aggravates and accelerates its course.

(8) The effect of certain *poisons* must also be considered. In addition to ergotin which, as Tuczek ascertained by thorough investigation, produces a disease of the spinal cord somewhat resembling tabes but not identical with it, and which may be designated as "ergotin tabes," I must also mention "alcoholic tabes," which is merely a peripheral multiple neuritis, some of the symptoms of which resemble tabes.

Several authors have maintained that mercury is a "tabes poison," and that syphilitics who have long been treated with large doses of it are apt to develop locomotor ataxia. Fournier and others have disproven this ridiculous assumption, and Neisser and P. Cohn have also lately shown it to be absurd; to say nothing of the fact that tabes never appears as a sequel of chronic mercurial poisoning (in mirror workers, etc.), it has been demonstrated that by far the overwhelming majority of tabetics have never been treated by Hg, or very incidentally and for a short time. This should be sufficient to stamp this constantly expressed opinion with its true value.

But the question is quite permissible whether poisons such as *alcohol and tobacco* taken immoderately have any influence upon the development of tabes. I find them mentioned in only about 18 per cent. of the cases, a figure which appears to be too low. That either of these poisons alone is capable of producing tabes I believe to be exceedingly unlikely, and, considering that we have recognized another specific poison as the chief cause of tabes, I think this may be absolutely excluded; on the other hand, the fact that these poisons may possibly prepare the soil for the syphilitic noxa, or in combination with it may produce tabes, seems very likely. Further researches must be undertaken to solve this question.

(9) Little is known of *other diseases, such as infections,* etc., which may cause tabes. Tabes may occasionally appear after such affections provided they have decreased the resistance of the nervous system to the syphilitic virus. The experience of the last 15 years has taught others as well as myself that *influenza,* which was at first regarded as a very mild malady, has this effect; it is certain that if influenza be added to tabes (as is also the case with many other diseases) it decidedly aggravates the affection and accelerates its course, and there is no doubt that the first tabetic symptoms occasionally appear after an attack of influenza; but whether this alone without preceding syphilis will produce tabes is doubtful, and remains to be proven. At present I am ignorant of such an occurrence.

(10) *Gonorrhea,* which I recently[1] brought into this discussion, is still to be mentioned. It is true statistics show only 50 cases of tabes, in 90 per cent. of which there was a history of gonorrhea (of course 88 per cent. of syphilis also). In the male population of the higher classes (600 cases), only 50 per cent. were found to have gonorrhea. Close research, however, shows that since almost all the tabetics previously had syphilis only those cases could be used for comparison in which there had also been syphilis or chancre, and among these there were 75 per cent.; therefore there is not a sufficient difference to indicate the predominating influence of gonorrhea in the origin of tabes. This is also shown by another investigation which reveals that of 45 tabetics with gonorrhea 90 per cent. were simultaneously syphilitic, and of 265 cases of gonorrhea which were not tabetic, only 34.7 per cent.

My present opinion is that it is most unlikely that gonorrhea has any influence upon the development of tabes. Nevertheless the relation of these diseases should be further investigated; the combination and cumulation of "poisons," of "toxins," is possibly of importance. Here I may refer to the opinion Hitzig expressed in 1892 that tabes is perhaps not due to the actual poison of syphilis but also to some other specific poison, and that these are simultaneously transmitted, occasionally also with the toxin of soft chancre (perhaps with that of gonorrhea?). In this connection who does not think of the remarkable theory of "syphilis a virus nerveux"?

Nothing is now known of other injurious factors as causes of tabes, but it is self-evident that those which have been enumerated may occasionally exist in great numbers. There are many persons who expose themselves to all the injurious effects of over-exertion and cold, also drink and smoke to excess, and pass their nights in gambling or with women, etc., and who readily succumb to tabes provided they have been previously infected. A number of tabetics have admitted this to me.

In considering the numerous conditions which may be the "cause" of tabes, the presumption is obvious that tabes may frequently *result from the combined action of several,* and it is well to decide which are the most important; statistics show a certain degree of uniformity. They indicate that in nine-tenths of all the cases of tabes there is a syphilitic infection, in about one-third tabes is preceded by exposure to cold, in about one-fourth by over-exertion, in about one-sixth by sexual excesses, in about one-twentieth by traumata, and that more than one-third of all tabetics belong to neurotic families.

This shows the powerful influence of syphilis, and it becomes still more evident when we endeavor to ascertain whether, and in what proportion, these causes alone are operative. In 1892 I published a Table which shows that in 281 cases of tabes the disease was preceded by:

Syphilis alone	in 27.0 per cent. of the cases
Neuropathic predisposition alone	" 0.7 " " " " "
Refrigeration alone	" 1.4 " " " " "
Exertion alone	" 0.3 " " " " "
Sexual excesses alone	" 1.0 " " " " "
Trauma alone	" 0.3 " " " " "

[1] Erb, *Berliner klin. Wochenschr.,* 1904, Nr. 1–4.

This fully demonstrates the predominating influence of syphilis. It also shows that the other damaging factors alone are unimportant, and *that almost without exception they are active only when combined with syphilis* (which is also shown by the Table published at the same time).

These facts and considerations lead to the conviction *that syphilis is the most common and chief factor in the development of tabes.* In the overwhelming majority of cases it is the main cause, beside which all others, widely divergent in themselves, are merely auxiliary factors and predisposing causes.

Curiously enough, tabes frequently follows mild, rapidly healing forms of syphilis which have been almost without symptoms, and have had little or no treatment. This should be investigated. Certainly experience has proven that even a thorough and long-continued specific treatment will not absolutely prevent a subsequent outbreak of tabes, a fact which is true also of some of the other forms of the tertiary stage.

The recognition of syphilis as the principal cause of tabes sheds light upon the peculiarities and the frequent occurrence of the disease; this was clearly set forth by Möbius in his article on "Tabes" (1897) as follows: "This accounts for the appearance of tabes in middle life, rarely in the aged and in children; its overwhelming frequency in men, its extreme rarity in women of the higher, its greater frequency in those of the lower classes; its uniform occurrence in children of both sexes; its prevalence in large cities, the liability of certain occupations, and the exemption of others (that of theologians!). I advise the reader to investigate these features for himself. They are highly convincing."

This makes it clear that civilization with all of its nerve-damaging consequences, with its increased facilities for debauchery and luxury, with its wide distribution of syphilis, its tendency to over-exertion and irritation of the nervous centers, etc., has an essential influence upon the development of tabes, and that for this affection, as v. Krafft-Ebing tersely remarked in Moscow of progressive paralysis, "syphilization and civilization" are the causative factors. This explains the great rarity of tabes among uncivilized people, even when they are thoroughly infected with syphilis.

But, after all, it cannot be denied that there are cases of tabes in which the most careful research will disclose *no trace, and will even refute the possibility, of syphilitic infection.* Upon close investigation the 10 per cent. of non-infected in my chief Table is decidedly reduced, for in many cases there is a possibility, or likelihood, of syphilitic infection. Three per cent. at least are beyond suspicion, and I have seen not a few cases (about 5 per cent.) in which the most minute questioning revealed absolutely no cause for the affection, neither syphilis nor any other factor. What is to be done with these cases?

As is well known, Möbius energetically maintains, and for excellent reasons, *that under all circumstances tabes is a meta-syphilitic (syphilogenous) disease, a primary atrophy of the nervous elements of which syphilis is the conditio sine qua non.*

All the facts lead to the conclusion that only a poison with chronic action can be the cause of tabes. This applies to the poison of syphilis, which in itself is just as specific and peculiar as is tabes as a disease. There is little

likelihood that in pathology some other poison produces exactly the same disease; certainly there is no example of it. Consequently, syphilis with its poisonous effects must be regarded as the exclusive cause of tabes.

I must admit that Möbius's reasoning is very forcible. The small proportion of tabetics in whom no syphilis can be demonstrated does not prevent us from regarding *tabes as exclusively due to syphilis*; our knowledge of syphilis occulta and the fact that in from 30 to 65 per cent. of the cases of undoubted late syphilis (gummata, etc.) there is no evidence of preceding infection prove this. But the number of "doubtful" cases is so great that we cannot at once ignore them on merely logical grounds. It is still possible that tabes may be produced by unknown infections, by bacterial or other poisons, or a combination of various baneful factors, and although such poisons may be unknown to-day they may be discovered to-morrow. We have not yet reached the end, but only the beginning, of knowledge regarding such action of poisons.

Although I incline very strongly to Möbius's theory, and believe it more than likely to be true, yet I adhere to my reiterated opinion that there is as *yet no absolute proof that tabes is invariably a syphilogenous disease.*

So far as I can see, the result of these discussions concerning the cause of tabes, and the conclusion which is to-day shared by most experts, are as follows: "*That, among all the causes of tabes, syphilis is by far the chief and most common, that tabes in the overwhelming majority of cases—perhaps in all—is due to syphilis*, that it is, however, a relatively rare sequel or manifestation of syphilis, and that other deleterious factors are auxiliary and predisposing causes for its development."

How syphilis acts, what is the nature of the tabetic disease, what its pathogenesis, and what part the so-called auxiliary causes play, will be explained after we have discussed the pathological anatomy of tabes.

We will first consider the symptomatologic picture of the affection.

III. SYMPTOMATOLOGY

Since my comprehensive description of the pathologic picture of tabes appeared in Ziemssen's Handbook (1876) many additions, although perhaps not of vital importance, have been made to it, and in many ways the symptomatology has been minutely elaborated and made more accurate.

To avoid being prolix, I will delineate as succinctly as possible the individual features which form the nucleus of the pathologic picture, and will emphasize their fundamental and most important points; special consideration will be given to new view points, those which aid in the diagnosis, and the symptoms which are most important to the practitioner; the numerous and variable symptoms which are not typical will form a framework surrounding the many-sided, interesting, and inexhaustible symptom-complex.

As a rule, we first note in tabes an *introductory*, prodromal, neuralgic, or *pre-ataxic stage*, the duration of which may vary from a few months, which is rare, to many months or several years, occasionally even decades,[1] the dis-

[1] Within the last few years I have seen two cases in which typical lancinating pains with objective tabetic symptoms had persisted for fully 22 to 24 years without a trace of ataxia.

ease terminating on the appearance of the characteristic and peculiar motor disturbance known as ataxia.

This pre-ataxic stage is characterized by a series of *subjective disturbances*, localized in various regions, which usually introduce the affection; the most prominent are peculiar *disturbances of sensation, lancinating pains, paresthesia* of the lower extremity and of the trunk, *a feeling of exhaustion or of uncertainty in the leg*, weakness of the bladder and sexual organs, finally the disturbance of certain cranial nerves (*the nerves of the muscles of the eye*—diplopia, strabismus; the *optic nerve*—amblyopia, feebleness of vision); on the other hand, there are a number of significant *objective symptoms* which, even from the beginning, permit the recognition of the severe affection: *Reflex rigidity of the pupils, absence of the patella tendon reflex, swaying upon closure of the eyes, and certain typical sensory disturbances in the legs and in the trunk* (definitely localized hyperesthesia of the feet and of the trunk, hyperalgesia, analgesia, and the slowing of pain conduction, regional hyperesthesia to cold, etc.); finally, *strabismus, double vision, and amblyopia* from atrophy of the optic nerve.

The disease may set in with any of these symptoms. Sometimes one or another subjective or objective symptom may precede for a varying time, and then other symptoms are added. Certain symptoms, however, almost invariably introduce the affection, while others are less constantly noted.

To the former belong extremely typical and *lancinating* neuralgic (or lightning-like) *pains*: Acute, stabbing, boring, darting, paroxysmal pains, localized sometimes in one region, sometimes in another, often persisting in the affected area for hours, sometimes for days. They are of varying intensity, often appearing as sudden, flashing, but very mild sensations of contraction; often the attacks are of almost unbearable severity, and by their frequency and duration they keep the patient in misery (tabes dolorosa); mild stroking, the touch of the clothes, etc., will usually, to a greater or less degree, increase the hyperesthesia, while firm pressure over the painful spots will often give relief. They are sometimes localized in the skin, frequently in the deeper soft parts, in the bones, and in certain nerve regions (the sciatic nerve); almost invariably they first attack the lower extremities—the calves, the shins, the dorsum of the foot, and the thigh; they then extend to the trunk where they appear as most severe girdle pains, and to the thorax; rarely, at least at the onset of the affection, they attack the upper half of the body (the arms, in the ulnar nerve), the neck, or even the face (tabes superior). They always appear to be influenced by changes in the weather (by rain, fog, wind, the first snow, a low barometer, storm, or humidity), therefore are most frequent in the spring and autumn; for this reason they are usually regarded as "rheumatic," and many patients never refer to them as "pain" but as "rheumatism!"

Over-exertion, shock, strong emotion, sexual intercourse, exposure to cold, etc., are especially liable to produce pain.

Every physician of experience will easily recognize these tabetic lancinating pains from their localization and the above described severity, and will at once differentiate them from rheumatic or true neuralgic pain—and this is very important. They form an almost invariable symptom of the early stage, are present in about 90 per cent. of all the cases, and in the majority

(about 70 per cent.) form the *first* symptom; sometimes these pains exist for years and even decades without any other symptoms.

Paresthesia, which is rarely absent, follows the pain in varying forms and localization. There is formication, numbness, or a furry feeling in the soles of the feet and the legs which makes the patient feel as if he were walking upon carpet, wool, inflated rubber, or the like; also hot or cold sensations, and a feeling of tension around the joints of the foot and knee as if these were encased in apparatus, etc. Sometimes their localization is very peculiar: They appear in the region of the peroneus nerve, upon the anterior surface of the thigh (the lateral cutaneous femoral nerve), are especially typical and distressing around the anus, in the perineum, in the scrotum, or in the vulva, and are here referable to the lower segments of the spinal cord; the path of the ulnar nerve in the forearm and hand is especially liable to these sensations, and this has led to the term "ulnar nerve sensation"; they have also been occasionally observed in the face along the course of the fifth nerve (a feeling of numbness, etc.).

Perhaps those paresthesias are most characteristic which occur upon the trunk as the familiar *girdle sensation*: That peculiar paresthesia which, like a broad zone or girdle, encircles the trunk at various heights (the thorax, epigastrium, umbilical region, hypogastrium), and manifests itself as formication, or, more frequently, as a disagreeable sense of tension or constriction as though the body were in armor, which in some patients becomes almost unbearable. As stated, it is occasionally felt around the joints of the knee and the foot, and also in the calves.

Some patients have reported an increased sensitiveness to cold water and the like, that they suffer from *hyperesthesia of the skin,* usually upon the trunk, and particularly on changes of temperature; that in the dark and especially when ascending stairs their *gait is uncertain,* and that early in the morning when washing the face they readily lose their balance and fall, etc.—the subjective appearance of Romberg's symptom.

Disturbance of the ocular muscles, which becomes apparent to the patient as indistinct vision, double vision, or ocular vertigo, is an early accompaniment of the disease, frequently appears long before Romberg's symptom as a single premonitory sign, and probably indicates a preceding localized syphilis. These are pareses and paralyses of the muscles of the eye which usually implicate the oculomotor and abducens nerves, more rarely the trochlear; they are frequently transitory and fleeting, continuing in varying intensity for a few days, or weeks, or even months, and then disappearing, or they may be permanent.

Some patients report that *one or the other pupil is dilated,* that reading is prevented by disturbances of accommodation, and the like; but these conditions must be accurately determined by an objective examination.

In a certain proportion of the cases (10 to 15 per cent.) there is a slow or rapid but progressive *decrease in the acuity of vision*; a dimness and narrowing of the field of vision, and a diminished color sense; usually this increases rapidly and continuously, and leads to permanent blindness; occasionally, however, there are long periods of arrest, even of improvement in the condition, or it is limited to one eye, and may not attack the other for years. This symptom frequently precedes the appearance of the other tabetic phenomena, perhaps for 10 years.

Following this are subjective signs in the legs—*fatigue, loss of activity in walking and decreased endurance in standing*—a slight feeling of uncertainty, especially in the dark, which at first is unnoticed; usually these symptoms are referable to the sensory sphere, perhaps to paresthesia of the sensory nerves of the muscle; but unquestionably there is often a decrease in motor activity, until at last ataxia distinctly but gradually develops.

Among the most common phenomena of the first stage (appearing in about 80 per cent. of the cases) are disturbances of the urinary and sexual functions, usually paretic conditions, rarely irritative phenomena.

In the bladder these are manifested chiefly as dysuria, as hyperesthesia at the neck of the organ, sometimes also as neuralgic pain, more frequently as paresis. At first there is a tendency to *retention*, to a slow discharge, or to dribbling, very rarely to rapid micturition—or there may be a moderate degree of *incontinence*, the frequent necessity of immediate relief, sometimes in the clothes, or in the bed. This disturbance is usually slight, rarely so marked that the patient is obliged to use the catheter or to carry a urinal, but this occasionally happens, or is persistently the case, and cystitis soon develops.

Analogous functional disorders of the rectum are more rare; the most frequent complaint of the patients is that, on account of hypesthesia of the anal region, they are unaware of an approaching evacuation, and therefore, especially if there is diarrhea or purgatives have been taken, the discharges are apt to be involuntary.

There are almost invariably *disturbances of the sexual functions* even in the first stage of tabes. Irritative phenomena do not often appear, but the chief symptom is *sexual weakness* which shows itself by a decrease of sexual power, incomplete erection, premature ejaculation, a feeling of fatigue and weakness as well as pain *post coitum*; finally there may be pollutions and spermatorrhea without erection, etc. Libido may be undiminished. The condition terminates in *complete impotence*, but this may occur very late; I know tabetics during the course of whose disease the sexual functions were more or less unimpaired for years, yet—often to their detriment—they practised intercourse more frequently than was advisable, and raised a number of children.

In rare cases there is *increased sexual excitability*, even satyriasis, at the onset of the affection; this is seldom accompanied by increased potency, but usually with all the signs of beginning sexual debility which is likely to become more marked.

We rarely hear of these conditions in tabetic women; as a rule there is a decrease of sexual desire and sensations of lust during coitus; but a tabetic prostitute once admitted to me that during the first stage of her disease there was increased libido sexualis. Menstruation, pregnancy, and the puerperal period are not affected by tabes dorsalis.

In contrast with the principal *subjective* symptoms of the first stage are certain *objective* symptoms which are usually noted early, and permit the certain diagnosis of tabes; the first four to be mentioned are the most invariable and important.

First we observe *reflex rigidity of the pupil* (Erb) with or without *myosis* (the so-called *Argyll-Robertson symptom*). This is one of the earliest and most common symptoms of tabes, and has been the subject of innumerable

investigations. It occurs in at least 80 to 90 per cent. of all cases, appears very early, sometimes in advance of any other symptoms, and, as a rule, persists after it has once appeared.

The pupil, while retaining its power of sight, contracts neither directly nor indirectly to light, therefore is *rigid to light,* while in convergence and accommodation for near objects it distinctly contracts. Occasionally there is no contraction, and we then speak of total pupillary rigidity, which is not so important as the pure reflex form. The tabetic pupil shows no reaction to light either upon direct or indirect stimulation. The recognition of the condition is not difficult, but it presupposes accurate investigation and some practice. Accommodation must be excluded, and the pupil must be examined with a sufficiently powerful light (daylight or lamplight, with or without a reflector); the corneal reflex, the extreme narrowness [pin-point] of the pupils, and a dark color of the iris, may cause some difficulty, but this may be overcome. Sometimes reflex rigidity is unilateral; frequently it reveals itself only by sluggishness and a very slight reaction, which, however, in youthful persons is of great significance. (The very rare paradoxical reaction of the pupil —i. e, apparent dilatation of the pupil to light—is of no practical importance, and probably must be referred to processes of accommodation.)

Reflex rigidity of the pupil is almost always combined with an *abnormal size,* often with more or less extreme contraction, of the pupil—*myosis.* The pupils may then be reduced to ½ or one mm. in diameter, and the so-called "stabbing glance" is produced. To this smallness of the pupils the term "spinal myosis" has been given. It is the most common pupillary anomaly, and in spite of a narrowness of the pupil which is often astonishing it contracts further upon accommodation. Myosis and reflex rigidity, however, do not necessarily accompany each other; they are substantive symptoms which appear at different times, and each may exist alone; usually reflex rigidity is first noted, more rarely it is combined with myosis. Occasionally, however, there is unilateral or bilateral *mydriasis,* particularly when total pupillary rigidity is present. The difference of the pupils in tabes is of much less significance than in paralysis. Occasionally the tabetic pupil is changed in form, it may be distorted, elliptical, or the like, but this is unimportant. As I was the first to maintain, in the pupil which is rigid to light there is a permanent absence of the *sensory pupillary reflex,* i. e., of *dilatation* of the pupil to sensory cutaneous irritation; therefore the pupil is *reflexly rigid* in every sense. We shall refer later to the exceedingly abstruse question of the accurate localization and pathogenesis of these pupillary phenomena.

The second objective symptom of paramount importance is the *absence of the tendon reflexes,* particularly of the *patellà tendon reflex* (Westphal's symptom), that is, the utter impossibility of producing it (even by means of the Jendrássik method), as may readily be done in normal persons provided, of course, there is no disturbance of motility, no atrophy of the quadriceps, no peripheral nerve disturbance, etc. This also is one of the earliest and most common symptoms, appearing in 90–95 per cent. of the cases, and is of the greatest diagnostic import; it may be unilateral or bilateral, usually appearing merely as a unilateral decrease and then totally disappearing; or it may occur late, in well developed tabes may be absent for a long time, even for years, and in rare cases it may be retained. I need scarcely describe the tests

for this condition, but they are best made with the patient in either the recumbent or sitting posture so that the knee is flexed almost at a right angle, with a complete relaxation of the quadriceps; a practised observer will rarely encounter any difficulty. But this test is frequently perplexing in non-tabetics, in whom it is sometimes impossible at once to evoke the patella tendon reflex (from inability voluntarily to relax the quadriceps, from too close attention, etc.); experience, however, enables us properly to appreciate even these cases. Recently our investigations have ceased to be limited to the patella tendon reflex, but now embrace all of the tendon reflexes, and valuable data concerning them have been obtained.

Above all in importance are the *tendon Achilles reflexes.* These are apt to disappear on one or both sides synchronously with the patella tendon reflex, *sometimes even earlier,* or they may remain unimpaired longer than this. The act that the tendon Achilles reflex may be evoked in almost all normal persons (Sarbó) is of special value in the early diagnosis of tabes. The test is best made with the patient in the lateral position, with the knee flexed, the ankle-joint flaccid, and the sole lightly supported by one hand; we then tap with the percussion hammer at various points upon the Achilles tendon from the calcaneus upward (usually but one point is active); finally, we try Jendrássik's method. The reflex is also well developed while the patient kneels upon a chair, supporting himself with his hands. According to our present knowledge, *the absence of the Achilles tendon reflex* (especially its *unilateral* absence) is just as important a symptom as that of the patella tendon reflex.

In the *upper extremities* also the tendon reflexes may be decreased or absent in tabes, but the tendon and periosteal reflexes in the wrist-joint and the *triceps tendon reflex* are of little importance. Frenkel maintains that the absence of this reflex is just as important, perhaps occurs earlier, and is even more invariable than the absence of the patella tendon reflex, but this is certainly incorrect. The reflex in normal persons is by no means constant, and is absent in only a few tabetics during the first stage. The triceps reflex, which originates in the upper thoracic and cervical segments, is absent only in so-called tabes superior, and its unilateral absence is, under some circumstances, of great significance and exceedingly valuable.

The third and most familiar objective symptom is *swaying upon standing with closed eyes,* the so-called Romberg's symptom; it is exceedingly common, occurring in over 90 per cent. of the cases, is significant, and easily evoked and recognized. The patient should stand still, best with his feet in juxtaposition (for the final test the patient may stand upon one foot), and is then told to close his eyes, whereupon we observe a more or less distinct swaying, a sort of pendulous movement about the axis of the body, which increases and, if extreme, makes the patient feel in danger of falling, or he actually falls. It is undoubtedly to be attributed to a disturbance of the so-called muscular sense, that complicated centripetal sensation originating in the skin, the joints, the tendons, and especially the muscles, which is essential for maintaining position in space, for preserving the equilibrium of the body, and for the erect position of the trunk, and depends upon the uncertainty produced by complicated muscular action while standing quiet; in tabes it usually appears early, long before actual ataxia. It is the first expression of those disturbances of the muscular sense which implicate the sensation of posture

of the members, sensations as to bulk, and the direction of movements produced by them, as well as of the passive movements arising therefrom, conditions to which I will revert in discussing disturbances of sensibility.

The disturbances of sensation in the skin and in the muscles—form a fourth important group of symptoms in the first stage, embracing many individual phenomena which, from their nature, peculiarity, and localization, are characteristic and of great value. In the last 25 years our knowledge of these has been greatly amplified.

Beginning with the *lower extremity*, sensation may be tested by the simplest methods, but it should be carefully done and frequently repeated; it usually requires much time, and is exceedingly tiresome to both the investigator and the patient. (Tactile sensation may be sufficiently tested by the brush or by slight contact with the finger, with any smooth or rough substance, or we may use the head or the point of a pin; for the sensation of pain the prick of a pin, sharp pinching of folds of the skin, or the faradic current with Erb's electrode may be used; the temperature sense may be tested by contact with test-tubes filled with hot or cold water, or with glass or stone paper-weights, etc.; for sensations of space any well known method may be employed.)

It will usually be apparent that, in the first stage, the *tactile sense* has suffered but little or not at all; sensation is slightly decreased in the sole of the foot and in the toes, at the sides of the foot, on the anterior surface of the leg, and occasionally up to the knee and thigh. Sometimes in the late phases of the disease we observe a decided diminution or complete loss of the tactile sense which extends to the trunk, perhaps to the upper extremity and the head.

Disturbance of the *pain sense* is almost invariable and of more importance; in the stage of onset we frequently note *hypalgesia* which sometimes amounts to *complete analgesia*; a fold of the skin may be pierced by a coarse needle, yet the patient be quite unaware of it. Folds of skin upon the dorsum of the foot or in other areas may be pinched hard (which is exceedingly painful to normal persons), yet the patient is conscious of nothing more than contact; at all events there is no pain. Not rarely this hypalgesia is confined solely to areas of the lower extremities and the trunk. Combined with this, or existing independently of it, there is *a delay in the perception of pain*, which amounts to $\frac{1}{4}$ or to $\frac{1}{2}$ of a second, or to 2 seconds or more, and is followed by an acute sensation of pain, sometimes by an *actively increased after-sensation*. In the cases in which pain sense is decidedly retarded but not actually decreased, upon painful stimulation of these parts there is a so-called double sensation: First, that of contact, then of pain.

The conditions in regard to *farado-cutaneous sensibility* are somewhat different; this is usually analogous to the pain sense; i. e., it is decreased below the minimum for sensation, as well as for pain; even powerful currents no longer cause pain (see Erb "Electro-Therapeutics"); feeble currents analogous to tactile sensation are only rarely perceptible, and strong currents produce no pain. This method may, therefore, be utilized for accurate tests, and for comparing the results of other tests.

The *temperature sense* upon the whole corresponds to the sensation of pain; not rarely it varies somewhat for heat and cold.

Except for lancinating pain in the leg *hyperesthesia* is rare; occasionally, however, there is a moderate hyperesthesia to cold, similar to that observed in the trunk. Here and there a so-called *polyesthesia* is observed; i. e., a variety of sensations upon mere contact, also an *allochiria* in which the sensation is referred to the wrong side; these are of no practical significance.

Quite analogous conditions are found in the *upper extremity* in later stages or in tabes superior: Mild or extreme hypesthesia, hypalgesia and analgesia, thermal hypesthesia, etc., these are usually limited to the ulnar nerve in the forearm and the inner surface of the upper arm (the median cutaneous branchial, etc.), and thus indicate the involvement of the upper dorsal segments and root area (eighth cervical and first dorsal roots).

Actual *astereognosis* is rarely noted in tabes; in the later stages it appears in the hands, especially if muscular sensation is greatly disturbed, often to an extreme degree and in striking contrast to the tactile sense, which is still fairly well retained. On the other hand the frequently demonstrable *analgesia of the ulnar nerve*—absence of sensation upon firm pressure on this nerve above the the elbow-joint, this in normal persons usually producing acute pain—is worthy of mention. A similar condition is observed in the *peroneal nerve* behind the head of the fibula.

Analgesia of the testicle, which has lately been emphasized, is quite analogous. In contrast to the normal conditions, firm pressure upon the testicle produces no pain. In women there is *mammary analgesia.*

Especially worthy of note, and of equal practical and theoretic importance, is the degree of sensibility in the *trunk.* Hitzig, subsequently Laehr, Marinesco, Déjérine, and others demonstrated that, especially in the earlier stages of tabes, there are girdle-like zones of hyperesthesia and anesthesia upon the trunk, and from their localization these conform to the "root type" —that is, the area of distribution of the various higher dorsal posterior roots corresponding to the segments of the spinal cord to which they belong—and therefore permit the recognition of a decided "root predisposition." These zones, as broad as the hand or broader, encircle the thorax like a girdle either completely or incompletely, beginning at various isolated areas—at the scapula, the axilla, the cardiac or gastric region—and gradually forming a girdle which is often unequally developed upon the two sides of the body, being higher on one side than on the other; or two or three such girdle zones may be situated one above the other, and be separated by cutaneous areas in which sensation is good. They may readily be recognized by tactile and pain tests because in this form they rarely occur in any other disease, and they demonstrate most convincingly the root and segmental origin of these tabetic anesthesias. Since these have been minutely studied, we know that the same conditions are found in the extremities. In this region, however, they are not always so distinct, because the various root innervation areas are situated one above the other, and are frequently not equally implicated in the disturbance. To this category belong the paresthesias, above mentioned, which are localized in the buttocks, at the anus, in the perineum, and in the genitalia, these being the root areas of the conus terminalis.

Another valuable and common sign which appears upon the trunk of tabetics, and to which I have lately attached decided significance, is a conspicuous *hyperesthesia to cold* which is noted in the trunk, the abdomen, and the back,

and may reach an astonishing degree. The patients recoil on coming into contact with any cold substance, often crying out aloud, and making strong resistance, declaring that the sensation of cold is extremely unpleasant and even painful to them. They notice that they no longer bear cold water, are no longer able to take a cold bath, etc.; it is true there are some healthy persons to whom cold applied to the abdomen and the sides is more or less unpleasant, but it never reaches the extreme degree observed daily in tabetics. *Hyperesthesia for heat* is more rare and much less conspicuous, but, even when present, it is not so marked as hyperesthesia for cold.

These are the disturbances of cutaneous sensibility which are apt to be found in the first stage of tabes,—more or less well developed, with variations and degrees in nature, intensity, and distribution. With the progress of the affection all these signs increase, and a high-graded anesthesia or analgesia may be distributed over the entire body,—even up to the face and the top of the skull.

Passing to the *disturbances of deeper sensibility* (inaccurately designated *muscular sensibility*) we find the *Romberg sign* which has already been discussed. It is usually most distinct in the earlier stages, and other symptoms are gradually added which, as a rule, can only be determined by minute and painstaking investigation. We note primarily that the patients have *more or less completely lost the sensation of posture and position of their limbs, especially of the legs*; with closed eyes or in the dark, they do not know where their legs are; if in the recumbent posture, whether they are hanging out of bed or not; if one leg is passively placed in a certain position, they are unable to bring the other leg into exactly the same position, which normal persons can do with great precision; furthermore, they lose all *control of the measure and direction of movements which they are asked to perform*, provided their eyes are closed or it is dark. The movements then are aimless, excessive, shooting far beyond the mark, apparently because the patients have no clear idea of the direction of the movement to be made.

The *sensation for passive movements of the limbs* is also more or less completely lost in such patients. Even energetic passive movements of the various joints are either recognized imperfectly or not at all, and when their eyes are closed these patients are absolutely ignorant of the position in which their limbs have been placed. This test should always be made in the lower extremities, but is even applicable to the upper extremity which may show similar changes. In the small joints of the finger it has led to very interesting theoretical conclusions which, however, are practically of little value.

Finally, we may mention the absence of the *sensation of fatigue* which tabetics report (for example, the patients can hold their arms for a comparatively long time in one position without feeling tired). The latest observation in this realm is the following: *The arrest of sensation in the bones*, determined by testing with a tuning-fork the *sensation of vibration* which appears in the bones (Egger). Besides these invariable and important objective symptoms of the first stage, investigation frequently reveals other conditions, some of which will be briefly mentioned.

Chief of these are the *cutaneous reflexes*; they deviate but little from the norm, and only when there is marked disturbance of the tactile sense are they at all parallel with it. The *plantar reflexes* are sometimes retained and

as active as under normal circumstances, or they are diminished, or are absent; the less the tactile sensation in the sole of the foot, the more the plantar reflex is decreased; with retarded pain conduction a prick in the sole of the foot is sometimes followed by a delayed reflex. The *cremaster reflex* is evidently longer retained than the reflex of the sole of the foot. On the other hand, the *abdominal reflexes* (as is well known there are at least three of these at various heights of the abdomen) are usually present and *conspicuously active,* no matter what the cutaneous sensibility of the abdomen; with a very thick belly, a profuse layer of fat, or with very flaccid and atonic abdominal walls, they are of course absent, as in normal persons.

In tabetic *paralysis of the muscles of the eye* the findings are more significant, and are frequently referable to the oculomotorius or some of its branches or to the abducens, much more rarely to the trochlear nerve; they manifest themselves by the familiar disturbances of binocular sight, double vision, vertigo, paralytic strabismus, etc., and are readily determined by ophthalmological tests. The manner of their appearance varies; sometimes there is a paresis, at other times a profound paralysis of one muscle, or several, or all of them, unilaterally or bilaterally, with or without ptosis. They are usually transitory, lasting for a few days or weeks, or at most months. They generally appear very early, as the first symptom, or in the first stage of the disease. Subsequently relapses may be frequent and the condition may become permanent. They usually indicate nuclear changes, while primary lesions in the peripheral nerves of the muscles of the eye are rarer. The associated paralyses of the muscles of the eye, which appear in the later stages as more or less complete external ophthalmoplegias, are of serious import and indicate nuclear atrophy. When they occur in persons of middle age with a history of preceding syphilis but without accompanying symptoms, they awaken our suspicions of beginning tabes; this is true of the much rarer, usually unilateral, ophthalmoplegia interna accompanied by mydriasis, general pupillary rigidity, and paralysis of accommodation.

Of most serious import for the patient is *progressive tabetic atrophy of the optic nerve* which fortunately occurs only in a small proportion of the cases—about 10 to 15 per cent. In two-thirds of all cases this severe complication appears in the pre-ataxic stage. When patients complain of feebleness of vision, or veiling of the field of vision, etc., the physician finds a distinct, and, as a rule, rapidly progressive *decrease in the acuity of vision,* soon combined with a *loss of color sense* (first red-green, then yellow-blue) up to complete *color blindness* [Daltonism]; simultaneously there is a *limitation of the field of vision* beginning peripherally, and often progressing in a typical manner. More or less rapidly beginning *amblyopia* changes to complete *amaurosis,* first in one eye, sooner or later but invariably also in the other eye. It is characteristic that the *pupils* are frequently narrowed and rigid, instead of being widely distended as in other cases of amaurosis.

The ophthalmoscope shows the cause of these disturbances to be the so-called *white atrophy of the optic nerve.* At first we notice a grayish, subsequently a white, glistening appearance of the sharply defined papilla, with contracted arteries and veins, yet no change in the retina, a picture which is readily differentiated from the choked disc of simple neuritis and of optic neuro-retinitis, and from the temporal pallor of the papilla in multiple sclerosis.

It is noteworthy that patients with optic atrophy which appears early often present a mild form of tabes, and the former seems to have an inhibitive effect upon the development of the latter. Such patients have comparatively few symptoms—little pain, and but slight bladder disturbance; if ataxia develops it is late. This remarkable and inexplicable circumstance has been noticed by many observers as well as by myself.

Motion still remains to be investigated; in the first stage, provided there are no rare symptoms (neuritis, atrophy, arthropathies, etc.), it is, as a rule, but slightly disturbed; *coarse* power is apparently well retained, and may be considerable; the exactness and certainty of movements, such as marching, leave nothing to be desired. On the other hand, we note in the motor activity of the patients an early but distinct general loss of the power of sustained movements. They soon tire, can no longer walk much, cannot stand so long as formerly while at work, and, in a fair proportion of these cases, in the first stage a certain, distinct, muscular weakness gradually develops. The familiar description of an abrupt "giving way of the legs" must be regarded either as a sudden symptom of exhaustion or as the effect of a fleeting, momentary, lancinating pain. The rare pareses and paralyses which are limited to individual nerve and muscle regions will be described later.

In this stage there are no quantitative or qualitative *changes in the electric contractility of the motor nerves* and muscles which can be pratically utilized.

But, even in this stage of tabes, if the muscles are powerfully developed, the *easy movability and flaccidity* of the members is very conspicuous. ***Muscular tension*** is ***never seen*** in genuine tabes. Passive movements of unusual extent may be performed with the greatest ease, which shows that the tonus of the muscles has decidedly decreased; this usually becomes noticeable in the second stage from the fact that the legs of such patients can readily be moved in any direction. The extended leg may without difficulty be flexed so far at the hip-joint that the thigh almost touches the abdomen, the foot reaches the head, etc. This phenomenon, which has long been observed, which was mentioned by Leyden, myself, and others, and which Frenkel thoroughly studied, has lately been appropriately designated by him as "*hypotonia* of the muscles."

It consists in a decrease, or the entire *absence, of inhibition for certain active and passive movements,* and is *due to abnormal flaccidity of the muscles,* without any change in the joints, ligaments, or tendons. These patients can extend the leg at an angle of 90° or even more from the hip-joint, whereas the normal person can scarcely reach more than 50° to 60°; with extended knee pressed upon the underlying surface of a couch the heel can be raised some distance from this surface (hyper-extension at the knee-joint); in standing genu recurvatum is conspicuous, and the same abnormal movability is apparent in almost all of the joints and muscle groups (the leg and foot, the adductors and rotators of the thigh, the vertebral column, the muscles of the trunk, the joints of the hand and fingers, etc.), as was so clearly shown by Frenkel. Therefore, tabetics are able readily to execute movements which are impossible for normal persons unless they are professional dancers, clowns, or "contortionists."

When the muscles are powerfully developed and firm, this hypotonia is usually invariable in the first stage, and sometimes appear very early; it

by no means runs parallel with sensory disturbances nor those of the reflexes; it exists for a long time without ataxia; the latter is certainly not the consequence of hypotonia, but the hypotonia greatly influences the posture of the body (genu recurvatum, forward flexion, and laxity of the trunk, etc.), therefore also the ataxia. Frenkel includes it among the pathognomonic and initial symptoms of tabes. It is probably due to a decrease of the reflex muscle tonus conveyed by special reflex tracts.

In the first stage of tabes, therefore, the *constitutional condition* of the patients appears to be entirely undisturbed, their vegetative functions are normal, the *vertebral column* shows no change, the brain functions normally and intellection is unimpaired, provided no complications are present.

Numerous symptoms, however, which appear in almost all cases in addition to these constant and typical ones, we shall not at present touch upon, but shall first consider the *most important motor symptom* of tabes—the ataxia. This introduces the *second stage* of the disease.

In the later course of the first stage, usually after the passage of years or decades (up to 25 years or more), but sometimes after only a short duration of the affection—3 to 6 months—new motor disturbances appear: There is a want of certainty and precision in the movements of the limbs, the patients become awkward, or combined and complicated movements are performed with increasing difficulty and are less exact; in a word, *tabetic disturbance of coordination—ataxia*—appears, and the patient enters the "ataxic stage" of the affection. Sometimes, however, after a more or less prolonged prodromal stage, ataxia may develop within a few days or it may suddenly appear during the night.

It is characteristic of tabes and one of its main features that the precision of combined and finer movements is decidedly decreased, while the motor power and the strength and certainty of individual movements have suffered little if at all. Therefore, *it is not a paralysis*, but a disturbance of the *coördination of movement*. Even if we occasionally demonstrate a decrease of motor power, or a distinct paresis, the disturbance of movement due to the ataxia is in no proportion to it.

Almost invariably ataxia begins in the *lower extremities*, and here it is typical and uniform. In its *mildest degree* there is some uncertainty and irregularity in walking, as shown by unequal steps and an uncertain position of the feet; in more difficult movements, such as rapid turning, there is slight swaying; running and jumping, the ascent or descent of stairs, stepping upon a foot-stool, walking on a smooth floor or on a line, become more difficult. This is most apparent in the dark or on closure of the eyes. On standing with the feet in juxtaposition, we note swaying, and contractions in the anterior muscles of the leg; standing upon one foot is uncertain, or even impossible. In the recumbent posture there may be no evidence of motor changes.

These mild grades are readily diagnosticated by minute investigation; the patient should be directed to sit down slowly and then to rise, to flex the knee, to stand up quickly upon command and begin to walk, to halt suddenly while walking, to walk in a straight line, to stand upon a chair, to turn rapidly, to stand with the feet close together or upon one foot, to ascend or descend stairs without the aid of the banister, finally, to walk backward; in all of these movements there is considerable uncertainty and swaying, especially if the patient

performs them with the eyes closed, and thus has no assistance from the sense of sight.

Gradually the disturbance becomes *more marked,* the tip of the foot is projected forward and outward, the heel stamps upon the floor, the knees are stiff and hyperextended, the body is bent forward, the eyes are riveted on the feet and the floor; the steps become unequal and jerky, the route in walking more and more irregular, finally, the gait becomes uncertain, aimless, and waddling. Thus is developed the characteristic *ataxic gait* which enables us to recognize the tabetic at some distance on the street, and which differs so markedly from the spastic and paralytic gaits. In standing the disturbance becomes more pronounced, especially when the feet are together, the patients oscillating to and fro. Spasmodic movements are noted in the legs and trunk, and the patients are unable to remain quietly in one position—a condition which has been designated *static ataxia,* in contrast to the "locomotor form," and which also apppears when the patient is sitting still, or when he quietly extends the legs or the arms.

An examination with the patient in the recumbent posture also reveals ataxia: Slow and uniform movements are impossible, or they are performed paroxysmally or interruptedly; even the mere raising of the leg is done in an irregular, zigzag way, the raised leg falls at a spot more or less remote from the place intended; a given object can be touched with the great toe only after many irregular, secondary movements; instead of describing a circle with the tip of the foot the figure becomes angular, and often irregular; touching the knee with the heel of the opposite foot—"the knee-heel test"—is only possible by means of awkward contractions which often cause the foot to shoot far beyond the mark; the heel can be slowly moved along the tibia of the other leg only with the utmost difficulty. Here it is evident that the ataxia, especially the irregularity of movement, becomes more distinct when these maneuvers are performed with the eyes closed, but only when noteworthy disturbances of the so-called muscular sense are simultaneously present; if these are absent, the closure of the eyes has but little effect upon the ataxia, and it is also evident that, if well marked, the ataxia is *not prevented* by the careful use of the eyes but at most is merely decreased.

In the most *extreme grades* of ataxia, walking and standing finally become impossible; the legs lose their hold, are projected in all directions, if left to himself the patient falls, and even if supported upon both sides or carried the legs wabble about wildly. Spasmodic and paroxysmal movements invariably appear, and the patient can no longer assume the position necessary for standing or walking. Yet coarse power may be quite well retained, although the patients may have become entirely helpless, and are confined to their beds or to a rolling chair.[1]

Ataxia usually appears much later in the *upper extremities,* only the cases of tabes superior being exceptions to this rule. Ataxia, of course, interferes with all of the finer movements for which the hands are constantly used, such as buttoning the coat, tying a cravat, embroidering, cutting, writing, draw-

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[1] For other details see the works of Frenkel ("Die Behandlung der tabischen Ataxie," Leipzig, 1900) and Otfried Förster ("Psysiologie und Pathologie der Coördination," Jena, 1902); these authors have made careful and exhaustive investigations of this subject.

ing, piano-playing, etc. The movements become uncertain, spasmodic, awkward, zigzag-like, etc., particularly if they are performed without the aid of the eyes (buttoning the suspenders at the back!). Ataxia becomes distinctly evident when the patient tries to touch with his finger the tip of his nose or an object held in front of him, or if the fingers of both hands, being some distance apart, are gradually approximated at their tips ("index finger test"); when they attempt to grasp an object held in front of them, the fingers are drawn apart and awkward grabbing is noted, as well as when they try to describe figures in the air with the finger, such as circles, numerals, etc., and in writing and piano-playing—all movements are irregular, aimless, jerky, and angular!

Finally, the hands become useless for the purposes of daily life; the patients can no longer eat or dress without assistance, can hold nothing in the hands, and, in fact, are quite helpless.

Ataxia may also appear in the *muscles of the trunk*: The harmonious action of the muscles necessary to maintain the equilibrium in standing, sitting, and in many movements, becomes disturbed, and the bodily movements are irregular or wabbly. The same is also true of the muscles of the neck and the nape.

In rare cases even the *muscles of the face*, of the *lips*, and of the *tongue* are not exempt: This is shown by certain grimaces, especially in laughing and speaking, in the impairment of the powers of mimicry, of speech, and of mastication. *Ataxic nystagmus*, however, appears to be a manifestation of Friedreich's ataxia only.

Ataxic movements have frequently been observed in the *muscles of the vocal cords*.

It is of the utmost importance that these ataxic disturbances of movement be differentiated from all related conditions. This, as a rule, is easy, difficulties arising only in rare cases when similar disturbances of movement appear in other diseases. But the mere differentiation of tabes from the other symptoms, usually no arduous task, will preserve us from error.

Above all, it must be borne in mind that ataxia is a *disturbance of voluntary movements*, and does not appear during rest; this alone distinguishes it from chorea minor, from paralysis agitans, and the other forms of tremor; the only disturbance of movement with which it may occasionally be confounded is the *intention tremor of multiple sclerosis*, which also appears on voluntary movement. In this condition, however, there are regular oscillations around the axis of movement; in ataxia there are irregular movements, zigzags and angles in walking, which by no means resemble tremor. Moreover, the accompanying symptoms of these affections are absolutely decisive. But it should not be forgotten that true ataxia may occasionally occur in multiple sclerosis, and even be combined with intention tremor!

The ataxia of *Friedreich's disease* ("hereditary ataxia") is almost identical with that of tabes. Naturally, the experienced observer will detect some differences, but these are not marked (possibly more waddling and uncertainty in walking, a marked and early implication of the arms, distinct disturbance of speech, ataxic nystagmus, marked "static" ataxia, etc.). The disease should be clearly recognized from its other symptoms: Its appearance in childhood, in several children of the same family, the absence

of sensory disturbances and pain, normal state of the pupils, the absence of syphilis, etc.

The differentiation of *cerebellar ataxia* from spinal tabetic ataxia is much more simple; the gait in the former is unlike that of the latter, being of a more waddling character, resembling that of a person under the influence of alcohol, and the individual movements are quite different in character, being coarser, more complete, and less zigzag in appearance. The arms rarely show any sign of this, and, instead of the typical symptoms of tabes, we note those of disease of the cerebellum.

This exhausts the description of the second stage of tabes, ataxia being its only differential and essential symptom.

Investigators have busied themselves with the pathogenesis of this symptom, and in trying to explain its occurrence have referred it to pathologico-anatomical changes intimately connected with the preceding and accompanying sensory disturbances; but, unfortunately, from the earliest times to the present with most unsatisfactory results. We shall revert to this when describing the anatomical changes in tabes.

Not much more can be stated in regard to the second stage. The symptoms of the first stage continue, and some of them become more distinct and intensified, as, for example, the sensory and bladder disturbances and the pain; not infrequently this stage is complemented by the addition of other symptoms.

We now come to the discussion of some *rare and unusual symptoms of tabes,* no less interesting and remarkable, which complete the typical picture of the two preceding stages, rendering it extraordinarily rich in types and varieties.

These phenomena are almost innumerable, but they all belong to tabes; volumes might be filled with their enumeration, exact description, and analysis. We shall attempt a brief outline, arranging them according to their frequency and importance. They may appear in any stage of tabes—early or late—and, for the sake of simplicity, these stages will be discussed together.

Visceral Crises.—I must first mention the peculiar phenomenon to which the term *visceral crises* has been attached. Those longest known and most familiar are the so-called *gastric crises* which were mentioned by Gull (1858) and Delamare (1866), and introduced into the pathology of tabes by Charcot's classical description (1868).

These consist of sudden attacks of *severe "gastric pain,"* occurring at various intervals, radiating into the epigastrium, the abdomen, the lateral aspect of the body, and the back; the pain is intense, it is girdle-like, boring, screwing, or contracting in character, and is accompanied by moans and cries, general prostration, and a feeling of extreme illness which makes the patient utterly wretched. *Uncontrollable vomiting* soon follows, first of food, then of mucus and gastric juice, rarely of blood. The patient is unable to take the slightest amount of food because it is at once vomited; there is unquenchable thirst—briefly the picture of intense suffering. This may last many hours, for several days, sometimes even for two or three weeks, new attacks constantly appearing, and only gradually disappearing. Vomiting is not invariable. It may be slight or absent, and the attack may be limited to the pains, the general prostration, and the disturbed ingestion of food. Owing to the pain, the vomiting, inability to retain food, and the insomnia, the patients lose flesh and become greatly debilitated.

The attack ceases more or less suddenly, the patient feels relieved, is able to eat and drink and to retain food, and he recuperates in the shortest time and rapidly increases in weight.

During the intervals between the attacks the stomach, as a rule, is absolutely normal, but there may be slight dyspeptic symptoms—a mild gastric catarrh—such as the patient has had before the attack.

The examination of the vomitus and the gastric juice during the attack shows varying conditions; in some cases marked hyperchlorhydria and hypersecretion, in other hypochlorhydria, even complete anacidity. These are apparently of little importance, and the remaining investigation reveals nothing essential. The epigastrium and abdomen are markedly contracted and more or less sensitive to pressure; there is no splashing sound, no tumor, nothing to indicate an ordinary gastric affection.

The intervals are of varying length, weeks, months, or years, but in the individual case the attacks usually show a certain periodicity, and increasing frequency; mild attacks may alternate with severe ones; they are easily recognized, and with some attention may readily be differentiated from true gastric affections. Formerly these attacks were always regarded (as they are sometimes to-day) as "gastric spasm," ulcer, or gall-stone colic, and were so treated until they reached the neurologists. Now they are treated in sanatoria for gastric diseases at Carlsbad, Kissingen, Homburg, and other spas, and, as a rule, correctly; the examination of the pupils, of the tendon reflexes, etc., for tabes renders the diagnosis positive.

These "gastric crises" appear in many cases of tabes, and form one of the most severe symptoms of the affection. They may occur at any stage of the disease, sometimes at the onset of the affection. Their explanation is still uncertain, their point of origin unknown, and the view that they are analogous to severe attacks of lancinating pain is probably correct; but this analogy extends no further. As slight, rudimentary forms we may note pyrosis, salivation, burning in the esophagus, singultus, irritation of the throat, causeless nausea, and the like.

Similar to gastric crises are *intestinal crises.* These consist of attacks of severe colicky pains with copious and stubborn diarrhea; either symptom may, however, occur alone, and neither in itself is characteristic; in the intervals, the intestinal functions are absolutely normal. Intestinal crises are, upon the whole, much more rare than gastric crises.

Analogous to these are so-called *rectal crises,* which come on with severe pain, a burning sensation as of a hot plug in the rectum, combined with tormenting tenesmus, and with muco-purulent discharges. This may be associated with similar symptoms on the part of the bladder—*vesical crises*—a condition which has frequently been described as *neuralgia anovesicalis,* an exceedingly painful strangury and tenesmus. We also frequently note *urethral crises* in which the pains are localized chiefly in the urethra, and are combined with tenesmus, severe and burning pain upon micturition, the pain radiating to the perineum, to the thighs, the hypogastrium, and the anus.

Attacks have been described as *renal crises* which may simulate renal colic; there is severe pain which is difficult to differentiate from colic due to calculi if there are no objective nor subjective phenomena of tabes.

Furthermore, we observe *testicular crises,* paroxysmal and severe attacks of

pain in one or the other testicle. In women so-called *clitorial crises* appear, either in the form of attacks of pain localized wholly in the vulva, the region of the clitoris, the urethra, or the bladder, and in characteristic sexual orgasms with painful sensations of lust, a feeling as of coitus with subsequent discharge, and simultaneous or subsequent prostration and exhaustion. These attacks may also occur at night in the form of "feminine pollutions" with sensations of pain.

In the organs of the throat and chest "crises" are quite common. The most common and important are the so-called *laryngeal crises*, first described by Féréol in 1868, which may be more or less severe. The onset is sudden, there is a feeling of tickling, a spasmodic contraction of the neck with severe dry cough, suffocation accompanied by stertorous respiration, unconsciousness, pallor, and cyanosis; sometimes an epileptic attack occurs, and may be dangerous to life. Usually, however, the spasm of the glottis ceases in a few minutes, consciousness returns, and the attack is relieved.

In mild cases there is a *spasmodic cough* which resembles whooping cough; the respiration is noisy, the cough is paroxysmal, and a tough foamy mucus is expectorated. This may recur with great frequency.

Especially severe attacks of tickling in the throat, arrest of respiration, unconsciousness, and epileptic attacks, have been described as *ictus laryngeus*. These laryngeal attacks are sometimes combined with a paralysis of the muscles of the larynx which will later be described.

Oppenheim has designated as *pharyngeal crises* painful sensations in the pharynx frequently combined with movements as in deglutition.

Tracheo-bronchial crises have also been mentioned. They are analogous to the laryngeal attacks, and are accompanied by spasmodic cough, and difficulty in respiration and deglutition.

Finally, *cardiac crises*—more or less severe attacks of angina pectoris which may be referred to a disturbance in the tract of the pneumogastric nerve—have been observed in tabes. Since the possibility of another cause, such as sclerosis of the coronary arteries, cannot be excluded in these patients, this view is not incontestable.

We now turn to another series of phenomena referable to the *motor apparatus*, and chief among these are the "*atrophies, paralyses*, and *atrophic paralyses*" in the various nerve and muscle regions of the body.

Déjérine lately called attention to a form of muscular atrophy which, although previously described, was never sufficiently appreciated. He believes that this forms an integral factor in about 20 per cent. of the cases of tabes; it is symmetrical, usually develops early and insidiously in the extremities, more markedly in the lower, but occasionally also in the trunk, and may finally reach an extreme degree. As a rule it appears in the late course of the disease, and is not rarely observed in the pre-ataxic stage. In the lower extremity it begins in the small muscles of the foot, where it produces weakness and paralysis, a claw-like position of the toes, a sort of "tabetic equinovarus," then it passes to the leg, especially to the peroneal region, then to the calf and to the thigh, finally some of the muscles become contracted, and a deformity of the feet becomes permanent.

In the *arms* this atrophy begins in the small muscles of the hand, produces the characteristic ape-hand or claw-hand, etc., and slowly involves the forearm

and upper arm. Similar conditions are occasionally noted in the trunk and in the muscles of the shoulder-blade.

The paralysis of the muscles is in exact proportion to the atrophy; the paralysis therefore does not precede. Fibrillary contractions are absent. Electric contractility is either decreased or absent; as a rule, there are no DeR. Finally we have the common picture of ataxics—people who are greatly emaciated, with wabbling legs and sometimes wabbling arms.

The nature and pathogenesis of these diffuse atrophies is still uncertain; they resemble poliomyelitic and other spinal amyotrophias less than they do mere wasting of the muscles, or the terminal stages of chronic, neuritic, muscular atrophies. The latter possibility is favored by the autopsy findings, which, in addition to degeneration of the muscles, chiefly show degenerative changes in the peripheral nerves, the anterior roots not being implicated.

Furthermore, there are many *local paralyses* and *atrophies* which may occur in any region of the body.

The more or less slight paralyses of the *muscles of the eye* which occur so frequently in the early stages have already been mentioned; but in all stages of the affection severe and permanent paralyses of varying nature and combination may be observed. There may be *complete paralysis of individual nerves,* of the oculomotor and abducens, either unilateral or bilateral, and with or without ptosis, with marked strabismus, with paralytic contracture, also *paralysis of associated muscles*: The lateral rotators, the raisers and depressors of the bulbus, of both internal muscles, etc., and this, which unquestionably indicates a *nuclear origin,* may develop into a more or less complete *ophthalmoplegia externa* in one or both eyes. The details cannot be described here.

Not rarely we note in addition an *ophthalmoplegia interna,* a complete paralysis of the muscles of the iris, usually with mydriasis and paralysis of the muscles of accommodation. These are usually persistent and incurable. I must reiterate that the variations in, and the possible combinations of, these symptoms are extraordinarily great.

It is remarkable that in tabes the facial nerve is scarcely ever attacked by paralysis, although it has been noted in rare cases (syphilitic facial paralysis?); usually it is localized in the muscles of the lips as a partial phenomenon of "bulbar paralysis."

More frequently, but still quite rarely, do the *muscles of mastication* become paretic and atrophic; this is usually only in combination with other bulbar forms of paralysis.

Paralyses of the muscles of the larynx (therefore of the vago-spinal accessory region) are much more common, and have lately been thoroughly studied; of the most frequent form, *posticus paralysis* in tabes, there is already quite a voluminous literature. It may be unilateral or bilateral; in the latter case it shows itself by a disturbance of the respiration, by dyspnea, and by an inspiratory stridor which appears upon exertion, and not infrequently may lead to laryngeal crises. These may be so serious and menacing to life that tracheotomy becomes necessary; I saw a patient of this kind, with his tracheal cannula, who lived for six years afterward; the laryngoscope at once reveals the affection. Other paralyses of the muscles of the larynx are also noted: Unilateral or bilateral *paralysis of the vocal cords* with hoarseness and aphonia, and also unilateral or bilateral paralysis of the recurrent *laryngeal nerve.*

(But little has been reported of anesthesia of the larynx in such cases.) Very rarely isolated paralysis of the external branches of the spinal accessory with paralysis and atrophy of the trapezius and sternocleidomastoid is observed.

Finally, we come to the rare *paralyses of the hypoglossal nerves* in tabes. *Unilateral atrophy of the tongue* and its drastic consequences (atrophy, wrinkling, and decreased size of the affected half of the tongue, deviation of the tongue to the atrophic side, fibrillary contractions, and probably DeR) are known and have been described. But *bilateral atrophic paralysis of the tongue* occurs, as a rule, as an accompanying phenomenon of bilateral bulbar paralysis.

Combined, distributed, and usually progressive, paralysis of the cranial nerve is by no means rare in tabes: Sometimes in the form of *nuclear paralysis of the muscles of the eyes,* sometimes combined with paralysis of the muscles of deglutition, etc., sometimes as *typical progressive bulbar paralysis* (tongue, lips, and pharyngeal paralysis), but also occasionally combined with paralysis of the muscles of mastication and those supplied by the spinal accessory. And even these numerous forms may be so interwoven with each other that the pathologic process appears to implicate all of the motor nerve nuclei of the trunk of the brain (III to VII–X–XII). The previously mentioned unfortunate patient, who for the last six years of his life wore a tracheal cannula, not only had almost complete bilateral ophthalmoplegia externa and interna with ptosis, but also total atrophic paralysis of the muscles of mastication, severe progressive bulbar paralysis with extreme atrophy of the tongue, and, finally, complete paralysis of the muscles of the pharynx. During the last 14 months of his life he was nourished entirely through the stomach-tube. In addition to typical tabes he had partial atrophic paralysis in all four extremities.

With increasing frequency recently paralyses of individual *nerves of the extremities,* with and without atrophy of the muscles or DeR, have been demonstrated; they may be transitory and curable, but are often permanent; they are most likely to be found in the peronei and radials, but also in the tibial, median, ulnar, and other nerves. They chiefly impress us as a form of peripheral neuritic paralysis, but may sometimes indicate disease of the gray anterior columns.

Little is to be said of the *irritative motor phenomena* in tabes; I have already stated that contractures and spasmodic conditions are usually absent. Here and there we note a certain unrest of the limbs which prevents the patients from keeping their arms and legs quiet for a moment, or other marked spontaneous movements may appear which in some cases assume the characteristics of athetosis; but in the main this is rare.

A few notes in regard to *disturbances of other cranial nerves in tabes* may here be included. Symptoms on the part of the *olfactory nerve* are very rare, but unilateral and bilateral loss of smell and derangements of the sense of smell have been described.

Taste is scarcely ever disturbed; the unfortunate patient with bulbar paralysis, previously referred to, retained his "wine tongue" to the last. Disturbances in taste and perversions of taste are only now and then reported (Klippel).

More significant and common are *disorders of hearing*; difficulty in *hearing and deafness* not due to disease of the conduction apparatus have repeatedly been described, and have been attributed to atrophy of the nerve of hear-

ing, also permanent *tinnitus aurium* with or without *vertigo* and *difficulty* in hearing; this sometimes occurs in paroxysms; now and then Ménière's symptom-complex has been observed in association with tabes.

Finally, as a common symptom probably referable to the pneumogastric, *tachycardia* may be mentioned; this is manifested by a more or less rapid increase in the pulse rate from 90 to 120 per minute; it is permanent, causes no distress, and is not explained by any objective finding in the heart.

A few nervous symptoms of *cerebral origin* are now to be mentioned: *Migraine* is here included, provided it occurs for the first time during the course of tabes; but this is most uncommon, and its intimate relation with tabes doubtful; a simultaneous arteriosclerosis is much more likely.

The same is also true of occasional *epileptiform* and *apoplectiform* attacks; they probably do not belong to the pathologic picture of tabes, but are more likely co-effects of the same cause (syphilis), or are to be regarded as incidental complications essentially due to arteriosclerosis. The patient mentioned frequently suffered from tinnitus aurium combined with attacks of *vertigo*, which occasionally caused a prolonged loss of consciousness and somnolence; here an intimate connection seemed likely.

Polyuria and *glycosuria*, now and then observed, are of no great significance. They are both probably due to neurogenous alimentary glycosuria; but an accidental complication with true diabetes mellitus is however possible.

We now come to exceedingly important and remarkable *trophic disturbances*. The most common are the changes in the *joints and bones*: *Arthropathia tabidorum* and *spontaneous fractures* of the bones due to their *abnormal friability*. We chiefly owe our accurate knowledge of these to the researches of Charcot and his school based upon the rich material of the Salpêtrière; but in Germany, as well as other countries, much was done to pave the way to an accurate recognition of these conditions.

Tabetic arthropathy is an extremely peculiar joint affection which is characterized clinically by the *most marked objective arthritic changes*—in the acute stages by enormous swelling of rapid development, in the chronic stage by extreme deformities depending upon atrophy of all the parts of the joint, sub-luxations, and luxations—as well as by the almost *total absence of pain*.

In fact it makes a remarkable impression upon us when we discover in a tabetic that his knee-joint—which during the night has become a tense, fluctuating, unsightly tumor of the size of a man's head, and from which marked edema extends far up the thigh and down the leg—is painless to touch and motion, without redness or fever; or when, later, the joint becomes loose, and sub-luxation and colossal deformities appear such as are scarcely ever seen in any other arthritic affections.

The arthropathy usually occurs in the early stages of tabes, usually long before the ataxia, the onset being often *acute*, without prodromes, without pain, and without fever. There is a marked swelling of the whole joint and its surroundings, due to a profuse serous, sero-fibrinous, or hemorrhagic effusion, as well as to a doughy, tense, cutaneous, or subcutaneous edema. The *chronic* form begins more insidiously, there is less effusion, and either slowly or rapidly the subsequent coarse changes develop. In both forms of the disease the later course is the same: Deformities appear in the joints, there are creaking and crackling sounds, the atrophy of the joint surfaces and cartilage

produces luxations and sub-luxations and various kinds of proliferation, osteophyte formation, and connective tissue thickening, result in unsightly alterations in form, and more or less impair the usefulness of the joint.

This is most conspicuous in the knee-joints: Enormous hyperextension, luxation of different degrees, and unsightly swelling produce a typical picture. Similar abnormalities appear in the joints of the shoulder, the elbow, and the hands. These changes involve many joints of the tarsus and the other bones of the foot, and not rarely cause a loss of the natural curve of the foot, a shortening of its middle portion—the different varieties and forms of which have been described by Charcot and Féré as the "tabes foot."

The combination here presented of serous exudation, atrophy, erosion of the cartilage and epiphyses, destruction or proliferation of the ligaments, tearing of the tendons and ligaments, osteophyte formation, loosening of portions of the bone, luxation and loosening of the joints, and atrophy of the para-articular muscles will be minutely described under the pathologic anatomy. These changes make up a picture of the arthritic affection such as is never observed in ordinary arthritis, neither in arthritis deformans, nor in ordinary chronic articular rheumatism, nor in gout. From this clinical picture, therefore, arise the peculiarities which led the first observers to regard tabetic arthropathy as a special type, as a tropho-neurotic arthritis. And this view is confirmed by the fact that the same arthropathies are found in only one other spinal disease, syringomyelia (and here chiefly in the upper extremities).

This tabetic arthropathy is most common in the *knee-joint* (among a large number of cases Kredel found it here 104 times, in the hip-joint 56 times, in the shoulder-joint 36 times, in the ankle-joint 25 times, in the elbow-joint 15 times, in the toe- and finger-joints 10 and 8 times respectively). The condition is not rarely bilateral in the same joints; occasionally, however, it implicates various joints of the upper and lower extremities side by side, and this indicates a widely distributed disturbance of the nutrition of the joints and bones.

This is also distinctly evident from the fact that these arthropathies are frequently combined with an analogous disturbance in the diaphysis of the long bones which leads to *spontaneous fractures* upon the slightest cause. This sometimes appears in the early stages, not rarely, however, in the ataxic stage. These fractures either occur spontaneously on any brisk movement, such as turning in bed or putting on the shoes, or they follow insignificant trauma.

They run their course without pain and without reaction, are readily recognized, and under appropriate treatment unite rapidly and completely, although there is sometimes a marked callus formation, but they may soon reappear at some other point. Not rarely we find multiple fractures in the same patient.

They are more common in the thigh, and especially at the neck, than in the leg, forearm, or upper arm, and are found in decreasing frequency in the clavicle, pelvis, etc.; they are apparently due to an *abnormal friability of the bone*, probably to a rarefying osseous degeneration, with an increasing deposit of medullary substance. Their analogon is the *spontaneous tearing of tendons*, such as has been observed in the Achilles and the patella tendons; this also is unaccompanied by pain.

In addition we note in some cases *atrophy of the alveolar processes of the*

jaws with consequent falling out of the teeth. Within a few weeks or months all the teeth of one jaw, or of both jaws, may fall out without causing pain; or the entire alveolar process, having become necrotic, sloughs away, and in comparatively young people the jaws appear as atrophic as those of the aged.

An illustration of the not uncommon trophic disturbance of the ***skin*** is the condition known as *mal perforant* (perforating ulcer of the foot). This appears in the early stages, usually upon the sole of the foot in the region of the metatarso-phalangeal joint underneath the great toe, but it may appear in other areas of the sole, on the heel, or on the dorsum of the foot. It is a small, round, cutaneous ulcer which penetrates more and more deeply, even into the bone; it is chronic, torpid, only slightly if at all painful, and heals with the greatest difficulty. It is not remarkable that many persons regard it as a manifestation of syphilis. I have known it to heal most readily under specific treatment—the use of mercurial plaster.

Among other cutaneous disturbances the first to be mentioned is *herpes zoster* which—otherwise rare—is very common in tabetics in the usual (trunk) and in unusual localities; we can scarcely doubt that it is attributable to the recently discovered anatomical changes in the nerve roots and spinal ganglia; this is confirmed by the lymphocytosis which has also been demonstrated in herpes zoster. Atrophy of the skin, *falling out of the nails* and of the hair, pigmentary changes, cutaneous hemorrhages, anomalous sweat secretion, erythema, chronic edema, etc., have all been considered in connection with tabes, whether correctly or not cannot be stated.

Valvular lesions of the heart have also been referred to tabes; particularly *aortic insufficiency* which has been by far most frequently noted, and to which great significance has been attached; but it is certainly not frequent. In 400 cases I found it only 7 times; others have observed it in a greater proportion. After discussion it seems likely that this is either a purely accidental complication (due to articular rheumatism) or it is due to a syphilitic aortitis which causes disease of the aortic valves.

In conclusion, we will cast a glance at the numerous *psychical disturbances* which appear in tabes.

In our former conception of tabes as an affection of the spinal cord, we expected to find no psychical disturbances in the symptom-complex, but, having seen that tabes produces symptoms in nearly all parts of the nervous system as well as in the entire body, it does not appear remarkable that this affection should influence the cerebrum, particularly its psychical functions; hence, from the earliest periods psychical disturbances have been mentioned in the pathology of tabes, and recently its undoubtedly close relation to severe psychical disease and to progressive paralysis has been especially discussed.

Leaving this last affection out of consideration for the time being, there is no doubt that in many cases of typical tabes psychical disturbances are lacking: Intelligence, memory, and the general mental capacity of tabetics remain entirely intact; so far as mental work is concerned the patients may follow their occupations for decades without hindrance. In temperament they are often extremely jolly in contrast with the severe disease from which they suffer; at other times they are depressed, irritable, moody, or distressed by their symptoms, are sleepless, etc. On the other hand, accurate observation has shown that quite a series of psychical disturbances sometimes appear in

tabes, which, however, must be regarded as incidental symptoms or complications: Severe neurasthenic disturbances or slight dementia, hysterical depression, in rare cases actual paranoia, and the like appear; later there are psychical disturbances combined with alcoholism or morphinism, and, finally, what is not altogether rare, those due to syphilitic diseases of the brain. These conditions are, as a rule, easily recognized, but it is sometimes difficult to estimate them at their true value; in practical importance they are far behind *progressive paralysis.*

After Westphal had demonstrated how common is degeneration of the posterior columns in progressive paralysis, after the symptom-picture of paresis (reflex rigidity of the pupil, absence of the patella tendon reflex, Romberg's sign, lancinating pain, certain disturbances in gait, etc.) had been many times described among the important symptoms of tabes, and especially since we learned that progressive paralysis, like tabes, is undoubtedly a syphilogenous affection, among other causes this being in both the important one which develops the disease, and since the one disease is exceedingly apt to follow the other, the conviction was more and more strongly forced upon us that tabes and progressive paralysis are related to each other, that they represent analogous forms of disease produced by the same conditions, that they have the same anatomical foundation, and differ only in their localization. Some authors (Möbius, Raymond, Nageotte, Mott, and others) have not hesitated to declare that tabes and progressive paralysis are really one disease, a metasyphilis, the former chiefly or exclusively attacks the spinal cord, the latter implicating the brain. The combination of these affections might then be designated "*taboparalysis.*" But this is going too far: The identity of the actual pathological process in these two diseases has not yet been proven; the combination of the affections is not so common that they must be identical; but *they are closely related,* and their consideration from the same points of view is undoubtedly justified.

Clinically the condition presents itself by the addition of progressive paralysis to a tabes which has for a long time existed, or *tabetic symptoms* are demonstrated in the pathologic picture of a *progressive paralysis already present.* The first case appears to be the rarer; the addition of progressive paralysis to a long existing tabes is by no means frequent; it is interesting to know that among the numerous tabetic inmates of Bicêtre Déjérine observed this condition only twice in 8 years; in the tabetic the psychical disturbances of progressive paralysis are subsequently observed, chiefly as those of simple dementia, with paralytic attacks, disturbances of speech, etc., and the patients succumb in the usual way.

The other condition—progressive paralysis to which tabes is added—is probably more common; among a number of patients with progressive paralysis Nageotte observed that two-thirds had symptoms of tabes; this is probably over-estimated. Besides the typical symptoms of progressive paralysis, we find in these patients the symptoms of tabes; besides pupillary differences there is reflex rigidity, instead of being increased, as is the rule, the tendon reflexes are absent, there are lancinating pains, paresthesias, the typical sensory disturbances belonging to tabes (Romberg), marked bladder symptoms, and probably also ataxia; but these symptoms must be searched for, and are usually secondary to the symptoms of progressive paralysis.

Of course, this "taboparalysis" is seen in many varieties and subdivisions, the symptom pictures being of unequal formation. It is unnecessary to describe this more minutely, but in practice the combination in itself is exceedingly important, serious, and of theoretic interest.

From this comprehensive description of the many-sided symptom-picture of tabes in its early stages, we may proceed more rapidly to the final act in this pitiful drama.

The duration of the first stage is prolonged by many variations, many arrests of the affection, and is exceedingly long, yet for many a one desirous of living not long enough; it may be 5, 10, 15, 20, or more years. But the suffering, the helplessness which constantly becomes greater, the pains and the recurring crises, the amaurosis, the incontinence of the bladder, the cystitis, the decreased nutrition, the obstinate constipation, and many other evils exhaust the strength of the patient. The *terminal stage* of the disease, "paralytic" or "paraplegic" as it has been called, appears. The motor power diminishes more and more, the greatly emaciated legs become paralyzed and stiff, contractures and deformities appear, the digestion suffers, appetite is lost, constipation becomes more obstinate, and *incontinentia alvi* becomes more serious; to the increasing paralysis of the bladder, cystitis and its sequelæ, pyelitis and pyonephritis, are added. Bed-sores appear, and increasing cachexia, marasmus, and general exhaustion finally terminate the sufferings of the patient.

Fortunate are those in whom an intercurrent affection (enteric fever, pneumonia, influenza, pulmonary phthisis, dysentery, or the like) early brings about the final act.

In some cases the disease terminates with a peculiar nervous prostration, a deep somnolence, with increased pulse, Cheyne-Stokes respiration, and a rise or fall in temperature which may last from one to three days—apparently a general paralysis of the cerebral functions. The duration of the disease—counting its three stages—may be calculated at from 20 to 30 years. Of course some cases terminate much earlier, even in 5 or 10 years, especially when there are grave complications (cerebral syphilis, progressive paralysis, severe pyonephritis, etc.).

Before minutely analyzing the principal symptoms and their pathogenesis, it is most necessary to study the pathologico-anatomical changes in tabes.

IV. PATHOLOGIC ANATOMY

It is almost impossible, from our present knowledge, to give a satisfactory, conclusive, and didactic description of the pathologic anatomy of tabes.

More than 40 years of painstaking study and innumerable investigations carried out with most exact methods have gradually taught us the localization of the process and how to explain it, but not its actual starting-point or its nature. In regard to these two points there has been much discussion in the last few years, old and long-forgotten views have been revived, and supported by new and important findings, the seemingly positive explanations generally accepted are combated by others, one theory annulling another, all being more or less opposed to each other, and for the objective investigator no single one to be regarded as certain. After such long and patient study this is really

an admission of which to be ashamed! The subject is, however, extremely intricate, many-sided, and complex, the investigation of each individual case requires so much time and research, so much insight and exactness, that we readily comprehend why we cannot arrive at a satisfactory explanation, especially as each individual case presents its own peculiarities and variations.

We shall attempt briefly to describe the essential and positive facts, and will refer the reader to innumerable treatises and compilations which have been published in the last few years by v. Leyden, Déjérine and Thomas, and Collins, and especially to the excellent description given by Schmaus in his "Lectures upon the Pathological Anatomy of the Spinal Cord." Of course it is impossible for me to discuss fully the literature of this difficult subject.

Upon *microscopic examination* of the spinal cord in recent and mild cases, we observe externally little or nothing; in all more advanced cases, however, it is evident that the spinal cord as a whole, especially its lower sections, is somewhat smaller than normal, and that this change affects mainly the posterior half, especially the *posterior columns*. These are gray, narrower, somewhat sunken, and of coarse consistence; they are most noticeable in the lumbar cord, and decrease as they extend upward toward the cervical cord.

Decided changes take place in the *posterior roots*, these usually being most conspicuous in the cauda equina, but extend upward to the thoracic or cervical cord; the roots become atrophied, are of a grayish luster, are often reduced in size to the diameter of a thread, and differ markedly from the well-retained, round, whitish, and glistening anterior roots.

As a rule, the *meninges* show distinct but, as a rule, insignificant changes; in some cases they may be marked. There is usually slight turbidity and thickening of the soft membranes, with firm adhesion to the spinal cord to the roots and to the connective tissue threads, etc.; a mild leptomeningitis posterior, generally confined to the posterior surface of the spinal cord and its lumbo-dorsal portion.

In *fresh*, unstained, *cross sections* the gray discoloration of the white posterior columns becomes most obvious, and their transparent, coarse consistence. The posterior columns appear to be depressed beneath the posterior surface and decidedly shrunken, hence they approximate one another; they also, as a rule, appear to be decreased in size and atrophic. The lateral and anterior columns, and the gray anterior columns, present a normal appearance.

This "*gray degeneration* (*sclerosis*) *of the posterior columns*" is most conspicuous and complete in the lumbar cord, it decreases in the upper thoracic cord, and is less marked in the cervical cord; in these regions it usually attacks only Goll's columns, and Burdach's tract.

This change is rarely uniform—and in individual cases it varies extraordinarily according to the duration of the disease and the varying height in the spinal cord. In tabes superior the process is chiefly localized in the cervical and upper thoracic cord, being merely indicated in the lumbo-dorsal region. The intensity and distribution of the posterior column degeneration is also irregular.

In the later stages, here and there in the posterior lateral columns, pyramidal columns, and cerebellar lateral columns, an indistinctly limited gray discoloration is noted.

Accurate knowledge of these topographical relations is only to be obtained

by hardening and examination of the *stained cross sections* (after Weigert, etc.), taken from various heights in the spinal cord; and should be done with low as well as high magnification.

We need not minutely discuss the exact *histological details* of the process. It aids but little in explaining the pathological condition as a whole. Everywhere in the degenerated areas we find the same simple processes: More or less *advanced decay of the nerve fibers with a decided increase of the glia tissue.* The nerve fibers degenerate, and more or less uniformly lose their medullary sheath; some of them become swollen and thickened, they subsequently atrophy with the axis cylinders, and finally perish. The proliferated glia appears as a fine fibrillary tissue, in which the spaces left by the nerve fibers which have disappeared are frequently visible; later it changes into a fine, wavy, striated, and tough connective tissue, which shows many nuclei and neuroglia cells, and often numerous *corpora amylacea.* The *vessels* are usually but slightly changed; they show thickening, hyaline formation, a narrowed lumen, and here and there profuse nuclear deposits. In the early cases which show a rapid progress granular cells have been seen.

In the *posterior roots* of the nerve fibers we find exactly the same degenerative, simple, atrophic, processes, varying in intensity in different areas of the same fiber; a conspicuous increase of the connective tissue structure and thickening of the vessels is also noted, and here and there profuse small-cell infiltration.

These findings permit us to conclude with some degree of certainty that *primarily the nerve elements are destroyed, and secondarily there is proliferation of the glia*—therefore a primary degeneration and a secondary sclerosis take place. The process, as a whole, *cannot be differentiated from secondary degeneration of the nerve tracts* due to other causes. This is recognized by most recent authors, and Schmaus expressly maintains that the areas of possible primary degeneration cannot be distinguished from the areas of secondary ascending degeneration.

It is certainly remarkable that the degeneration of the extramedullary posterior roots is often much less advanced and less intense than that of the intramedullary continuations.

But these histologic details are less important than the *localization of the tabetic process in the posterior columns.*

In typical and slowly progressing cases, the process begins in the *lumbar cord* by two symmetrical fields of degeneration in Burdach's columns, which, at about their centers or a little more anteriorly, lie alongside of the posterior columns, and correspond to the so-called root entrance zones ("bandelettes externes" of Pierret); in the higher segments these fields advance toward the center, and finally enter Goll's columns, while new fields of degeneration appear beside them in Burdach's column; thus, a large portion of the transverse section of the posterior column in the upper lumbar and the lower dorsal cord may be implicated, while in the middle and upper thoracic cord the degeneration is limited to Goll's columns (the middle portion).

If the process advances higher, new fields of degeneration in Burdach's column constantly appear in the various segments of the *thoracic cord*, and those fibers originating from the lumbar and sacral cord, and degenerating upward, more completely fill the posterior portion of Goll's columns—until

at last almost the entire transverse section of the posterior columns in the thoracic cord has degenerated.

This process is then repeated in the *cervical cord*—the same advanced degeneration of Goll's columns that is seen in any conspicuous secondary ascending degeneration—and new fields of degeneration corresponding to the entrance zone of the roots in Burdach's columns are constantly added, until, finally, the morbid process gradually terminates in the upper cervical cord at the height of the calamus scriptorius.

This is the typical course; it may, however, show variations as the individual root bundles are not always uniformly diseased from below upward; for instance, when the process skips individual segments, or when it begins in the cervical cord; then there is at first no degeneration of Goll's column, but the change is limited to the cervical root fibers and the higher areas, etc.

Closer investigation teaches us that *in the earlier stages this degeneration is chiefly localized at the entrance of the posterior root fibers.* In the first place it directly affects the ascending coarse fibers which pass through Burdach's column into Goll's columns, then the finer root fibers situated in Lissauer's marginal zone (peripherally from the tip of the posterior columns); in tabes this zone almost invariably degenerates; it also affects the coarse fibers which pass into the posterior columns in an arch, on one side into Clarke's columns (a constant decrease of the fibers which enter these columns while the cells are retained), on the other side anteriorly through the posterior columns to terminate in the vicinity of the large ganglion cells of the anterior columns, the so-called reflex collaterals. There is a decrease of spongy and gelatinous substance in the fibers, and in the posterior columns in general, which permits the recognition of a corresponding glia proliferation.

Hence, certainly in the early stages and for a long time, there is no degeneration in the so-called *ventral posterior column field* (at the tip of the posterior columns), nor in the semilunar *posterior external field* (throughout the entire spinal cord) and, in the lumbar cord, only in the so-called *dorso-medial bundle* (a small oval field at either side of the posterior longitudinal space).

It is assumed that these fields are chiefly composed of *endogenous* fibers, therefore they do not come from the posterior root fibers or their collaterals. But, in the later course of older cases of tabes, these also may completely degenerate, so that, finally, especially in the lumbar and thoracic cords, the posterior columns appear to have completely and uniformly degenerated.

Flechsig attempted to prove that the tabetic fields of degeneration correspond with the embryonal fields of the posterior column which he discovered; as a matter of fact there is a close analogy, but some authors doubt this, or consider it to be a mere coincidence.

In many cases it has been found that the *shorter* intramedullary root fibers, probably also their collaterals, are most completely degenerated, and from this, as well as from the longer exemption of fields which are still included within the posterior root tracts, it has been concluded that the condition is one of *selective* disease of the root fibers, some being earlier, some later attacked, but this is still a matter of doubt.

Observations and the preceding description have, however, shown with certainty that tabetic disease of the posterior columns is nothing more than a *segmental disease of the posterior root areas ascending from below upward.*

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The lesion of the tabetic posterior column is, therefore, *unquestionably radicular,* and is exactly the same as in a secondary degeneration after compression of the root (for instance, by a tumor of the cauda equina, and the like); the atrophy of Burdach's columns and Goll's columns is generally in proportion to the position and number of the degenerated posterior roots.

This picture reveals the nature and the development of the process which must finally lead to columnar degeneration of nearly the entire posterior column. Segments of the individual posterior root bundles are attacked one by one in an upward direction; each field passes through a characteristic degeneration, first in Burdach's, and then in Goll's columns; these fields are situated one above the other in Burdach's columns, and they elongate and distribute their processes in Goll's columns, until from below both pass far upward, and appear as columnar degeneration in the vicinity of the medulla oblongata. In this sense, therefore, tabetic disease, in so far as it affects the posterior columns, is a *systematic lesion*; it affects the various fiber systems which are situated in the posterior roots.

The process usually begins symmetrically in individual root bundles, constantly implicating more and more of these, and ascending and progressing with varying rapidity; it may begin in the lumbar region, almost invariably in the upper lumbar, sometimes also in the lumbodorsal, more rarely in the sacral cord or the conus medullaris, and quite as rarely in the cervical cord, in which case it gives rise to tabes cervicalis or superior, to which we have frequently referred.

Occasionally the process appears to be confined to a few roots (then clinically differentiated as tabes incompleta) or even to an individual root bundle, in which case it has been called *mono-radicular* tabes. Such cases, of course, only accidentally become subject to histologic investigation.

Therefore, we are dealing primarily with a strictly limited disease of the *peripheral sensory neuron* (ganglion cells in the spinal ganglion), and chiefly of its *spinal axon.*

Before discussing this view, we must mention other anatomical changes which have been found in the spinal cord itself and the other parts of the nervous system.

In the earlier stages we invariably find in the *spinal cord* the decided degeneration of the *gray posterior columns* which has already been described. Not infrequently, but always later, the gray degeneration implicates the *lateral columns,* there is usually marginal degeneration of the cerebellar lateral column tracts and Gowers' bundles, which is not strictly confined to these boundaries but extends even beyond them to the pyramidal columns. Occasionally, especially in long-standing cases, marked changes may be widely distributed and included within the combined system diseases (observations of Kattwinkel and others).

Changes are rarely found in the *gray anterior columns.* Only in isolated cases of amyotrophia has degeneration of the cornual cells, etc., been noted.

In the *meninges* there is cloudiness and thickening with connective tissue adhesions; this is most marked between the posterior roots, rarely encircling the entire spinal cord. In quite a number of cases published in Germany (Hoffmann, Dinkler, etc.), there was conspicuous syphilitic leptomeningitis. Later, however, French authors (Nageotte, Déjérine, etc.) attached special

significance to the almost invariable occurrence of *mild* meningitis with proliferation of small embryonal cells in the membranes and the vessels, being more marked in the veins than in the arteries; Nageotte maintains that these are syphilitic. They are identical with the meningitis accompanying myelitis syphilitica, but are usually confined within moderate limits.

The *constant* degeneration of the *posterior roots,* as well as their histologic condition, has already been described; in the *anterior roots* there are usually no degenerative changes or these are but slight, and are found in cases complicated by amyotrophia, and in degeneration of the gray *anterior columns.*

On account of certain views which have been discussed, the *spinal ganglia* have recently been thoroughly studied, but they show no invariable changes; what little has been found in the cells (vacuoles, contraction, atrophy, pigmentation, tigrolysis, etc.) is unimportant, and does not permit the assumption of constant or great anatomical change, but neither does it exclude a functional disturbance. In 1904 Thomas and Hauser again announced the great significance of these changes in the pathogenesis of tabes; they invariably found them to be more or less intense.

The fact—noted by most investigators—is very remarkable that the afferent posterior root fibers (following the physiologic direction of conduction) are markedly degenerated above the ganglion, while the afferent peripheral nerve fibers are intact or but slightly changed.

It has been known for some time (v. Leyden, 1863) that the *peripheral nerves* sustain degenerative neuritic lesions, etc.; these are most severe in the sensory nerves, especially in the small distal cutaneous branches, being less severe in the nerve trunks and certain nerves, and least so in the proximal parts (Westphal, Pierret, Déjérine, Oppenheim and Siemerling, Nonne, and others), but this is by no means constant. It is certainly remarkable that the peripheral sensory nerves near the ganglion, as well as the ganglion itself, should show such insignificant changes, while the posterior roots belonging to them have degenerated to an extreme degree.

The peripheral *motor nerves* rarely show degenerative change; here we must except the *nerves of the ocular muscles* which are frequently altered, and show neuritic change. Sometimes other cranial nerves are affected: The hypoglossal, the pneumogastric and spinal accessory, the recurrent laryngeal, etc.

Especially important and common is *atrophy of the optic nerves,* which is evidently a primary parenchymatous degeneration, usually combined, it is assumed, with changes in the nerve-cells of the retina. Degenerative changes have occasionally been noted also in the *olfactory and auditory nerves.*

Here must be mentioned *degenerations in the nuclei of the cranial nerves in the trunk of the brain,* the nuclei of the muscles of the eye, the bulbar nuclei, etc., with the clinical disturbances belonging thereto.

In the *sympathetic* nothing positive has been found; a degeneration of the fine medullary fibers has sometimes been noted, the *coarser* ones remaining intact.

In the *brain,* in the cerebrum as well as in the cerebellum, cortical fiber atrophy has been occasionally demonstrated, and unless the frequent combination with paresis is excepted, this appears to be incidental and to have but little in common with tabes. What importance is to be attached to the

changes in the *molecular layer of the cerebellum*, which appear to be constant and to which C. Weigert called attention (*Neurolog. Centralbl.*, 1904, p. 738), remains to be seen; it may be considerable.

Therefore, the essential feature of tabes is undoubtedly a *segmentary disease of the posterior root tracts*, both intramedullary and extramedullary. There is almost complete unanimity of opinion concerning this. But the questions arise, What is the nature of the process? What produces it? Is it a primary or a secondary degeneration? If the latter, what is the *actual starting-point* of the process?

All these questions have been much considered of late, much investigated and discussed, but the answers have differed greatly; important facts have come to light, new views have arisen—but unanimity has not yet been attained. The questions are still mooted, and at present no conclusive opinion can be reached. Hence only a brief report concerning the various views is possible; a critical and comprehensive review of all the facts, views, theories, etc., that have been advanced, would lead us too far from our subject.

The old view that tabes is a *chronic inflammation of vascular nature* with a connective tissue sclerosis and secondary degeneration of the nervous elements, and that this inflammation essentially depends upon the vascular division of the posterior columns and their localization, was long ago disproven and generally discarded. But, in a modified form, a medullary theory depending especially upon the lymphatic tracts of the posterior columns has lately been promulgated by P. Marie and Guillain.

The view has steadily become more prominent, and in the last few years has become the dominant one, that the condition is one of *primary parenchymatous degeneration* of the actual nerve tracts (the posterior roots), and that this is due to unknown deleterious influences, and, with increasing certainty based upon clinical experiences, this baneful factor is assumed to be the *action of certain poisons generated by syphilis*. The analogous action upon the spinal cord of other poisons, of ergotin, pellagra, lathyrus poison, lead, and alcohol, also of certain autointoxications as in severe pernicious anemia, etc., which preferably affect the posterior columns, and produce lesions very similar to tabes, appears to support this view. But this theory is disputed, and, at present, is merely a hypothesis.

Anatomical findings have gradually forced us to recognize in the tabetic change a *secondary degeneration of the posterior root tracts*; although these do not exactly correspond with the common secondary degeneration caused by coarse root lesions (in fractures of the vertebræ or tumors of the cauda equina) it does not appear remarkable that in such an exceedingly chronic process in which the individual root bundles either simultaneously or successively become diseased (and the origin of this secondary degeneration was eagerly sought for) a dynamic or mechanical action, which must be found, had at some point interrupted the nutrition and conduction of the posterior root tracts in such a way as to lead to secondary degeneration.

Under the domination of the neuron theory, and after the trophic centers of the posterior root tracts were recognized in the ganglion cells of the spinal ganglion, what was more natural than to think of these *spinal ganglia*, and to search in them for the actual starting-point of the tabetic posterior column lesion? This highly plausible theory, unfortunately, could not be proven,

although many special investigations were undertaken for the purpose; the histologic changes in the cells of the spinal ganglion are not sufficiently distinct nor constant enough to be regarded as the anatomical starting-point of severe changes in the roots which go on for years. The forcible objections have been raised that, besides the almost intact ganglion, the *centripetal* root fibers close to the ganglion are markedly degenerated, the *centrifugal* peripheral nerve fibers, however, being but little altered, and that even the degeneration of tabes is often entirely absent in the peripheral nerves.

The same facts controvert the otherwise plausible hypothesis that the condition is essentially one of functional disturbance of the spinal ganglion cells, and this produces the trophic changes in the axons, particularly the spinal ones. The theory was first suggested by me 20 years ago (and has since then been generally accepted) that functional disturbances in the trophic centers (neuron cells) which are microscopically unrecognizable may produce visible and more or less decided trophic disturbances (degeneration and atrophy) in the motor nerves and the muscles innervated by them. It seems plausible that a definite toxic action which does not histologically damage the cell may, nevertheless, disturb its function to such a degree that the axon, first at its distal ends, and subsequently even the cell itself, may degenerate. It is unnecessary to discuss this possibility; whether it is true in this case cannot be stated; hence the conception of a functional or anatomical change of the spinal ganglion as the origin of tabes—of course only in so far as it is manifested in the posterior columns and posterior roots—is merely a hypothesis which must be proven.

The same is true of the view proposed some time ago by Leyden, and lately also by Goldscheider, that tabes begins in the *peripheral sensory nerves* (chiefly in the cutaneous nerves), that here the first degenerative changes appear, and that these extend centripetally through the spinal ganglion to the posterior roots and the spinal cord. The anatomical facts (inconstant affection of the peripheral nerves, slighter degeneration of these than of the posterior roots, and exemption of the ganglion itself), the extent, the localization, and the development of the tabetic process, the early implication of the reflex collaterals, etc., are opposed to this view, while for the etiologic factors (cold, trauma, etc.) which certainly do not play a prominent rôle, as well as for the appearance of affections of the spinal cord after amputations, all grounds of support are removed. Therefore, this theory of the development of tabes, which v. Leyden maintains with some modifications, is rejected by almost all recent authors.

Finally, the starting-point of the tabetic lesion was sought in the *meninges*, in a chronic meningitis. This obsolete, and apparently long forgotten, view has recently been restored to a place of honor, naturally in a new form. The fact is certain that severe meningitis—that form leading to coarse indurative thickening, or even syphilitic meningitis—as a rule produces no tabetic root lesion. The same may be occasionally said of syphilitic meningitis, but in this there may be coördinative consequences of syphilis. It is also certain that in tabes there are never any decided and invariable, although mild, meningitic changes. Therefore, stronger warrant for this assumption must be found.

Obersteiner and Redlich demonstrated that the *entering posterior root bundles on the side of the somewhat thickened pia mater suffer a slight con-*

striction, consequently they show intramedullary degeneration. The point at which the roots pass through the pia offers less resistance, therefore facilitates this process. There is much in opposition to this view; the most important argument, in my opinion, is that this hypothesis does not explain the invariable degeneration of the posterior roots, and the changes in the pia are too slight to explain the severe compression, etc. But even if the condition seen in most cases is not an artifact (due to hardening), and subsequent examinations have shown it to be by no means constant, this hypothesis is nowhere accepted, and we may say, has been withdrawn even by its originators.

On the other hand, Nageotte [1] believes that he has found another invariable starting-point for the tabetic root degeneration—this upon the basis of accurate and, apparently, very important investigations which we must consider somewhat more minutely.

In his opinion tabes is the *result of a local affection of the spinal roots at the height of the "nerfs radiculaires"* (by this Nageotte means that portion of the roots from their common entrance into the dura mater up to the spinal ganglion; see diagram); this disease sometimes extends as far as the spinal ganglion. It consists in an *interstitial transverse root neuritis* (perineuritis and endoneuritis with interstitial and parenchymatous changes) which is due to a mild but *chronic syphilitic meningitis*, its development and course being determined by the arrangement of the lymph tracts. This *root neuritis* is also *syphilitic.*

According to Nageotte, this *diffuse syphilitic meningitis* which may be recognized by a slight thickening of the pia, with diffuse or focal cellular infiltration (especially profuse in recent cases!), with specific vascular changes in the arteries, capillaries, and especially in the *veins*, and which upon lumbar puncture invariably shows *lymphocytosis, always precedes tabes*, but tabes is developed only by its *combination with root neuritis.* It may be demonstrated by cytodiagnosis from the earliest symptoms of tabes (isolated reflex pupillary rigidity), and even prior to its appearance.

Neuritis of the posterior roots consists in a *more or less severe interstitial change* which finally leads to sclerosis; it always permits us to recognize recent cellular infiltration (the persistent activity of the process corresponding to the progressive character of tabes); it also consists in *degeneration of the root fibers* without a tendency to regeneration; *finally, the fibers are completely destroyed*, some sooner, some later, and then secondary ascending degeneration of the posterior roots and their spinal continuations results. The fact that the degeneration at the beginning often shows only an intramedullary character Nageotte explains by the assumption that the short root fibers (collaterals?) degenerate first at their distal ends, the process then extending cellulipetally (apparently a retrogressive change).

Strange to say, the *motor roots* are not exempt; but their injury is less severe, is usually higher than the focus in the posterior roots, and the degenerated fibers show a distinct tendency to regeneration. (This may possibly account for the absence of motor symptoms.)

The "nerf radiculaire" is a channel for the lymph circulation in the central nervous system, and lesions or irritation in the cerebrospinal fluid may

[1] "Pathogénie du Tabes dorsalis," 1903.

perhaps at this point have a more concentrated effect. In syphilitic meningitis a *syphilitic interstitial root neuritis* here establishes itself; this is not necessarily present in all cases of syphilitic meningitis, but *it is constant in tabes.* Nageotte found it in 11 cases which he carefully investigated.

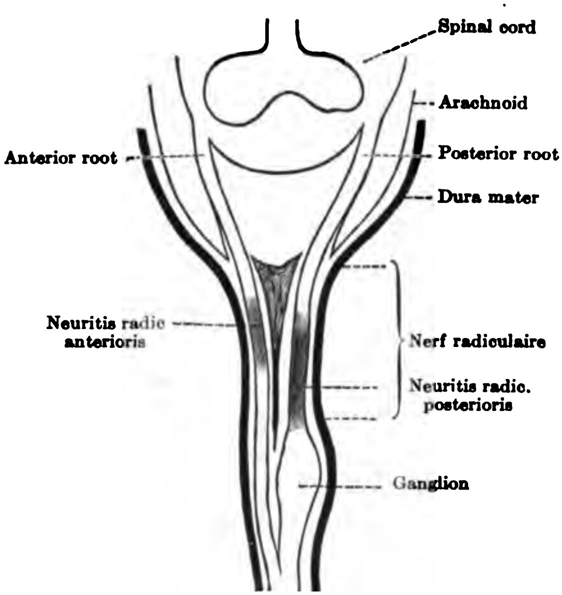

FIG. 173.—DIAGRAM OF THE COURSE OF THE SPINAL ROOTS AND OF THE NERF RADICULAIRE NAGEOTTE.

It may be present in many roots, sometimes in the lumbo-sacral, at other times in the dorso-cervical roots; or some of the roots may at various heights be separated by normal ones, sometimes limited to a few, to one, or to two pairs of roots—therefore in very irregular distribution.

In the main the foregoing embodies the findings and the views of Nageotte. It is evident that all sorts of objections may be raised to this theory, but it seems satisfactorily to explain the anatomical as well as the clinical picture of tabes—certainly its spinal lesions.

But we have as yet no proof that these findings are invariable; hence we cannot at present decide in favor of this pathogenesis.

From the same base the assumption of a "syphilosis" of the meninges, therefore of a chronic syphilitic meningitis, P. Marie and Guillain[1] have recently arrived at a quite different opinion; they invariably found in tabes a posterior meningitis, especially in the dorsal region, and assumed the existence of a peculiar and highly important lymphatic system in the posterior roots, in the pia, and in the posterior columns, and that syphilitic inflammation of this system is the cause of tabes. They point to the strict root localization of tabes in the posterior column, and believe that there is often a merely pseudo-radicular distribution dependent upon division of the lymph tracts; these authors regard tabes as nothing more than a *syphilitic lymphangitis* in the posterior lymphatic system of the spinal cord.

I am unable to understand how these authors reconcile the conditions histologically, and how they can deduce therefrom the entire tabetic lesion; here also we must await developments.

Thomas and Hauser have recently propounded another theory according to which tabes is a *toxic neuritis* (a "dystrophy," as they call it) of the *peripheral sensory neuron*, chiefly existing in the *central fiber distribution* of

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[1] "Revue neurologique," 1903.

the neuron. They rigidly hold to primary degeneration in consequence of toxic influences, but make all sorts of concessions concerning other related views.

These are the most important of the recent theories as to the pathogenesis of tabes; others which have been promulgated we may ignore. At this time it does not appear to me proper to declare positively in favor of one or the other. In my opinion, Nageotte's views based upon carefully ascertained facts most deserve confidence. I have seen some of his preparations, and they are very convincing; whether or not his views are correct depends upon whether or not his findings are constant, and whether or not his root neuritis is the co-effect of the same (toxic?) cause as the degeneration of the posterior root tract; therefore whether or not the intermediary link of a root neuritis is required.

In spite of numberless investigations the *secondary* disease of the posterior root tracts is still undetermined, and it is possible that in tabes we are dealing with a *primary degeneration of the root tracts,* which at the onset of their intramedullary course are due to *toxic action* in combination with certain auxiliary causes (Schmaus). And with this we come at last to the question of the actual *remote cause* of tabes, and its *relation to the anatomical findings.* Here everything revolves about the question whether *syphilis* is this ultimate cause, and whether it can produce these anatomical changes. This will be briefly discussed.

Anatomical facts (aside from clinical facts) which favor the syphilogenous origin of tabes have recently accumulated. The French (Nageotte, P. Marie, Guillain, Déjérine, and others) speak merely of a "syphilosis" of the meninges, a syphilitic root neuritis, etc., in tabes, and with good reason; for these authors the question has undoubtedly been decided; they also, in the old sense, find "specific" changes in the meninges and roots.

That these typical changes, the gray degeneration of tabes, are in this sense "not specific" is certain. That they may depend upon syphilis I *no longer doubt.* Even the fact that, in a large number of cases of tabes, besides the "typical" tabetic lesion we recognize quite a series of "specific" changes in the meninges and vessels, in the form of small cell infiltration, gummata, etc., renders it very likely; I have recently compiled these cases in an article [1] in which I attempted to prove the syphilogenous nature of these simple and apparently non-specific changes by the same reasoning and facts (the only decisive ones!) as the so-called specific gummatous changes.

I believe that no one to-day will seriously maintain that these conditions *cannot* be of syphilitic origin. But we have no scientific proof that they are actually syphilitic, nor for the assumption that the "gummatous" changes are invariably specific.

I shall refer only to the views of Leredde [2] who maintains with great warmth, and for the reasons indicated by us, that the tabetic lesions are certainly syphilitic; also to the recent view of Fritz Lesser [3] that tabes belongs to the *fourth stage of syphilis,* that the *interstitial* chronic inflammatory

[1] "Bemerkungen zur pathologischen Anatomie der Syphilis des centralen Nervensystems." *Deutsche Zeitschr. f. Nervenheilk.*, 1902, XXII.

[2] Leredde, "La question de la parasyphilis." *Progrès médical,* 1902, Nr. 14.

[3] *Berliner klin. Wochenschr.*, 1904, Nr. 4.

processes are directly produced by the syphilitic virus, a view which at present I do not share, and which has been rejected by the competent scientists of Berlin; proof of this is still lacking; time will disclose whether there is any truth in the opinion.

Notwithstanding this—and considering the impossibility of pathological anatomy leading us to any decision—there is no well founded objection to our recognizing the tabetic changes as actually syphilogenous; on the contrary, it is in a *high degree likely* that they *depend directly upon syphilis,* and are in some way produced by this affection.

This conclusion is borne out by the recently recognized results of *cytodiagnosis of the cerebrospinal fluid,* as elaborated by Widal, by numerous authors in France (Sicard, Babinski, Nageotte, Raymond, Brissaud, Pierre Marie, Ravaut, Meige, and others) and also in Germany (Schönborn, Frenkel, D. Gerhardt, Mayer, and Nissl); this is a subject of absorbing interest, to which a few words will be devoted.

While but *few lymphocytes* are found in the normal cerebrospinal fluid, even by minute methods of investigation—i. e., not more than 2 to 4 in a microscopic field—in cases of *active syphilis* of the meninges, of the spinal cord, or of the brain these are *decidedly increased,* and from 20 to 200 may be counted in a field; this same increase is found almost invariably in recent and old cases of tabes and in progressive paralysis, therefore in so-called metasyphilitic affections, while in all other chronic diseases of the brain and spinal cord it is either absent or only occasionally met with (as in multiple sclerosis). A similar lymphocytosis has been observed only in herpes zoster and in certain acute forms of infectious meningitis, especially the tuberculous, in which it is combined with a polynuclear leukocytosis. It is a specially interesting fact that the same leukocytosis is found in isolated cases of reflex pupillary rigidity, which is almost pathognomonic of syphilis, in the headache of chronic syphilis, and in hemiplegia with a luetic basis. How often it may occur in the secondary and tertiary stage of syphilis without nervous symptoms can only be determined by further researches. In his investigations of syphilis in all stages of the disease, Ravaut claims to have found lymphocytosis without even a trace of disease of the nervous system, and it is absent only in syphilis with mild cutaneous and mucous membrane implication.

These reports were tested by Dr. Schönborn [1] in a large series of cases in my Clinic, and in the main were confirmed; among 35 cases of tabes, lymphocytosis was found in every instance.[2]

If these findings continue to be corroborated—and there can scarcely be doubt of this—they are certainly of paramount importance in the conception of tabes (and of paresis) as a syphilogenous affection. Lymphocytosis may therefore be included among the positive pathologico-anatomical findings, and with syphilis of the meninges is probably a chief factor in the pathogenesis of tabes.

Of course, I am far from regarding these investigations as final. The time has been too short, and the number of cases observed is not large enough.

[1] "Die Cytodiagnose des Liquor cerebrospinalis." *Neurol. Centralbl.,* 1903, Nr. 13.

[2] In the last case, just investigated, in which syphilis could not be demonstrated, lymphocytosis was not present; the diagnosis is not quite clear, and we incline somewhat to the view of a neuritic pseudo-tabes.

But I believe them to be important auxiliaries for those who regard the pathologico-anatomical changes in tabes as most likely the sign of a *syphilitic infiltration* and a late *syphilitic manifestation.* We will no doubt come to a conclusion in the near future.

Of the pathologico-anatomical processes not referable to the nervous system but little need be said; probably the only noteworthy ones are the changes in the *bones and joints. Tabetic arthropathy* I shall briefly sketch, referring the reader for details to text-books on pathological anatomy. It is a peculiar form of arthritis deformans which sets in with an enormous effusion of serous or slightly hemorrhagic fluid into the joints and their surroundings (subcutaneous edema), with swelling and proliferation in the synovial membrane, and in all cases leads with comparative rapidity to destruction of the joints, atrophy and loosening of the cartilage, flattening, atrophy and rarefication of the head of the joint, of the epiphyses, and of the floor of the joint, to loosening of the osseous parts, etc.; there is also accompanying but slight osteophyte formation. The ligaments undergo atrophy and tear, the surrounding muscles also atrophy, and sub-luxations, luxations, loose joints, pseudo-arthroses, and anchyloses are the consequences. Suppuration or tuberculosis of the joints is rare. The joints most frequently implicated are those which have been mentioned.

The *abnormal friability of the bones* which leads to *spontaneous fractures* is the result of a rarefying osteitis and of osteoporotic processes, of an increase of the medullary substance, of atrophy of the solid bony tissue, and of osteomalacic softening. The tabetic foot, atrophy and necrosis of the jaws, the falling out of the teeth, and mal perforant, etc., which result from similar conditions, require no description.

Every possible form and degree of atrophy is present in the *muscles,* in part simple diffuse emaciation and atrophy, in part more localized forms and degenerative atrophy of the usual type, as is evident from the clinical description.

In the internal organs—the respiratory, circulatory, digestive, urinary, and sexual systems—as a rule nothing characteristic of tabes is found, certainly nothing more than those conditions which appear in the terminal stages of any chronic spinal affection.

Fritz Lesser's pathologico-anatomical investigations are noteworthy, for he demonstrated that *every fifth tabetic* has an *aneurysm,* and this has some bearing on the syphilitic etiology. His figures, however, have been disputed.

We must still devote a few words to the *special pathogenesis of the anatomical changes,* i. e., their *deduction from the causes* of tabes, and, owing to our limited knowledge of these affections, we may be brief.

We believe that under the discussion of the etiology we proved that syphilis is by far the most important and most common, perhaps even the invariable, cause of the development of tabes, that the anatomical changes may be regarded as directly or indirectly produced by syphilis. Therefore, they may represent a *third* or, as was recently proposed by Fritz Lesser, perhaps a *fourth* stage in the manifestations of syphilis, or are certainly closely related to them. Many hypotheses have been suggested to explain the nature and manner of this intimate relation.

Unfortunately, we have as yet no actual knowledge of the pathogenic cause of syphilis, but it is most likely of bacterial nature (somewhat analogous to the pathogenic agent of tuberculosis, but having quite different effects); we know nothing of its directly irritating and directly damaging action on the tissues, nor whether the pathologic agent itself, its products of metabolism (toxins), or the poisonous products produced in the tissues themselves and their antitoxins—immunization products, etc.—play a rôle in the secondary and tertiary syphilitic manifestations, or, if so, to what extent. We cannot understand as yet how the syphilitic virus or its products can so often remain latent in the body for years and decades, yet preserve their vitality, then suddenly become active, and produce new forms of the disease.

We are, therefore, absolutely restricted to theories, and it remains a mooted question whether an investigator shall regard as the cause of the tabetic changes the actual syphilitic virus, or its products of metabolism, or the products of an interchange of tissue by which the syphilis present in the body produces its pathologic effects.

Nevertheless the resemblance, already referred to, of the tabetic changes to certain inorganic, organic, and bacterial toxic effects, makes it seem likely that in tabes there is such a *poisonous effect,* and that it probably originates from syphilitic infiltration. This thought was first developed in a most explicit way by Strümpell (syphilitoxin).

Whether these poisons are to be sought for among the actual syphilitic toxins, or in the toxins and antitoxins developed in the organism, possibly in the superabundance of the products of immunization which have developed, is still unknown. The unending complexity of this realm which has been revealed by modern bacteriology scarcely permits an opinion, much less a satisfactory conclusion. I refer here to my detailed description of this condition in a clinical lecture, "The Etiology of Tabes," published in 1892.

Therefore, except for the *fundamental fact of its syphilogenous origin,* everything is still uncertain.

The rarity of tabes among syphilitics, and the undoubted effect in certain cases of a so-called *predisposing cause,* necessitate the brief consideration of this form of development. We must, however, bear in mind that these conditions in themselves never give rise to tabes except when there is a preceding syphilitic infiltration.

We cannot comprehend how a single, or repeated, *exposure to cold* could produce such a prolonged and progressive affection, which, after decades, implicates different regions of the nervous system; if it damages the tissues at all, it may in this way form a starting-point for the syphilitic poison, but scarcely more.

This is more readily comprehended in the case of *trauma,* the damage done to the tissue having been determined with certainty, and it may also occasionally give rise to chronic progressive changes in the nervous system. But trauma is very rare as a preceding cause of tabes, and in most cases it produces an effect simply because it forms a point of attack, a port of entrance, for the syphilitic poison which leads to the development of tabes.

The effects of *over-exertion* and *sexual excesses* can be clearly understood from Edinger's masterly substitution hypothesis; in this we assume processes of metabolism, increased consumption, etc., which, with insufficient substitu-

tion, may very likely lead to demonstrable degeneration; but the fact that these conditions are operative only in the case of syphilis decidedly militates against its general or decisive importance. That these processes in other disturbances of metabolism, in anemia, cachexia, faulty nutrition, etc., rarely cause tabes, and only when there is preceding syphilis, decidedly favors the fact that they merely open the way to syphilitic tissue disturbance, and, combined with imperfect substitution, produce the progressive tabetic disease.

We must consider in a similar way the etiologic effects of certain *poisons* (alcohol, tobacco, ergotin) and of some *infections* (influenza, etc.), also a damage to tissue which in itself rarely leads to the disease but only when it is associated with the syphilitic poison; in all of these cases there is a combination of toxic effects, among which the syphilitic is the most important and decisive.

Therefore, much is still uncertain, and the *hypothesis of intoxication* appears to me to be the most plausible one; in many cases, so far as syphilis is concerned, it appears to be quite sufficient; in other instances we must also consider disturbances of metabolism and a loss of regenerative power in the used-up material. The latter can only wisely be assumed with limitations, since it appears that the causes *without* syphilis probably do not produce tabes or only in the rarest cases. It is really most remarkable that tabes does not more commonly result from other toxic and infectious causes, such as debilitating influences, anemia, cachexia, etc.; the fact that it does not is an impressive confirmation of the assumption that it has a definite and specific cause.

We will now attempt to show, as far as possible, the relations of the pathologico-anatomical changes to the symptoms of tabes.

V. PATHOLOGIC PHYSIOLOGY OF TABES. PATHOGENESIS OF THE DIFFERENT SYMPTOMS

In spite of their apparently very simple relations, the deduction of the individual symptoms of tabes from the anatomical changes, and the minute analysis of their development and form, still occasions perplexity. This becomes greater rather than less with increasing knowledge, and while our former views, based upon comparatively crude physiologic tests, simply maintain that " tabes is essentially a disturbance of the sensory centripetal functions, the conduction of which takes place in the spinal cord, particularly in its posterior half, and that in tabes we find the posterior columns degenerated, therefore its symptoms depend chiefly upon this degeneration," yet, when the complicated structure of the posterior columns was disclosed, and the extraordinarily manifold disturbances of sensation occurring in tabes were considered, it was recognized that this simple statement of the facts did not sufficiently explain the subject under discussion. Considering the enormous difficulty of obtaining from animal experiments any results regarding sensory conditions, it is evident that the physiology of sensory conduction in the posterior columns is much more likely to be solved by tabes than that the pathogenesis of the sensory disturbances in tabes can be explained by physiology.

The certain demonstration that the posterior columns are chiefly composed of the nerve tracts brought to them by the posterior roots, that the pathologico-anatomical changes in tabes are unquestionably confined to the posterior root

areas, and that the initial symptoms of the same indicate a radicular arrangement, sustains the former views, although with slight modifications. Of course, our insight into these processes is still far from satisfactory; we will, therefore, be brief in our presentation.

Undoubtedly various *disturbances of cutaneous sensibility* may be deduced from the progressive degeneration of the posterior root tracts. That paresthesia as well as simple hypesthesia, the radicular and localized disturbances of the tactile sense and pain sense, probably also the manifold symptoms of hyperesthesia, may in general be referred to these degenerations can hardly be doubted. But in the individual case it is difficult to explain the many variations in the sensory disturbances: Why in one case the tactile sensation, in another that of pain, almost exclusively suffers; and why isolated hypalgesia and thermo-hypesthesia appear; where the retardation of pain conduction and the peculiar localized hyperesthesia to cold, etc., originate, cannot be stated with certainty. Is it the different localization of the process in the individual root bundles, or the varying intensity or nature of the process, or the localization at various heights of the intraspinal tracts, or the implication of the gray substance to which we attribute it? Nothing but theories can be adduced in answer to these questions.

The implication of the *peripheral* sensory nerves in this disturbance is also quite obscure.

The attacks of *lancinating pain* are exceedingly difficult to explain on account of their singular variations as to intensity, frequency, and localization. Naturally, we locate them in the sensory root tracts, but what process causes them? To what is their paroxysmal character due? Must we consider marked but transient inflammatory irritation, or toxic irritation, or a seeming discharge of nervous energy similar to that in neuralgia, or are they to be referred directly to the peripheral nerves? For the present all these are matters of doubt.

Disturbance of the so-called *muscle sense*, the entire gamut of symptoms arising from a decrease of sensation in individual areas of the skin, the joints, the ligaments, the bones, the muscles, etc., and which early manifests itself as Romberg's sign, undoubtedly can be attributed to a disease of the centripetal tracts of conduction. But whether these are situated in the roots and spinal cord, whether in special tracts, or whether they possess different nerve fibers, no one can state with certainty.

The *sensation of fatigue*, so often present at the onset, is best included here, as well as the occasional *absence of the sensation of fatigue.*

We have more accurate knowledge regarding the pathogenesis of the *reflex disturbances*, especially of the *tendon reflexes.* The apparently positive fact that the root fibers which come from the lateral root zone, radiating into the gray posterior column, and perhaps anteriorly into the anterior column, the so-called reflex collaterals, have been found degenerated, furnishes the simplest explanation, although it is not entirely satisfactory. Concerning the tendon reflexes, we know more definitely the area of the spinal cord in which the respective root tracts are situated; for instance, for the patella tendon reflex it is the region from the second to the fourth lumbar segment, for the tendo Achillis that of the third to the fifth sacral segments, disease of the corresponding root zones being indicated if the reflexes are abolished. For the

tendon reflexes of the upper extremities, the localization must be sought in the sixth and seventh cervical segments.

The conditions are different with the *cutaneous reflexes*; since these are by no means parallel with the tendon reflexes, and they may possess other reflex arcs—complicated tracts of reflex processes—we cannot maintain that the reflex collaterals are situated at a height the same as, or similar to, those of the tendon reflexes. Clinical experience leads us to consider the conduction tracts (the long ones?) and probably also their collaterals which transmit the tactile sense; but where are these situated? We do not know. It is very likely that the conspicuous *hypotonia* of the muscles is also due to disturbances in the reflex tract, but this cannot be positively proven.

We are still in doubt as to the tracts implicated in *bladder and sexual disturbances*; we know that their centers are situated in the lumbar and sacral portions of the cord, also that at many points these must be connected with the higher portions of the spinal cord and of the brain. That the centripetal tracts must also be primarily affected is self-evident; but there is no doubt that in these disturbances motor and vasomotor (therefore centrifugal) tracts must also be considered. In regard to their situation (in the sacral segments and root regions?) we know but little (I shall not discuss the theory recently suggested that some of them lie *external* to the spinal cord in the sympathetic), and still less of their implication in tabes; hence it is useless to propose complicated hypotheses to support this view.

In a small number of cases we demonstrate the cause of the *spinal motor disturbances* (pareses, paralysis, atrophies) to be degeneration of the gray anterior columns and atrophy of the ganglion cells, the gray anterior roots, etc.; in a large proportion of the cases, however, only peripheral neuritic changes in the motor and mixed nerves can be proven, but these furnish a satisfactory explanation.

For the more widely distributed atrophy of the muscles (Déjérine) no anatomical foundation is as yet known, but this also is probably a chronic neuritic change. Whether a lesion of the anterior root, of the *nerf radiculaire*, discovered by Nageotte, is in any way connected with it, is still a matter of conjecture.

For the many *trophic disturbances* which we have learned to recognize among the symptoms of tabes there is as yet no rational anatomical foundation; it is obvious that we must assume a *neurotic origin* for the typical processes of arthropathy, osteoporosis, etc.; there are sufficient, and almost forcible, clinical reasons for such assumption; anatomical facts, however, upon which to base it are not at hand. Therefore we need not minutely discuss the various hypotheses, some of which refer the lesion to the posterior columns, through this to the gray posterior columns, and even to the anterior columns; some to the spinal ganglia or to the peripheral nerves.

It is quite unlikely that these processes, such as chronic syphilitic arthritis, traumatic or gouty arthritic changes, are all to be traced to the nervous system. Perhaps, as Vulpian assumes, an entire series of deleterious effects of nervous or accidental nature is here combined.

According to all experience, however, *herpes zoster*, which often occurs in tabetics, depends upon a degeneration of the posterior root or of the spinal

ganglia, primarily in a peri- and endoneuritis of Nageotte's "nerf radiculaire," and this is sometimes communicated to the spinal ganglion.

The anatomical foundation of the so-called *visceral crises* is quite obscure. They have, perhaps correctly, been regarded as analogous to attacks of lancinating pain; in spite of some variations, this may be admitted. But, in this respect, we have no more accurate knowledge, for we also refer these symptoms to the posterior root regions, and, on the other hand, the sympathetic nerve has also been considered without our being able to adduce any actual proof of its involvement; changes in the pneumogastric and spinal accessory, or in their bulbar nuclei, have been considered to be the cause of gastric, laryngeal, bronchial, and cardiac crises, but this has not been proven.

We now come to changes in the *cranial nerves,* which cannot be regarded as forming part of the spinal changes, but which can nevertheless be designated as localizations of the tabetic process.

Amblyopia and *amaurosis* are always due to the well known gray degeneration and atrophy of the optic nerve, and this can invariably be demonstrated. Similar conditions are found in the *olfactory* and *auditory* nerves, but these disturbances, as a rule, are quite rare.

Paralysis of the ocular muscles, which, in the first stage, is usually transitory, is referred to neuritic or syphilitic processes in different nerves of the ocular muscles. In the later stages extreme peripheral degenerations, especially those in the *nuclei of the nerves of the eye* upon the floor of the third ventricle, have been demonstrated (nuclear paralysis of the muscles of the eye); analogous to these are *bulbar nuclear degenerations* which lead to paralysis of the facial, trigeminal (motor portion) and hypoglossal nerves, and to actual bulbar paralysis; furthermore, occasional atrophy of the *Gasserian ganglion* and of the *trigeminal nerve* has been found in the same symptom-complex; finally, degenerations of the pneumogastric, *spinal accessory,* and *recurrent laryngeal* have been noted in paralyses of the muscles of the larynx. All of these conditions, however, are quite obvious.

Two of the most constant and important symptoms of tabes are still to be analyzed and localized: *reflex pupillary rigidity with myosis,* and *ataxia.* Both have been the subject of comprehensive study and much discussion.

Reflex pupillary rigidity and *myosis* are two independent phenomena, but they occur in association with extraordinary frequency. The former usually precedes the latter, therefore more frequently occurs alone, or may long be an initial symptom. The anatomical localization of *reflex pupillary rigidity* may very easily be determined theoretically, but practically it has not yet been demonstrated. It is clear that this disturbance cannot be located in the *peripheral optic* (centripetal pupillary), nor in the *peripheral oculomotor* (centrifugal pupillary), tracts; it must therefore be sought in an intermediary tract, probably in the first optical, and its adjacent oculomotor, centers, or in a special center. This was supposed to be due to changes in the central gray of the cavities or in Westphal-Edinger's nucleus, and the region of the ciliospinal center in the cervical cord was also formerly thought of; the ciliary ganglion too, for weighty reasons, has been considered. Recent investigations, however, have shown such manifold localizations, and so many modes of development, as to make it almost impossible for us to obtain accurate knowledge

of these conditions. The researches of Bach[1] clearly portray these difficulties and obscurities. It is impossible for us here to discuss this question, which has engaged the attention of Majano, Bernheimer, Bach and H. Meyer, Cassirer and Schiff, Ruge, Levinsohn, Axenfeld, Uhthoff, v. Hippel, and others; moreover, it is unnecessary, since no conclusive opinion is possible, and the condition remains obscure.

It is now assumed that there are contained pupillary fibers in the optic nerve which produce a reflex, and these are subsequently detached from the original optic tract (in the neighborhood of the geniculate ganglion). Nothing is more obvious than the assumption that primary degeneration of *these* fibers (which may be regarded as analogous to the posterior root fibers and their reflex collaterals) produces pupillary rigidity; but it was found in reflex pupillary rigidity that if the optic tract was clear, as is usually the case, the nerve of sight was absolutely normal. Moreover, no conclusive clinical or anatomical reason could be assigned for the localization of the lesion in the region of the corpora quadrigemina, the nuclei of the oculomotor nerve (Westphal-Edinger's nucleus, etc.); therefore this localization was not accepted. This is opposed by anatomical, and chiefly by experimental, investigations (particularly those of Bach and H. Meyer), which clearly show that there are anatomical relations between the medulla oblongata—that is, the upper portion of the cervical cord—and the reaction of the pupils.

Bach's experiments first led him to assume a *reflex center* in the *upper cervical cord*, also an *inhibitive center* at the spinal end of the fourth ventricle near the median line for the reflex of light (contraction of the pupils), as well as for dilatation of the pupils, and he attempted to explain the tabetic reflex pupillary rigidity, as well as the myosis, by a persistent irritation of this inhibitive center. He is not exactly enthusiastic over this explanation, and it appears to me that the assumption of an *irritation* of these centers which lasts for years and decades, and which never produces paralysis, controverts everything that we know of tabes and its anatomical foundations; therefore it is absolutely inconclusive.

Bach found by somewhat contradictory experiments that *division* of the cervical cord somewhat below the medulla oblongata leaves the pupillary reaction intact; *destruction* of the same close to the medulla at once arrests the light reflex; *section* of the medulla at the spinal end of the fourth ventricle immediately results in rigidity to light; *irritation* of the medulla at its spinal end also causes rigidity to light with myosis, which, however, *disappears at once* if the medulla is divided in the center of the fourth ventricle, or even somewhat higher. In my opinion these findings permit the conclusion that an important, and not a subordinate, reflex center for the pupil is situated anteriorly and above the medulla, but that the main reflex center is to be found in the upper cervical cord; further experiments will decide. Most of these researches were made in cats, and, considering the peculiar pupillary reactions of these animals, they can be applied to man only with the greatest reserve.

The fact that, in a few cases of reflex pupillary rigidity, *changes in the posterior column in the upper cervical cord* have lately been found confirms

[1] L. Bach, "Pupillenstudien." *v. Gräfe's Arch.*, 1903, LVII.—"Was wissen wir über Pupillenreflexcentren," etc. *Zeitschr. f. Augenheilk.*, 1904, XI.

Bach's views. Reichardt (1904) has even declared them to be almost invariable, and he locates them in the ventral part of Bechterew's intermediate zone at the height of the second and third cervical segments.

The changes which Marina found in the ciliary ganglion in tabes and paresis must be further investigated to demonstrate their significance.

Taking all into consideration, we may state that the anatomical lesion which is the cause of reflex pupillary rigidity is not yet positively known, but that it is *most likely to be found in the posterior columns of the cervical cord close to the spinal end of the fourth ventricle.*[1]

The same is true of the common, but by no means invariable, accompanying *myosis*. Since cocain causes a prompt dilatation of the pupil, we cannot unreservedly attribute this myosis to a paralysis of the sympathetic fibers, although this is the most likely cause. Bach explained it as due to an irritation of his hypothetical reflex inhibitive centers for contraction and dilatation of the pupil. The absence of these two reflexes produces myosis, because the tonus of the stronger sphincter is decidedly greater than the tonus of the dilator. Neither has this view been as yet confirmed.

At all events the mechanism of pupillary movements and the tonus of the pupillary muscles appear to be much more complicated than was previously supposed; by continued research, especially in tabes, some unexpected conclusions may be reached.

Ataxia still remains to be described, and this is undoubtedly the most interesting, as well as the most difficult, chapter in the pathological physiology of tabes.

Since the accurate formulation of Duchenne's conception, endless discussions have taken place and innumerable clinical observations and animal experiments have been made, without, however, yielding any definite results, nor leading to a final unanimity of opinion in place of these diametrically opposite views. The nature and pathogenesis of ataxia are primarily of most theoretic importance for the normal and pathologic physiology of the spinal cord; but they have recently become of practical significance because the relief of this symptom dominates the treatment. We would digress too far were we to make at this point a comprehensive investigation of the question of ataxia. Volumes could be filled. We shall therefore attempt to be brief.

As a rule, we understand by *ataxia* the *disturbed coördination of voluntary movements*; but even the conception "coördination of movements" is difficult to understand. We may say that for either simple or complicated movements, also in steadying the extremities and the trunk for the purpose of maintaining the equilibrium, for balancing, for maintaining certain positions, etc., carefully graduated innervation of more or less numerous muscles is necessary, provided that actual *coördinated impressions* with the *least possible expenditure of strength* and *minimal sensory impressions* is to result. For this the correct choice of the many innervated muscles and muscle groups, the correct *degree, rapidity,* and *sequence* of their innervation (including the

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1 Compare W. Uhthoff, "Augenveränderungen bei Erkrankung des Nervensystems," in *Gräfe-Sämisch's Handb. der Augenheilk.*, 1904, XI, 2. Aufl.; and Bumke, "Die Pupillenstörungen bei Geistes- und Nervenkrankheiten," Jena, 1904.

antagonists) is necessary. If, in any of these essentials, there is even a slight disproportion, "disturbance of coördination" results.

This faculty of coördinating innervation is chiefly the result of long-continued practice in the accumulation of *certain* impulses of movement, which are then gradually exerted upon a certain tract, and the conjoint effect of impulses of the will is the result; but we assume that these are dominated by certain parts of the central apparatus which are designated *coördination centers*, these having for a long time been referred by physiology to the brain (the trunk of the brain, especially the cerebellum; some of them recently to the cortex of the cerebrum). It has been determined that the existence of such centers is absolutely necessary for the impulses by which we maintain the equilibrium and the erect posture in space. They may perhaps be only areas for the transmission of stimulations of the will, only a meeting point for the various innervation tracts, an apparatus for the reception of centripetal sensory stimulations, and a point of communication between these centripetal and centrifugal tracts; but for the comprehension of fine and complicated processes they can scarcely be dispensed with.

These so-called "centers" apparently act upon the motor apparatus only through centrifugal tracts—the spinal and peripheral conduction tracts and muscles—which may possibly unite in the brain or the pons, some of them perhaps in the spinal cord, with the direct motor conduction tracts. Apparently the spinal cord itself does not transmit combinations of voluntary movements, but by means of *complicated reflexes*, and by the assumption of so-called "coördinated reflexes," which are to be differentiated from the other spinal (tendon and cutaneous) reflexes, this is not inconceivable.

The exact relations of the centers of the will to these coördination centers are still obscure; undoubtedly the latter are influenced and set in motion by the will, but a clear conception of this is impossible.

Moreover, there are numerous *centripetal impressions* and stimuli which reach the apparatus of coördination from the periphery, and stimulate or modify its activity for every possible kind of movement; impressions which transmit to the external world the relations of the body, the relations of the internal parts of the body to each other and to the central organ (by no means always the organ of consciousness!), every possible position of the joints, the starting-point of movements and much besides, and thus aid greatly in the regulation of voluntary and involuntary movements—their production, duration, and increase, as well as their decrease, or entire inhibition. Among these are the centripetal stimulations originating from the sensory nerves of the skin, of the joints, of the muscles, of the tendons, of the ligaments, etc., some not extending to consciousness at all, and some being active only reflexly. Some of the areas from which these centripetal stimulations are transmitted to the motor ganglion cells and conduction tracts may be situated in the spinal cord ("spinal coördinative reflex tracts"), some in the cerebellum ("cerebellar coördination"), some in the cortex of the cerebrum, or in the motor cortical regions ("cerebral coördination"). To this must be added the important sensory stimulations which either by the optic tract and the optic centers, or by the semicircular canals of the labyrinth of the ear and the tract of the auditory nerve (vestibular nerve) exert a powerful influence upon the coördination of movements; these are the visu-cerebral and the ves-

tibulo-cerebellar centripetal mechanisms, which are undoubtedly of vast importance for coördination.[1]

Centripetal sensations are absolutely necessary to explain the *nature* of most of the complicated movements. The child at birth is in truth ataxic, and it learns its first coördinate voluntary movements, such as touching, grasping, standing, walking, and those involved in speech, solely by persistent attempts at movement, and by exercises which are regulated by the cutaneous, arthritic, and muscular sensibilities of sight and hearing. For the rapid and apparently automatic movements which have been acquired—manipulations, walking, writing, piano-playing, etc.—this regulation seems to be no longer necessary, or only to a slight degree; there is no longer time for it, especially if the movements are performed quickly or by a single energetic action (such as throwing, jumping, etc.). But accurate analyses, such as Frenkel and O. Förster have made, of these various movements show that both movement conceptions and coördinate movements are invariably dependent upon certain centripetal impressions. If these are new, unfamiliar, or rarely experienced, the movements in the normal person will again resemble the incoördinate movements of the child or the tabetic (for example, in walking upon rough or strange paths, slippery ice, narrow stairs, in the dark, etc.).

To maintain the equilibrium and an erect posture in space (*balancing* the body, especially the trunk, under various circumstances) these centripetal impressions are probably *always* necessary; they regulate most accurately and rapidly, and entirely without the exercise of the will, the numerous innervations of the muscles of the trunk, of the vertebral column, and of the legs, which are necessary for the performance of these acts. The slightest disturbance of these sensations (for instance, of the muscular sense) on closing the eyes (Romberg) betrays itself by faulty and uncertain muscular action in the form of swaying.

Frenkel recently demonstrated conclusively that the maintenance of the normal muscular tonus, particularly in certain muscle groups of the legs and vertebral column, is decidedly important for the correct coördination of voluntary movements. That certain *reflex effects* may also be instrumental in the performance of these complicated functions is clear, but we cannot minutely discuss them at this point.

It is therefore obvious that a *disturbance in the coördination* of movements may take place in very different areas, tracts, and centers. In addition to disturbance of the organs of volition, derangement of the *coördinating central apparatus* (anomalous stimulation and activity) comes into question; first, disturbances in the *centrifugal coördinating conduction tracts* originating from them, some of which are still hypothetical; next, those disturbances (especially a decrease of the stimulus conveyed by the *centripetal sensory*

[1] The ingenious diagram of Otfried Förster, which clearly explains the *centripetal* coördinative stimulations and transmissions, gives us a comprehensive, although complex, picture of the extremely involved processes of coördination which here take place. Unfortunately we have no *proof* of their actual existence and correctness; much contained in the diagram is hypothetical. But it must be admitted that it furnishes a plausible conception, and still leaves room for the assumption of *centrifugal* coördinative tracts which appear to be indispensable for the conduction of stimulations, especially the cerebello-fugal and cerebro-fugal, in the processes of coördination.

nerves) some of which are transmitted by paths in the spinal cord, some in the cerebellum, and some in the cerebrum; then disturbances of *certain reflexes*; finally, disturbance of the *muscle tonus, hypotonia of the muscles*, which is invariably present, and is certainly of vast significance in regulating the measure of individual movements.

Obviously the question now arises *which* of these disturbances is especially responsible for *tabetic ataxia*, whether it is only *one*, whence the ataxias would be of *uniform origin*, or, if *several act simultaneously, which* are they, and do they act in different combinations. Our entire clinical and anatomical knowledge of tabes at once makes it evident that we are dealing with complicated and obscure processes, which in different cases may vary greatly or may be combined. Apparently complex conditions act together, these being modified by different circumstances, and dependent upon a number of conditions and coincidences.

Actual disturbance of the *coördinating centers* in tabes, which is regarded as a disease of the *spinal cord*, is scarcely likely. It is true that a form of ataxia occurs in disease of the cerebellum, also of the pons (the so-called *cerebellar ataxia*), and of the corpora quadrigemina—perhaps also in disease of the frontal brain. But both in its nature and symptoms it distinctly differs from *spinal*, tabetic ataxia; therefore it need not be considered. Theories which attempt to refer tabetic ataxia to certain lesions in the cortex of the cerebrum and cerebellum have been rejected, and must be *regarded as untenable*.[1]

We must now investigate *true tabetic spinal ataxia*. For its form and mode of expression, as well as for a number of important details, we refer the reader to the description in Frenkel's book[2] which forms an original and comprehensive study of the whole subject of ataxia and its associated conditions, varieties, and complications; for more specialized instruction the work of Otfried Förster,[3] which treats the entire condition most exhaustively, is valuable. This author first studied each individual joint and each individual muscle group of the lower extremities—some for the relation of the "different factors"—namely, sensation, tendon reflexes, tonus, passive tension, etc.—then the behavior of the muscles as agonists, agonistic synergists, as antagonists (that is, also as collateral and rotary synergists), finally the resulting anomalies in position and movement of the affected portion of the members. He described and analyzed these, and was inclined to attribute them to a simultaneous disturbance of centripetal irritation. He then analyzed similarly the processes of standing, walking, rising, sitting down, ascending stairs, etc. Subsequently he examined in the same way the muscles of the trunk, the individual muscle groups, and the joints of the upper extremities, particularly during the act of writing; finally, the muscles of the head, face, tongue, larynx, and eyes.

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1 Time will show whether the communications of C. Weigert (published after the untimely death of the author) in regard to invariable changes in the molecular layer of the cerebellum are of any value.

2 H. S. Frenkel, "Die Behandlung der tabischen Ataxie mit Hilfe der Uebung." F. C. W. Vogel, Leipzig, 1900.

3 Otfried Förster, "Die Physiologie und Pathologie der Coördination." G. Fischer, Jena, 1902.

This author compiled an enormously rich mass of material which shows how many-sided and complicated, and how pathogenetically different, the ataxic movements are. His investigations will bear close study, and are certainly invaluable for the sensory theory of ataxia.

If we remember that tabes is essentially a disease of the spinal posterior columns—the spinal root areas—and therefore represents centripetal conducting tracts, that theory will appear to be the most rational which deduces tabetic ataxia from a disturbance of the centripetal sensory conduction tracts. Frenkel, who has recently made a most thorough study of its clinical aspects, states that *ataxia is the reaction caused by disturbances of sensation in the arthritic and muscular systems,* and to some extent in the skin.

This is the *sensory theory* of ataxia which was first promulgated by Leyden in 1863, and has since then been repeatedly and zealously advocated, being somewhat modified by numerous clinical and experimental facts.

This theory is chiefly based upon clinical observation, upon the apparently invariable disturbance of cutaneous sensibility, of conscious and unconscious sensations, above all of the sensations conveyed by the joints, muscles, and tendons; this Goldscheider and others stanchly maintain. The constancy and proportional relation of these disturbances to ataxia have been, and are still, doubted. Frenkel has recently strengthened this view by asserting that tabetic ataxia is usually combined with a derangement of *cutaneous sensation, always* with disturbance of *arthritic sensation,* and in *severe* cases with disturbance of *muscular* and *tendon sense,* and he declares that the assumption that *tabetic ataxia* is *not accompanied by sensory disturbance* depends upon erroneous and incomplete researches. He further declares that the degree of ataxia is *always* in proportion to the degree of sensory disturbance; that is, the most markedly hypesthetic joint, the extremely hypesthetic extremities, also show the most profound ataxia. This author refers the statement that *distinct sensory disturbances are occasionally present in tabes without ataxia* to inaccurate deductions (for the investigation of ataxia he has proposed a series of exceedingly minute tests!).

An effort was made to base this theory upon experimental findings. For these sections were made of the posterior roots of one or more extremities, either unilateral or bilateral, in frogs, dogs, and recently in apes; with regard to the questions to be solved, there has been an increasing refinement in the operation.

It is impossible here to discuss minutely these miscellaneous investigations; it is evident that only those performed upon apes will apply to human pathology; and here we refer the reader to the numerous subsequent investigations of H. E. Hering, Mott and Sherrington, A. Bickel, P. Jacob and A. Bickel, Korniloff, Merzbacher, and others, from which it is at least certain that the arrest of sensation after the severance of the posterior roots results in a decided disturbance of motility. Whether this is related to tabetic ataxia, or perhaps identical with it, is difficult to prove. It is certainly not true in the case of the lower animals; in dogs it must be accepted with some reserve, and probably also in the case of apes.

It is not my purpose to discuss these investigations at length. I am under the impression that they substantiate the sensory theory of ataxia, although many doubts arise, and under minute criticism, some of the findings are

susceptible of other interpretation. It is remarkable that many of these examinations have shown that the disturbance of motion after some time disappears, although there is never reestablishment of the sensory conduction tracts. Vicarious compensatory stimulations appear to be set in motion (by way of the collateral sensory tracts, or of the auditory and optic tracts, from the sensory sphere of the cortex of the brain, or the like), and after their suspension "ataxia" once more manifests itself.

To the adherents of the sensory theory the occurrence of ataxia (or, more correctly, a disturbance of motion resembling ataxia) in *peripheral neuritis* —so-called *neurotabes peripherica*—was of great significance, and quite properly so; but only when actual sensory disturbances existed, and secondary changes in the central nervous system were absolutely excluded. These conditions were certainly difficult to prove.

The foregoing facts, combined with the thorough consideration of the sensibility of the skin, of the joints, of the muscles, and of the tendons, make it appear extremely plausible that what we call tabetic ataxia chiefly depends upon sensory disturbances. It is certain that when there is anesthesia of the soles of the feet their position and movements upon the floor become uncertain; that with decreased sensation in the joints the excursus of their movements becomes more extensive and rapid, so that the production of movement is apparent to the consciousness; that for the same reason when muscle and tendon sensations are decreased the movements become uncertain, exaggerated, more marked, or weaker; that with a defective appreciation of the starting-point for the members the intended motions are incorrect, misdirected, or accelerated; that individual movements for which the tabetics have lost the necessary appreciation of sequence, knowledge necessary for movements made with a definite purpose, are grossly impeded, etc. It is doubtful, however, whether these facts sufficiently explain the fully developed ataxia of tabes. We know that disturbances of sensation, in the widest sense of the term, are compensated for by sensory control, especially on the part of the eyes; but tabetic ataxia in a greater or less degree unquestionably appears when the eyes are open, and there is rapt attention.

Some adherents of the sensory theory appear to have noted this weak point in its construction; and one of its most earnest supporters, Frenkel, has quite properly utilized a significant factor to explain many of the ataxic phenomena: *hypotonia of the muscles*, which this author has carefully studied. He holds this responsible for the jerky, aimless, irregular movements of tabetics, for the conspicuous disturbance of the gait, for the anomalies in the most important movements of the trunk when walking, and much besides. Are we justified in referring this hypotonia to the sensory centripetal disturbance? This seems doubtful! Is the hypotonia to be chiefly attributed to a *reflex* disturbance of tonic stimulation? It would seem so! But Frenkel has shown that the hypotonia, which he declares to be the *initial* and almost pathognomonic symptom of tabes, is not strictly parallel with the disturbance of sensation or with the reflex disturbances. In his opinion, *ataxia is by no means the consequence of hypotonia,* which may exist without ataxia—but it undoubtedly has an influence upon the form of the ataxia.

Otfried Förster, the latest investigator of this subject, after most comprehensive studies and close consideration, comes to the conclusion that tabetic

ataxia is due to a disturbance of the sensitive stimulations ("factors") which are indispensable for coördination, of the "subcortical" (spinal, cerebellar, that is, unconscious) as well as "cortical" (conscious) sensations.

Hence he finds the *anatomical foundation* for ataxia to be a degeneration of the reflex collaterals in the spinal cord—the collaterals passing to Clarke's columns which penetrate to the cerebellum—and of the long posterior column fibers (for the cerebrum); its *pathologico-physiological foundation,* however, is a disturbance thereby produced in the *spinal sensitive factors* (arrest of the tendon reflexes, the coördination reflexes, the spinal reflex tonus), in the *cerebellar factors* (arrest of the cerebellar reflex tonus), and in the *cerebral factors* (conscious sensations). These may individually or collectively be unequally affected and thereby produce different degrees and modifications of tabetic ataxia.

Plausible as these deductions seem, and rational as the sensory theory of ataxia thus formulated appears, many doubts still confront the candid investigator. Many have never accepted this theory, and some have designated it as untenable. Friedreich did this, upon the basis of his minute researches in "hereditary ataxia." Other authors as well as I (in my text-book) followed him. It is true we know that this theory is suggested for a disease which must be sharply discriminated from tabes, and that hereditary ataxia differs in many ways from true tabetic ataxia. This may be accounted for by the fact that in Friedreich's ataxia marked disturbances of conscious sensation do not appear for a long time; yet undoubtedly "ataxia" exists, and—what is of especial importance—also gray degeneration of the posterior columns, so that, from a scientific standpoint, tabetic ataxia cannot at once be eliminated, as the adherents of the sensory theory so naively require us to do.

For true tabes a number of circumstances may be urged in opposition to the unrestricted adoption of the sensory theory, although this, as now becomes apparent, is not based *merely* upon disturbance of conscious sensation. We need only mention the following: the occasional occurrence of ataxia *without* any evident sensory disturbance, and *the absence of ataxia with distinct disturbances of sensation,* this being not seldom observed in the so-called preataxic stage; the *late appearance of ataxia,* in contrast to the other symptoms, when there is most certainly a lesion of the posterior root tracts; and, when both are present, the common *disproportion between the degree of sensory disturbance and the ataxia.*

These reasons formed sufficient ground for the opponents of the sensory theory to reject it; for it is quite evident that the concurrence of sensory disturbances and ataxia by no means proves that the latter is produced by the former; they may be parallel, but entirely independent, symptoms of ataxia; but every case in which ataxia without sensory disturbances can be certainly proven, and also every case in which distinct sensory disturbances appear without ataxia, must help to shatter the sensory theory. Frenkel has attempted to nullify these objections by the supposition that the absence of sensory disturbance in the one case, as well as the absence of ataxia in the other, is only a seeming one, such absence not having been observed by the investigator because the examination was not thorough, and owing to the assumption that sensory disturbances and ataxia are always in exact proportion to each other. I must, however, express my doubts of the correctness of these views, and

shall suspend my opinion until these statements have been confirmed by others; unfortunately want of time has so far prevented me from investigating them, but I shall devote my attention to them in the near future. We are greatly indebted to Frenkel for formulating correct methods for testing sensation, ataxia, and hypotonia in tabes.

As an additional objection to the exclusive importance of the sensory theory we may briefly mention the *persistence of ataxia during sensory control by the eyes* (as well as by the semicircular canals of the labyrinth); these cases, in which, when sensory "factors" are absent, the optical control is insufficient to prevent ataxia, have lately been especially emphasized by O. Förster. He expressly points out in such cases the apparent *dissociation of motor impulses* in primary voluntary innervation which is quite independent of the secondary sensitive factors, the exaggerated, aimless, waddling movements in all extreme ataxia in spite of well-retained motility and coarse power, and the presence of compensatory sensory control; the undoubted appearance of profound ataxia, not unlike the spinal form, in diseases of the cerebellum, of the trunk, of the brain, etc., without any sensory disturbance (the same condition being noted in Friedreich's hereditary ataxia). He also maintains the development of extremely severe *spinal sensory disturbances* in other diseases of the spinal cord (syringomyelia) *without* any trace of ataxia; the familiar cases of Späth-Schüppel, Engesser, and others, cannot so readily be ignored! Finally, the want of unanimity and the unquestioned *rarity of ataxia in neuritis and polyneuritis* (so-called neurotabes peripherica)—in my long practice I have certainly seen numerous cases of polyneuritis, yet no typical case of this kind was ever observed—lead me to think there must be something else, some central or other change, which in neurotabes peripherica (which usually depends upon alcoholism) certainly cannot be excluded, but is very difficult to demonstrate.

In the decision of this question too much importance must not be attached to animal experiments. The deranged motion produced bears at most but a very indistinct resemblance to tabetic ataxia, and the results in animals, whose movements of the limbs are much more automatic than those of man, cannot readily be applied to human pathology; this is usually permissible only in experiments upon apes, in which, it is true, the results are quite remarkable.

Therefore the sensory theory of ataxia is even to-day incomplete; at all events, tabetic ataxia cannot be *exclusively*, and in a wide sense, attributed to tabetic sensory disturbances, although we do not absolutely reject this standpoint.

We must, therefore, seriously consider the possibility of a motor *theory*, according to which *disturbances in the centrifugal* (so-called coördinating) conduction tracts are held responsible for the ataxia, the so-called coördinate centers being combined with the peripheral motor neuron. These tracts must be situated somewhere in the spinal cord. But where? This we do not know, and hypotheses amount to little. It is certainly conceivable that by the tabetic process, irritation and degeneration in these tracts, which usually appear only in the further course of the disease—influence "coördination" of movements and more or less decidedly disturb it. We must view the disease from many standpoints in order to ascertain the components of ataxia. In the present state of our knowledge we are forced to admit the *possibility*, and even the *likeli-*

hood, of such a process. That the motor theory of well-developed ataxia combined with degeneration of the posterior columns is the only tenable one, at least for Friedreich's ataxia, appears to be established. In how far true tabetic ataxia is due to this cause remains a mooted question. We believe the *motor theory* to be more hypothetical and less well founded than the *sensory theory*, but, as it cannot be absolutely denied, the subject must be further investigated.

Special consideration of the *reflex theory* of ataxia is superfluous. That it bears no close relation to disturbances of the ordinary cutaneous and tendon reflexes no one doubts. What other factors may be operative in these unknown reflex influences—hypothetical "coördination reflexes," etc.—has been discussed under the sensory theory, and in our remarks concerning the influence of hypotonia; certainly disturbance of the reflexes *alone* will not fully explain tabetic ataxia.

This shows that our knowledge of ataxia is still obscure; probably there are complicated conditions which cannot be understood from one point of view; it may be that sensory disturbances, especially of the joints, the muscles, and the tendons, play a rôle. But later disturbances in the motor (coordinating) conduction tracts cannot be excluded, and hypotonia of the muscles may play an especially important part in ataxic motor disturbances. In how far these depend upon centripetal reflex disturbances, or whether or not they are to be reckoned among the motor disturbances, therefore are of vast significance for the motor theory, cannot be stated.[1]

At all events, ataxia is one of the most interesting symptoms in the rich clinical picture of tabes, and one of the most difficult to explain; it furnishes to physiologists, neurologists, and experimental pathologists many problems which await elucidation.

VI. COURSE, TERMINATION, AND DEFINITE FORMS OF TABES

The *course* of tabes varies greatly in different cases; usually one type predominates, forms the rule, and is found in the majority of cases, although with many modifications and peculiarities (the typical form of tabes). We have attempted to portray this variety in the description of the symptoms.

It may be simply divided into three stages: First, the *initial neuralgic stage*, the most important objective and subjective symptoms of which have been described above, including a number which are rare. The affection has a chronic and slowly progressive development. The first symptom is usually *lancinating pain* which is more or less frequent and intense, is usually regarded by the patient as rheumatism, to which but little attention is given; by and by other symptoms appear (paralysis of the muscles of the eyes, disorders of sight, paresthesia, fatigue, weakness of the bladder and sexual functions, etc.); the affection may actually begin with any of these symptoms, but others invariably follow, and may be complemented by many rare phenomena (gastric and laryngeal crises, arthropathies, partial paralysis, etc.), while objective

[1] In regard to this Strümpell's interesting report may be consulted: "Ueber die Störungen der Bewegungen bei vollständiger Anästhesie eines Armes," etc., in the *Deutsche Zeitschr. f. Nervenheilk.*, Bd. XXIII, p. 1, 1902.

examination reveals typical symptoms in the pupils, the tendon reflexes, in sensation, etc.

Just as the disease begins slowly and insidiously, so it progresses, usually becoming aggravated; but decided improvement, a long arrest, or rapid aggravation, may take place. With the gradual or rapidly developing disturbance of coördination which soon or late occurs, the patients reach the *second*, the *ataxic*, *stage* of the disease, and here a slow but progressive aggravation is the rule. For a long time ataxia is limited to the legs, then it gradually involves the arms, while the legs become so ataxic that the patients require a cane, or two canes, or crutch support; finally they are compelled to use a rolling chair. At the same time the symptoms of the first stage usually increase in severity the pain becomes more intense, the attacks more frequent, the paresthesias, the bladder disturbance, the sexual weakness, etc., increase, and new symptoms appear; yet the constitutional condition may for a long time continue good, or from the onset may reveal severe disturbance.

After a long but somewhat variable duration of this stage, in which the patients are often still able to follow their occupations, the mental powers being quite active, they become more helpless. Ataxia of the hands renders these incapable of use, the extreme ataxia of the feet, to which paralysis, atrophy, and contracture are soon added, prevent the use of the legs, and the patients are confined to a rolling chair or to bed, while the general condition begins to suffer seriously. This is the *third stage* of the disease, usually designated the *paraplegic*, although true paraplegia, actual paralysis, is not always present, but only an extreme degree of ataxia, with anesthesia, debility, atrophy, and contracture of the legs. The distress of the patients is usually increased by their helplessness, by pain, and the visceral crises, by paralysis of the bladder to which cystitis is soon added, by bed-sores, and by general cachexia, until, in different ways which are soon to be mentioned, death relieves the miserable sufferer.

Each of these stages may last a long time, especially the first two, which are sometimes prolonged through years or decades, so that the disease may last 25 to 30 years, or even longer; sometimes death takes place much sooner—in from 5 to 10 years. It is difficult to prognosticate concerning this point.[1]

In regard to rapidity of development, there are marked deviations from this course; in the first place, some cases are so exceedingly slow that the patients never progress beyond the first stage of the typical symptoms, hence we might almost speak of an abortive form of tabes; I have had under treatment for several years a patient, in whom Charcot in 1881 made a diagnosis of tabes, who presents all the classical symptoms of the first stage (spinal myosis, absence of the tendon reflexes, lancinating pains, laryngeal crises, preceding syphilis), yet who even to-day is not ataxic; another patient with prior syphilis has had lancinating pains for 26 years, with transitory paralysis of the muscles of the eye and of the bladder, but even now the tendon and pupillary reflexes are retained, and show no sign of ataxia. Similar cases of apparently very *benign* and insidious types have been reported in literature.

In contrast to these are the extreme cases in which the different symptoms

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[1] Pierre Marie has proven by statistics that about 83 per cent. of tabetics attain the age of 50 years, 51.5 per cent. over 60 years, and some 75 to 80 years.

of the affection appear in rapid sequence, the first stage lasting but a few weeks or months, being quickly, we might almost say in a night, followed by a high-grade ataxia; the patients soon become helpless; as a rule, there are extreme weakness and decided constitutional disturbance. These cases with *rapid course* are not rarely characterized by a profusion of symptoms, a combination of rare phenomena, of the various crises, arthropathies, etc., as well as an *aggravation* of the ordinary symptoms. They may be aptly designated the *malignant* forms of tabes; upon what this course depends we do not know.

Besides these chief forms of the disease—the ordinary, the benign, and the malignant—other *varieties* of tabes are readily differentiated by the symptom-complex of the individual case. In my text-book (1878) I enumerated such types, and Déjérine and Collins have recently described quite a series. This is of practical value, because the type appears to have a definite influence upon the course and duration of the affection. We shall, therefore, briefly sketch a few of them.

Types of Tabes.—First, there is a form in which from the onset *lancinating pain* forms the chief symptom of the affection (*tabes dolorosa*), the intensity often reaching an extreme degree, the painful attacks being very frequent, while the other symptoms are mild or wholly secondary; but the opposite also occurs, and represents one of the most severe forms, for in this the patient almost invariably becomes addicted to morphin.

Closely related to this form is another in which the sensory disturbances, such as *disagreeable paresthesia* and *almost constant pain*, become prominent (*tabes paresthetica*). Such patients often complain of a disagreeable girdle sensation, as though the trunk were enclosed in armor, there is difficulty in breathing, persistent and irksome sensations in the back; very often the trunk shows marked hyperesthesia to cold. In this form lancinating pains, gastric and laryngeal crises, and similar conditions are frequently noted; the remaining symptoms vary. In this type it is a question whether the meningitic irritative symptoms, the "syphilosis" of the meninges, do not form the most prominent feature. In other cases *motor disturbances* are most evident, the sensory being less marked. Such patients are either decidedly ataxic or they soon present paretic and paralytic disturbances (*tabes atactica* or *tabes paralytica*).

An especially important form is that in which *atrophy of the optic nerve* appears as the first and earliest symptoms, and with it *amaurosis* (*tabes amaurotica*). If the amaurosis is the earlier or prodromal symptom, the other symptoms are remarkably mild; the sensory disturbances are usually slight, but they can be objectively demonstrated, the tendon reflexes are absent, pupillary rigidity exists, and—a remarkable fact—in spite of almost total blindness, sometimes Romberg's symptom is present; such patients usually do not become ataxic, or only after a very long period of time; therefore the course of the disease is prolonged but mild. Benedikt, Charcot, Déjérine and Martin, Marie and Scwitalski, and others, have described and thoroughly discussed such cases, and I have seen several. What produces them cannot be positively stated, and I shall not enter upon hypotheses. If optic atrophy appears in the later course of tabes, it seems to have no influence upon the course of the affection. Benedikt maintains that, under these circumstances, the ataxia is less marked; but, according to Marie and Léri, this is frequently not the case.

In other instances *visceral symptoms,* gastric and other *crises, bladder symptoms,* and the like (*tabes visceralis*) are more prominent; it is reported that these are frequently coincident with *trophic disturbances,* arthropathies, spontaneous fractures, etc., so that the extensive implication of the sympathetic and its spinal roots is suggested.

Special significance attaches to a form of tabes which begins in the *upper portions of the spinal cord,* being localized in the *cervical cord,* not rarely somewhat higher, and implicating the bulbar and cerebral regions (*tabes superior, cervicalis, bulbaris*): the first symptoms are noted in the upper extremities (lancinating pains, ulnar anesthesia, and ataxia of the hands), and to these laryngeal crises and posticus paralysis, bulbar symptoms, atrophy of the tongue, a difficulty in mastication, paresthesia of the face and of the neck, nuclear ocular paralysis, etc., are added; tabetic symptoms in the legs appear later, and are not so prominent. It becomes obvious that, with these complications, the course is more or less unfavorable and more rapid. Another type, *tabes infantilis* or *juvenilis,* such as occurs in children from 5 to 10 years of age, or in adolescents from 13 to 18 years of age, calls for a special description. In these cases there is almost always a history of hereditary syphilis, or of tabes and paresis among the brothers and sisters, or among the ascendants. In most cases the classical symptoms of tabes appear, while sensory disturbances and ataxia are relatively less conspicuous. Unfortunately, there are as yet no autopsy reports of such cases, but a great number of them have been clinically diagnosticated.

Finally, we must mention another important symptom of tabes which is associated with severe changes in the brain, as a rule with *paresis,* and to which we have previously referred (*taboparalysis*). That this complication, which is common in the early stages of tabes and rare in the later, influences the course unfavorably and greatly shortens life, is obvious. It is unnecessary to describe its symptom-picture.

The number of these "types" may be easily increased; in spite of all its peculiarities, to say nothing of the distinctiveness of its fundamental type, tabes is an extremely many-sided disease, richer in symptoms than any other, and I wish expressly to emphasize that the types here described are not conclusive, but they form transitional stages and manifold gradations from one fundamental form; they are very different from those irregular forms of tabes which are *transitional stages* of the combined system diseases, or which *are combined* with other syphilitic diseases of the spinal cord and of the brain (with meningitis, meningomyelitis, meningoencephalitis, endarteritis, etc.). Their description would lead us too far from our subject. But it must be expressly stated that these varieties form a small minority in comparison with the regular, typical, fundamental forms of tabes.

Except for the relatively few cases in which tabes is of rapid *development,* its *course* is extremely chronic, slow, but progressive; i. e., the disease shows a tendency to slow aggravation. But it must be borne in mind that to this there are numerous exceptions. Decided variations and more or less improvement may appear, the condition of the patient may change with the seasons, improvement may lead to a prolonged arrest of the affection, or to a condition which is almost absolute recovery, so that the patients may be practically regarded as cured, being able to follow their occupations, to enjoy

life, and to fulfil its duties; such arrests may occur even in the ataxic stage, and the patient, who had been almost confined to a rolling chair, may find that his disease remains stationary for many years; not infrequently, however, these arrests are disturbed in a most undesirable way by the effects of extraneous factors (by exposure to cold, excitement, over-exertion, sexual excesses, by unsuitable and exhausting therapeutic measures, such as hot baths, too prolonged use of the gymnastic method, and the like, often by influenza, occasionally by other intercurrent diseases), and the disease then rapidly progresses toward its terminal stages.

The ordinary *outcome* of the affection is *death*. Not that tabes in itself is always fatal, for during its entire course the vital functions may be exempt. But it always shortens life by the sequels which it produces, by its reaction upon the entire organism, by many complications which are incidentally brought about. In some cases—often after a duration of 25 to 30 years—the disease is fatal because in the third stage it leads to paraplegia, to cystitis and pyelonephritis, or to bed-sores with all of their consequences, and thus the powers of the patient are exhausted, and, after a final stage of coma, delirium, acceleration of the pulse, respiratory disturbance, and great misery, the patient succumbs.

Or the affection itself may cause death. The patients may suffocate from paralysis of the posticus, or from severe laryngeal crises; or they may succumb to the consequences of fulminant gastric crises, either in collapse or from inanition; or the bulbar paralytic symptoms, difficulty in deglutition, or inanition, may have the same effect; or paralysis may rapidly bring about the catastrophe; all these are comparatively rare.

Finally, an incidental and intercurrent disease—pneumonia, influenza, diphtheria, enteric fever, cholera, erysipelas, or pulmonary disease—frequently causes death; or syphilitic manifestations, cerebral syphilis, apoplexy, aneurysm, a lesion of the aortic valve, or the like, may terminate the life of the patient.

But the termination is not always lethal. A few well developed cases of tabes (perhaps 4 to 6 per cent.) terminate in recovery, or, at least, the affection is so far arrested, and the patients so far improve, that they are "practically cured," they no longer have subjective difficulties, are able to perform their work, and to enjoy life in the ordinary way. If the patella tendon reflex is absent, or if the pupils are rigid, this but slightly affects the general condition. I know of a number of such cases, of course not cases of tabes incompleta or tabes abortiva, which are soon to be discussed, in which a permanent arrest of the affection usually occurred spontaneously; hence we can hardly speak in these cases of "cure."

Experience shows that in a certain proportion of the cases of tabes, unfortunately a very small one, actual or relative cure takes place, the duration of which may be limited.

We have heretofore spoken of typical tabes which has a full symptom-picture, the most important subjective or objective symptoms being present in greater or less, but naturally very varying, numbers. Undoubtedly many cases belong to tabes, yet the symptom-picture is not completely developed, and may give rise to doubts. In a short article[1] I some time ago called

[1] "Zur Frühdiagnose der Tabes." *Münchener med. Wochenschr.*, 1900, Nr. 29.

attention to such cases, and designated them as *tabes incompleta* (the French term these "formes frustes"). I have since seen many such cases, and other authors have called attention to them.

These cases are invariably observed in persons who were previously syphilitic, in whom we note prominent *subjective* difficulties (lancinating pain, paresthesia, a sensation of fatigue, slight bladder disturbance, and the like), but we recognize no objective symptoms or these are indistinct (unilateral or bilateral pupillary rigidity, slight hyperesthesia to cold, hypesthetic areas on the trunk or the feet, indications of Romberg's sign, with retained patella tendon reflexes, perhaps unilateral absence of the tendo Achillis reflex and of the triceps reflex, etc.).

In these cases, while all or nearly all *subjective symptoms are absent,* and the persons believe themselves to be quite well (possibly there may be ulnar paresthesia or a slight bladder weakness), nevertheless we find *distinct objective symptoms* of tabes (myosis with reflex rigidity, absence of the tendon reflexes, or the like).

Finally, there are cases in which symptoms not in themselves characteristic, especially the *onset of gastric crises,* dyspepsia, salivation, intestinal disorders, cardiac neurosis, constitutional disturbances or the like, may exist, but examination reveals one or another of the objective symptoms of tabes (most often reflex rigidity of the pupil, paresis of an ocular muscle, unilateral decrease of one or the other tendon reflex, perhaps hyperesthesia to cold).

These combinations of the *individual* subjective, and *individual* objective, symptoms of tabes are very numerous, much more so than was formerly supposed; the habit of investigating *all* cases which are at all suspicious, of examining the pupils and the reflexes, of seeking for Romberg's sign, and preceding syphilis, has shown them to be common.

I do not doubt that these cases all belong to tabes, and that when present in this form they must be grouped as tabes incompleta; moreover, in the last few years, I have often traced their development into tabes completa. In individuals who have previously been syphilitic, we will, as a rule, not be wrong in this assumption. Early in the discussion of the diagnosis we will return to this point, and I shall especially emphasize the value of cytodiagnosis in judging such cases.

There is, however, a group of cases which present even fewer of the symptoms of tabes, perhaps only one or two, and under subsequent and prolonged observation—10 to 20 years—nothing further is noted. These are patients who have had syphilis, but upon examination they show nothing but reflex pupillary rigidity; they have perhaps suffered for years from typical lancinating pains without any objective symptoms, or they now and then have a suspicious gastric attack, but during this time there is no tendo Achillis reflex, and nothing further develops. Following Möbius these cases may be regarded as *abortive tabes,* and, if we choose, we may await their subsequent development. We are certainly justified in considering these cases to be tabes which has become stationary, or has run its course, or, better, to be "syphilosis of the meninges."

But all these observations teach us how extraordinarily the pathologic picture of tabes may vary—from the mildest indications to the severest forms rich in symptoms—and how extremely full and varying is the symptomatology.

They warn us that in tabes, as in so many other diseases of the nervous system, especially in the toxic, infectious, and hereditary forms, we should not be too schematic, but should maintain a free and impartial attitude in considering the nature and appearance of the affection. We may state with some satisfaction that here, under all circumstances, careful clinical observation will yield most abundant fruit.

VII. DIAGNOSIS

In the present state of our knowledge and diagnostic advancement, ordinary typical tabes, even though in the first stages, is not difficult to recognize. If the affection has advanced further, and most of the subjective and objective phenomena are present, non-recognition of the disease can occur only in consequence of gross ignorance on the part of the physician, and a very defective examination. That this even now not infrequently occurs—every year I see cases which for some unaccountable reason have not been correctly diagnosticated—is a fact which mortifies me.

The not uncommon cases in which the affection sets in with some rare symptom, such as a gastric crisis, arthropathy, or weakness of the bladder, are, as a rule, easily recognized on careful investigation if the physician takes tabes at all into consideration.

The typical form of tabes should at once be diagnosticated by every physician. Among its *almost pathognomonic* symptoms the most conspicuous are the lancinating pains, the reflex rigidity of the pupils, the absence of tendon reflexes, Romberg's sign, the initial atrophy of the optic nerve, and the ataxia; less typical yet quite characteristic are the paresthesia, the girdle sensation, the local circumscribed hypesthesia, the analgesia, the retarded pain conduction, hyperesthesia to cold, bladder and sexual weakness, various paralyses of the ocular muscles, gastric crises, and arthropathies.

The diagnosis of tabes may be considered positive whenever we note merely *a few, perhaps only one or two, of these symptoms* (for example, lancinating pain, sensations of fatigue, reflex pupillary rigidity; or lancinating pain and the absence of the tendon reflexes; or weakness of the bladder, reflex pupillary rigidity, or girdle hypesthesia; or arthropathy, absence of the tendon reflexes, pupillary rigidity, etc.).

Naturally in a few cases the diagnosis may be perplexing in the first stages of the affection, or when it irregularly develops as in tabes incompleta, or is combined with other disturbances, partly organic, partly functional; in these cases, the diagnosis may for some time be doubtful or even impossible. If, however, these conditions are kept in mind, and the development is traced with care, as a rule a decision soon becomes possible by the increasing distinctness of the symptoms, or the appearance of new ones. But this is merely what takes place in all diseases, and requires no further explanation.

But the recognition of tabes in its *first stages*, its so-called *early diagnosis*, appears to me to be of the greatest practical importance. This alone permits the favorable influence upon the disease of treatment, an effect which in fully developed tabetic degeneration is almost impossible. For this reason neurologists have in the last few years devoted their attention to this finer and early diagnosis of tabes. It depends chiefly upon ascertaining the subjective diffi-

culties by a refined analysis, upon demonstrating that they indicate a beginning tabes, upon an exhaustive and most minute study of the different objective phenomena—although these may be only rudimentary—and such methods frequently lead to success.

Of the *subjective* symptoms, the typical, lancinating pains are usually the most suggestive, even though these may be transitory, and may appear only as isolated sudden stitches. All forms of paresthesia are to be noted, especially girdle sensation, localized paresthesia, hyperesthesia in the mammary and cardiac regions, ulnar sensation, paresthesia in the perineum, the anus, and the genitalia, the slightest bladder disturbances, decrease of sexual power, transitory diplopia, sudden dilatation of the pupils, etc., and the "rudimentary crises" which have lately been particularly described by Determann, and are as follows: gastric acidity, salivation, tickling sensation in the throat, a sense of burning in the esophagus, nausea, retching, anomalous fecal evacuation, attacks of cardiac palpitation, tachycardia, cardiac asthenia, marked emaciation, a sallow complexion, etc.; if these occur without a recognizable underlying cause, and recur with a certain persistency, they are especially significant.

Naturally the *objective* symptoms are more important, and chief among these are *changes in the pupils*: beginning sluggishness of reaction, although of *one* eye only, distinct pupillary rigidity either unilateral or bilateral, inequality of the pupils, and unilateral mydriasis and paresis of accommodation.

As long as the *tendon reflexes* are retained, slight differences between the two sides are of importance. We should always test the *tendo Achillis reflex*, for its unilateral or bilateral absence often long precedes the absence of the patella tendon reflex, and forms one of the most constant early symptoms of tabes. The *triceps reflex* should also be tested, although its absence is of less consequence; here differences between the two sides are often of great significance. Of course we must bear in mind the fact that the tendon reflexes may be absent in other diseases (paresis, atrophy, neuritis, and sciatica), and these must always be excluded.

Finally, a careful and early *investigation of sensation* leads us to diagnostic conclusions. Here the demonstration of circumscribed hyperesthesia in the trunk (mammary region, perineal region, lateral abdominal region) and in the extremities (ulnar region, soles of the feet, the toes, peroneal region, etc.), of analgesia and retardation of pain conduction in the leg, of hyperesthesia to cold upon the trunk, aids in the diagnosis; finally Romberg's sign, that often appears early. Rarely can *ataxia* be demonstrated in the early stages of the disease, and only by the finer tests. The accurate investigation of the eyes for *double vision*, and of the eye-ground for *beginning atrophy of the optic nerve*, is sometimes of use.

If, moreover, we are dealing in such cases with individuals who are particularly "susceptible to tabes" (as Möbius correctly expresses it), with middle-aged men in whom deleterious factors can be demonstrated, especially a preceding syphilis, the importance of these signs is decidedly enhanced.

We have recently acquired an exceedingly important diagnostic aid in Widal's *cytodiagnosis of the cerebrospinal fluid*, which deserves the most comprehensive investigation. *Marked lymphocytosis* appears to occur even in the earliest stages of tabes. Milian (1904) found it to be *absent* only in the cases of "tabes fruste"; in all other forms it was more or less marked.

My own cases, which have been quite numerous, confirm this. When, therefore, in doubtful cases we exclude those affections in which lymphocytosis also is present (tuberculous meningitis, herpes zoster, and paresis)—and this is usually easy—the proof of lymphocytosis is of great, even decisive, importance.

In illustration I quote the history of two cases recently under my observation:

(1) A land-owner, aged 35, had syphilis in 1889, and was under thorough treatment by Hg; in 1897 he had paresis of the muscles of the eye, dimness of vision in the left eye, girdle sensation, and neurasthenia. In May, 1898, objectively, *general* absence of tendon reflexes was noted, and was said to have existed for some time; the pupils were normal. He took a thorough inunction treatment in Wiesbaden, and was greatly debilitated. Objectively nothing was to be found; there was gradual improvement. In 1901 he took part in military exercises; objectively everything was normal, even the pupils; all tendon reflexes were absent. In February, 1904, there was again for a few weeks a *disturbance of the eye.* Right pupil larger than the left, left reflex showing rigidity, the right still reacting; on the right side micropsia—otherwise, no signs of tabes. March, 1904: *Lumbar puncture revealed marked lymphocytosis.* Diagnosis of tabes decidedly more positive. Hg treatment.

(2) A manufacturer, aged 35, *syphilitic* infection 12 years ago. For 3 years he had moderate but characteristic *lancinating* pain in the legs, with hyperesthesia both in the legs and arms; there was somewhat *decreased sexual power,* but no other symptom. Objectively there was neither ataxia nor Romberg's sign; all the tendon reflexes were retained and active (the left patella reflex was perhaps weaker than the right), and the pupils reacted promptly; there was distinct slowing of the pain conduction without hypalgesia, also mild hyperesthesia to cold in the trunk, and *testicular analgesia,* but no local hypesthesia. *Lumbar puncture: High-graded lymphocytosis;* therefore unquestionably *tabes incipiens!*

It is to be hoped that this means will further advance our knowledge. At all events, with our present refinement of diagnosis minute investigation will enable us to recognize tabes in its *early stages,* and *incomplete* and *abortive forms* will be more clearly and correctly differentiated.

As has been maintained by Nageotte and others, *cytodiagnosis* of the cerebrospinal fluid opens up a further perspective, by permitting us to recognize cases in which tabes is threatened but is not yet present. If "syphilosis of the meninges" forms a prerequisite for the development of tabes, and if this, as can scarcely be doubted, is revealed by cytodiagnosis, a diagnostic lumbar puncture should from time to time, perhaps every few years, be made in syphilitics, especially if they present any nervous symptoms; if lymphocytosis be positively found we should institute an energetic specific treatment to avert the threatening danger.

Differential Diagnosis.—We must now briefly consider the differential diagnosis of tabes from other nervous diseases, especially the spinal forms.

The differentiation of tabes from *functional neuroses,* from *neurasthenia* and *hypochondriasis,* in those patients who come to the physician with all sorts of subjective difficulties and consequent anxieties (especially in physicians, and in syphilitics who have heard of tabes), the diagnosis of the affection merely from the subjective symptoms and with the *absence of all objective phenomena* (in the pupils, in the reflexes, in sensation, etc.) is, as a rule, easy. In isolated cases we must be very guarded in our opinion, and await the further development of the affection. The absence of syphilis here plays an

important rôle; but in all persons previously infected we should be cautious in expressing an opinion.

The disease may be readily distinguished from *hysteria*, which occasionally also presents "tabetic symptoms" (pains, anesthesia, bladder disturbance, ataxia, astasia-abasia, etc.); minute investigation and the objective consideration of the disturbances will reveal the true nature of the affection; one or two objective symptoms (rigidity of the pupils, absence of the tendon reflexes, etc.) may render the decision easy and positive; but we must remember that tabes and hysteria may occur simultaneously. Close analysis of the symptoms appertaining to these affections will dispel all doubt—at least for the expert neurologist.

Among *chronic diseases of the spinal cord* we must primarily consider the *purely motor system diseases* (spastic spinal paralysis, amyotrophic lateral sclerosis, spinal amyotrophies, etc.); they form a diametric contrast to tabes by the absence of sensory and bladder disturbances and changes in the pupils, by the occurrence of paresis, by muscular tension and muscular contractures, increased tendon reflexes, Babinski's sign, and typical atrophies, so that confusion becomes possible only because of great ignorance.

Following these *syphilitic spastic spinal paralyses, transverse myelitis, tumors of the spinal cord* and of the *meninges,* and *gradual compression* of these membranes, must be considered. Here, too, we usually note pareses and paralyses, *spastic symptoms, increased tendon reflexes, Babinski's sign,* etc., so that in spite of the presence of pain, and of sensory and bladder disturbances, the decision, as a rule, presents no difficulty. Intricate cases sometimes form exceptions, but with close attention (normal pupils, no ataxia, no typical lancinating pains, sharp upper limitation of disturbance) the expert cannot long be in doubt.

This is more likely to occur in *multiple sclerosis* which, if the foci are chiefly localized in the posterior columns, may at first produce tabetic symptoms; the intention tremor may also resemble ataxia, or actual ataxia may occur. But multiple sclerosis presents a more complicated symptom-picture than tabes, and the diagnosis will chiefly depend upon the presence of cephalic symptoms (vertigo, headache, nystagmus, scanning speech, intention tremor, etc.), and upon the almost invariable increase of some of the tendon reflexes, upon paralysis, contractures, and the like. The absence of syphilis from the history may be utilized, as well as the age and sex of the patient; nevertheless in some rare cases a diagnosis is only possible after prolonged observation of the case.

Syringomyelia in its typical and *ordinary form* and localization (in the cervical cord with amyotrophy and absence of the tendon reflexes of the upper extremities, with dissociated sensory paralysis, with trophic disturbance, mutilations, with spastic symptoms and increased tendon reflexes in the lower extremities) will rarely cause diagnostic perplexity; but this may readily be the case when the affection has an unusual localization in the *lumbo-dorsal portion of the spinal cord*; here the absence of lancinating pains, of pupillary symptoms, of a specific etiology, etc., will lead to a decision.

Among spinal diseases we must mention Tuczek's *ergotin tabes* which in fact bears a strong resemblance to true tabes. Its epidemic appearance, its etiology, its rapid development, the absence of pupillary rigidity, the

accompanying psychical and convulsive disturbances, sufficiently characterize it.

In *combined system diseases* of the spinal cord (ataxic paraplegia, etc.) the differentiation will usually be easy, provided spastic symptoms and disease of the lateral columns dominate the clinical picture; it may be difficult or impossible when disease of the posterior column is the most prominent feature. Combined system disease has not infrequently been found in undoubted cases of long-standing tabes (lately by Kattwinkel).

Friedreich's disease, hereditary ataxia, which was once regarded as a form of ordinary tabes, but which must unquestionably be differentiated from it, is now usually considered a combined system disease; it differs from tabes (also the infantile form) by its appearance in families even in childhood, by the absence of pain, of sensory and bladder disturbances, and of pupillary rigidity; by the peculiar nature of the ataxia, its early appearance in the upper extremities, in the eyes, and in the organs of speech, and by the absence of syphilis. "*Hérédo-ataxie cérébelleuse,*" closely related to it, and first described by P. Marie, is differentiated by the activity of the tendon reflexes, by a peculiar club-foot, etc.

Diseases of the cerebellum (tumors, atrophy, etc.), in which the "cerebellar ataxia" produced resembles tabes, is usually differentiated by the course, by the form of the ataxia (swaying and staggering), by cerebellar symptoms (vertigo, vomiting, and choked disc), by the absence of all typical tabetic symptoms (rigid pupils, Westphal's and also Romberg's sign, lancinating pains, and typical sensory disturbances); in fact it so completely differs from tabes that it scarcely needs mention.

The differentiation from *chronic spinal meningitis, syphilitic root neuritis,* and the like, rarely causes difficulty; true symptoms of meningitis and the absence of typical tabetic phenomena may make this possible, but these forms of disease may be combined with tabes.

A number of affections remain to be considered, to which, because they produce some tabetic symptoms—such as paresthesia, anesthesia, pain, absence of the tendon reflexes, and especially ataxia—the inappropriate name of "*pseudo-tabes*" has been attached: to this category belong certain cases of *multiple neuritis* (*peripheral tabes*), especially in *alcoholics* (*pseudo-tabes alcoholica*); of *sciatica* with absence of the tendo Achillis reflex; of *post-diphtheritic paralysis* (with atrophic paresis, anesthesia, absence of the tendon reflexes, ataxia, ocular symptoms, etc.), perhaps also some cases of *leprosy and toxic neuritis* (from arsenic, nicotin, carbon bisulphid, etc.), and of *diabetes mellitus* combined with acute pain in the legs (but usually due to sciatica) and absence of the patella reflex. Although some of these cases may have an important bearing on the theory of ataxia, yet, as a rule, close investigation shows that they have nothing in common with true tabes, but may occasionally be combined with it (for example, tabes in alcoholism, tabes in diphtheria, or tabes in diabetes, etc.). Cases of this nature which actually resemble tabes are exceedingly rare; in my long experience I have seen but few. They are to be differentiated, as a rule, by demonstrating the etiologic factors, by the presence of atrophic paralysis and simple non-tabetic sensory disturbances, by the absence of pupillary rigidity, and bladder disturbance, etc.

That *diseases of the brain* can never be confounded with tabes is self-evi-

dent. We therefore briefly refer to the relatively frequent *combination of tabes with progressive paralysis,* because the early recognition of this condition is often of great practical importance. The demonstration of tabetic symptoms in the picture of an already existing paresis is easy, but the recognition of signs of beginning paresis in tabes is not always so simple; here, occasionally, slight rudimentary symptoms of paresis appear which are not followed by the severe pathologic picture of progressive paralysis. Paresis can always be differentiated by the familiar early symptoms, such as a change in temperament, mild psychical debility, inequality of the pupils, tremor of the lips and tongue, slight disturbance in speech, etc.

VIII. PROGNOSIS

Half a century has passed since Romberg remarked, "For none of these patients is there hope of recovery; all are condemned to death." In our knowledge of the disease, its nature, and its causes, we have advanced greatly. Many new therapeutic aids and processes have been tested, but the verdict quoted above has never been reversed, the prognosis of tabes has not become much more favorable.

Naturally, this applies only to the cases of advanced tabes with well marked ataxia and typical symptom-complex. We know to-day that under any circumstances tabes is a serious and usually progressive disease; that, however, the prognosis is decidedly more favorable in those initial cases of tabes in which the affection is recognized at the onset, cases of slowly progressive or mild tabes, of incomplete and abortive tabes, and we now have at command a certain number of therapeutic agents, although we cannot always state that they are valuable.

At an interesting session of the Société de Neurologie of Paris (Rev. Neurol., 1902, No. 1), the view promulgated by Brissaud that tabes has assumed a milder form, develops more slowly, and is frequently arrested, was thoroughly discussed. Brissaud leaves it uncertain whether this is the result of energetic treatment with mercury or of an attenuation of the syphilitic virus. P. Marie, Raymond, and Babinski agree with Brissaud, and attribute the benefit to specific treatment, while Joffroy doubts this effect, and Gilb. Ballet believes the subject not yet ripe for discussion; it is probably true that further investigation is necessary.

Our general description of the disease and of its course indicates the prognosis: tabes, as a rule, is incurable; only in a small proportion of the cases may recovery, or an arrest which amounts to recovery, be expected. Most cases run a slow course, many are extremely mild and benign; in these, therefore, the prognosis in regard to life and ability to follow an occupation is favorable, and many of these patients carry on their work for years; in others, on account of the rapidity or malignancy of the disease, the many severe symptoms, the definite complications, the prognosis is much more grave.

But at the onset of the affection, when the patient has been but a short time under observation, it is often difficult to give a prognosis that is at all reliable. Sometimes insidious and mild cases suddenly take a rapid and rather malignant course; in some cases which begin rapidly, and quickly lead to ataxia, improvement is noted; the disease is arrested for a long time, or

its progress is extremely slow. But this foretells little concerning the individual case.

It may be stated that the prognosis is *influenced favorably* by the following factors: a very slow development, moderate intensity of the symptoms, the absence of severe trophic disturbances and of visceral crises, and a slight systemic implication; by a previously robust constitution, by favorable external circumstances, by the benign nature of the etiologic factors, by the remoteness of the syphilitic infection, by the somewhat advanced age of the patient, etc.

On the other hand, it is *unfavorably* affected by a marked neuropathic predisposition, rapid progress of the affection, early ataxia, a combination of grave symptoms (such as severe pain, gastric and laryngeal crises, arthropathy, cystitis, and other bladder disturbances, etc.), an improper mode of life, inefficacy of therapeutic measures, etc.

In regard to individual symptoms, it must be stated that even the early recognition of the affection does not always avail, because the disease is of substantive character, and is not always amenable to treatment and a regulated mode of life.

The cases with slow progress and but few symptoms, especially if these have existed for a long time, are comparatively hopeful.

The forms designated as *tabes dolorosa* are prognostically very grave, both as regards this symptom and the course of the disease. The same is true of cases with *gastric, intestinal, laryngeal,* and *bladder crises*; these are not infrequently combined in the same patient, and severely damage the entire constitution, thus profoundly affecting the course. Cases in which *arthropathy* and *spontaneous fractures* appear belong to the same category.

Early and extreme *ataxia* is prognostically unfavorable, but we have learned to modify this symptom by appropriate treatment. *Ataxia* which comes on *rapidly and acutely* is almost always serious, although it may rapidly and wholly disappear.

Cases of *tabes cervicalis* and *tabes bulbaris* give little reason for hope, because the implication of the cranial nerves (difficulty in deglutition, etc.) considerably shortens life.

While *tabetic amaurosis* in itself is of exceedingly grave import, nevertheless it considerably modifies the subsequent course of the affection, which usually proves to be mild. A patient whom I treated in 1876 for tabetic amaurosis remained free from ataxia for decades, and is still living to-day.

The cases of *tabes incompleta* and of *abortive* tabes are comparatively benign. As a rule, they remain stationary or progress very slowly, but to this there are, of course, exceptions.

Severe complications (such as syphilis of the brain, apoplexy, cystitis, pyelonephritis, contracted kidney, aneurysm, valvular lesions, myocarditis, etc.) manifestly render the outlook unfavorable.

Tabo-paralysis is absolutely hopeless; it usually causes death in a few years.

Altogether, the prognosis of tabes is very uncertain, and it must be cautiously made. Only after prolonged observation can we attain any degree of certainty; but the patient should always be encouraged to hope for an arrest or improvement, and in any case we should refrain from expressing an unfa-

vorable prognosis, for most astonishing improvement sometimes takes place in apparently hopeless cases.

IX. TREATMENT

It is self-evident that a disease which is relatively so common, appearing among all classes of people, and, as a rule, threatening to interfere with the earning of a livelihood during the most active period of life—a chronic affection of the central nervous system, such as tabes, which lasts many decades—must from the onset have awakened intense professional interest, and inspired many therapeutic endeavors.

But the difficulties presented in the treatment, and, still more, in estimating the results of treatment, are almost incalculable; the tenacity with which this disease often resists all therapeutic measures, its slow development, the variations and peculiarities in its course, the occurrence of incomplete, mild, benign, or stationary forms, the appearance of unexpected remissions and exacerbations, all render it extremely difficult correctly to appreciate the results of treatment; our imperfect understanding of its actual nature and cause prevents a recognition of the precise indications; and although our knowledge of the etiology of tabes is steadily increasing, nevertheless its pathologico-anatomical foundations are still subject to change. Even recently they have again been constructed upon a new basis, yet they lack clearness and certainty. Comprehensive clinical investigation and innumerable therapeutic efforts have failed to bring about decided amelioration, to establish positive methods of treatment, and thereby brighten the prospects of the unfortunate tabetic.

Nevertheless a relatively favorable course of the disease, continued activity, and a tolerable existence are often observed—apparently owing to therapeutic measures; but this effect is uncertain, and only in a minority of the cases are we able favorably to influence the course, to arrest the disease, or to bring about improvement.

And if we bear in mind the changes produced in the nervous system as soon as tabes has manifested itself, the length of time the process usually remains latent, the number of nervous structures which degenerate beyond recovery or have been destroyed, the slight hope of restoration, from the onset there is little hope of success, and we will entertain no sanguine expectations; only too frequently these end in disappointment.

This is evident at the beginning; nevertheless, notwithstanding our unfortunate ignorance of the nature, cause, and course of the affection, as well as our inability to effect a cure, we should never desist from trying to relieve the patient, and should utilize the results of these efforts. Our knowledge of the etiology of tabes, which is definite and conclusive, to a great extent indicates the treatment.

We will, therefore, briefly consider the individual indications which result therefrom.[1]

Undoubtedly we may refer to the *prophylaxis of tabes*; since we know to a

[1] For a comprehensive description I refer to my article: "Die Therapie der Tabes." *Volkmann's Sammlung*, N. F., Nr. 150, 1896.

great extent its cause, it is quite possible but exceedingly difficult, *to prevent* the disease, yet the attainment of this object is most desirable, for when the disease is once present, it is hard to cure it or to arrest it, even in its first stages.

Tabes is probably due, in the main, to the combined action of a number of deleterious factors, the most important and constant of which is *syphilis*. Tabetics are almost exclusively persons who have previously been syphilitic, but even in these there may be other predisposing causes, such as a neuropathic predisposition, congenital or acquired weakness of the nervous system, refrigeration, bodily over-exertion, sexual excesses, trauma, abuse of alcohol and tobacco, excessive mental exertion, excitement and emotion, etc. This shows what the prophylaxis of tabes should embrace.

First of all, the *prophylaxis of syphilis* should be attempted. With the extinction of this disease, tabes also would in great measure disappear. This is not the place for a minute discussion of this point. In spite of all the measures recently brought before the public for the study and prevention of sexual diseases, there is practically but little hope of success in this direction, and we shall long have to reckon with the fact of the prevalence of syphilis, especially among the population of a large city, and among those who visit it.

Therefore, the principal condition for the prophylaxis of syphilis is the *timely, thorough, and sufficiently long-continued treatment of syphilis* in its first stage. Experience has proven that even the most vigorous and most careful treatment of syphilis is not certain to prevent the subsequent appearance of tabes. But the researches of syphilographers in regard to the influence of specific treatment by Hg in the earlier stages of syphilis and for the later tertiary forms, among which we may mention the voluminous statistics of Fournier, Neisser, and others, unanimously and convincingly prove that the *energetic treatment of syphilis in the first stages decidedly decreases the probability of the later appearance of tabes.* At the same time, it proves the absurdity of the foolish statement, so often made, that mercurial treatment favors the subsequent appearance of tabes, or even directly causes it.

How this treatment is to be conducted, how often repeated, and how long continued, need not here be discussed. Unfortunately in practice many of our methods are wrong. Treatment is often too lightly regarded by physicians, the patients are not sufficiently instructed in regard to the dangers of neglect of treatment, they are not impressed with the serious nature of their disease, and this levity on the part of the patient often baffles description. It is unnecessary to make our patients syphilophobes and hypochondriacs, but in many of them explicit instruction within certain limits will not fail of its purpose; Stintzing (Handbuch der Therapie innerer Krankheiten von Penzoldt und Stintzing, V, 3. Aufl.) has given us some valuable advice in regard to this.

But prophylaxis is not exhausted with the treatment of syphilis. Since, in syphilitics, various incidental causes may immediately produce tabes, we must issue a warning against these deleterious factors. Of course, when we consider the rashness of those who do not regard themselves as sick, we cannot hope to attain much; but with some persons such a warning will bear fruit; hence I have long considered it my duty to tell syphilitics expressly that their disease has so weakened them that they react more strongly to all deleterious

factors than do normal persons, and that they cannot expose themselves to these influences without danger, as others may do. This warning is especially needed by those who have a "hereditary or acquired" *neuropathic tendency,* those who perhaps exhibit all sorts of nervous symptoms (neurasthenia, etc.) They should guard against *refrigerations, especially repeated ones, and over-exertion and fatigue*—for example, in hunting, fishing, mountain climbing, rowing, bicycling, etc. They are to avoid *sexual excesses, and the abuse of tobacco and alcohol,* are to be exceedingly careful for a long time after trauma, carefully to avoid *immoderate work, mental strain, emotion, and great excitement,* and especially the combination of these harmful factors; after influenza or other serious infectious diseases, they should allow themselves a sufficient time for convalescence, and should not resort to *immoderately hot baths, steam baths,* nor the ridiculous *use of cold water* at extremely low temperature.

Persons who are predisposed to nervous affections, and who have acquired syphilis, must guard against the development of tabes by a sensible mode of life, by tonics, and by air and water cures, etc.

This about exhausts the prophylaxis of tabes, and we turn to the treatment of the *causal indication.* It is clear that any treatment of tabes has a better prospect of success if begun early, therefore the *early diagnosis of tabes* is of paramount importance.

In the causal treatment, *antisyphilitic measures* undoubtedly and almost exclusively first come into question, but their strict scientific applicability is at this time uncertain, and even impossible to prove. If tabes could be regarded simply as a late manifestation, a special form, of tertiary syphilis, the foundation for this treatment would be clear; and although many of the lesions and numerous tertiary gummatous manifestations in the meninges and vessels (especially the irritation of the meninges revealed by cytodiagnosis) must undoubtedly be regarded as due to syphilis ("syphilosis"), and as of extreme importance, this is not yet absolutely certain; the possibility that we are dealing with a specific toxic action, the effect of a syphilitic poison constantly furnished by old, latent, pathologic foci, and perhaps by abnormally increased antitoxins, immunization products and the like, necessitates a certain reserve, and the prospect of an effective syphilitic treatment becomes decidedly less.

Notwithstanding these considerations, the majority of modern investigators have come to the conclusion that antisyphilitic treatment is indicated in tabes, and that this should be more or less energetic; some have made only brief trials, others have pushed iodin and mercury; many have obtained good results, others only moderate, and with still others the results were chiefly negative; among those who admit the connection between syphilis and tabes, some reject specific treatment absolutely, to say nothing of those who declare, what has been sufficiently disproven, that specific treatment is injurious to the tabetic.

The justification of a specific treatment of tabes can be determined only by experience; and the difficulty of securing accurate reports has already been pointed out; this is increased by our newly acquired knowledge that some of the mild forms of tabes remain stationary, and that they follow a varying course which is difficult to determine beforehand; value can be attached only to extended series of observations, and to the impressions of experts.

The experiences of numerous authors are at hand. They are almost united

in the opinion that antisyphilitic treatment in tabes, especially if early resorted to, and with some necessary modifications, is undoubtedly beneficial in the majority of the cases, but in only a few is this benefit very decided.

It would be a digression to detail reports from literature or personal experience. I shall only state that in numerous cases in my own extensive practice I have often seen good results from a rational specific treatment several times repeated; that in a few cases these results amounted to complete recovery, for the patients were able to resume their occupations and to follow them for years (reflex pupillary rigidity and the absence of the tendon reflexes, etc., perhaps persisting); that in a series of cases the affection was often checked for years; but I must add that in a large number of cases, especially those well advanced, no arrest of the disease could be brought about.

At the same time my abundant experience has taught me that *specific treatment*—provided it is carried out rationally and carefully—*is absolutely harmless to the tabetic,* so that the loud warnings which have repeatedly been issued against this treatment are entirely uncalled for. It is self-evident that some individuals are susceptible to Hg or iodin, and do not bear this treatment well. But it is also true that most tabetics tolerate cautious and even energetic antisyphilitic treatment, and that it improves their general condition, and increases their weight.

Therefore, considering that syphilis is the most common, most important, and, primarily, the chief cause of tabes, that the pathologico-anatomical changes in tabes certainly do not preclude the effect of specific treatment, that undoubtedly true, specific, gummatous diseases often introduce tabes, that lumbar puncture reveals an irritation of the meninges which is most likely syphilitic, that perhaps, somewhere in the body (glands, etc.) latent, luetic, pathologic foci continue to exist, the removal of which is beneficial, and, finally, that specific treatment is never injurious to the tabetic, it is evident that *in tabes preceded by syphilis an antisyphilitic treatment is generally indicated.* It is also certain that it should not be blindly resorted to, but that the indications in each case should be minutely considered, and the treatment instituted more or less powerfully, according to individual circumstances; sometimes it yields no result whatever.

As a rule, *specific treatment is justifiable* in the following conditions: 1. *In all recent cases of tabes,* especially if the syphilitic infection is not remote; 2. In all cases in which *florid symptoms of syphilis* are manifest somewhere in the body (in the skin, mucous membranes, bones, etc.), and when these are combined with *symptoms of cerebral or meningeal syphilis,* especially in those cases in which *lumbar puncture* reveals a *typical lymphocytosis*; 3. In all cases, even long-standing ones, in which *an ineffectual syphilitic treatment had been previously instituted,* in which relapses had occurred, or in which there is marked *lymphocytosis.*

Of course, when the disease is well advanced and the ataxia is prominent, when the cases have been associated with special conditions, for example, with recent relapses, with new tertiary symptoms, with decided lymphocytosis, we usually conclude to reinstitute specific treatment. Even if this is unsuccessful, we risk little, and I have repeatedly known astonishing benefit to follow.

Specific treatment is *contraindicated* in very advanced and long-standing cases, in emaciated, cachectic, or dyspeptic patients, when the luetic infection

is remote, when specific treatment has been repeatedly unsuccessful, and Hg and KI have not been tolerated.

The *method of treatment* must be chosen according to the fundamental principles by which the late forms of syphilis are treated; primarily, we should consider *Hg* combined, or alternating, with *iodin treatment.* Whether this exhausts the therapy of late syphilis is doubtful; possibly the future may bring to light remedies more effective than these. I am under the impression that Hg and iodin are not so beneficial in these degenerative processes in the nervous system as they are when secondary and tertiary manifestations are present. But advancing knowledge, or, perhaps, a fortunate accident, may furnish something better.[1]

We prefer the old, well tested *inunction treatment,* but have no objection to other methods (internal administration and subcutaneous injections of soluble and insoluble preparations of Hg). It is our practice to institute a moderately active inunction treatment, and to repeat it at long intervals (30 to 50 inunctions, each of 4 to 6 grams, rarely more), observing all the precautions necessary to prevent stomatitis, including two lukewarm baths a week, plentiful nutrition, an outdoor life, the patient absenting himself from his business and occupation. The latter is most important, therefore we frequently combine this method with spa treatment (at Aix-la-Chapelle, Nauheim, Wiesbaden, Baden-Baden, Rehme, Nenndorf, Baden in Switzerland, etc., but avoiding hot baths), or this treatment may be carried out in the hospital or sanatorium. I must issue an express warning against using this treatment while the patient follows his occupation or remains at his home.

For years I have maintained that treatment with Hg first of all removes the underlying cause of tabes, and, so to speak, prepares the way for the action of other remedies—other measures (for instance, those usual in the treatment of meningitis) may be tried at the same time. But our chief reliance must be upon the former, hence the attention of the patient must be called to the fact that during mercurial treatment no marked improvement is to be expected; on the contrary, it may at first seem to aggravate the condition, the benefit appearing later.

After treatment with mercury I have attempted to bring about improvement by a *tonic process*—a residence in the mountains, mild hydrotherapy, sitz baths, electric treatment, the administration of tonics (arsenic, cacodylin injections, glycero-phosphates, lecithin, etc.), or a spa treatment at Rehme or Nauheim, and I believe that I have thus secured excellent results.

According to circumstances, *mercurial treatment* is to be repeated in from four to eight months, or even a year later, and thus an alternate specific and tonic treatment may be carried out for several years. The patients are usually satisfied with the improvement which appears, and are willing to continue the treatment.[2]

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[1] The combination of Hg with an organic preparation of arsenic, such as the so-called *ennesol* (Salicylarsinate de Mercure, Coignet), promises to be a particularly active remedy for tabetic and similar syphilogenous degeneration. I have instituted some trials with it.

[2] Others have advised similar methods, but the principle is always the same—regular treatment by mercury, continued for some time, with intermissions.

Bockhart advises an annual treatment of 20 to 25 inunctions, each of 2.5–4.0 grms.

In this form mercurial treatment is well borne by the patient, but whether it will effect a cure is another question. Some authors assert that the inefficacy of the treatment which many physicians report is due to a want of energy and persistence in the administration of Hg.

In France the institution of brisk and long-continued mercurial treatment in tabes and paresis has recently come into favor, many successes having been published. I feel very skeptical, for I have undoubtedly seen many ill effects from the "heroic treatment," which has been advised in Germany by O. Ziemssen (Wiesbaden). The result of these trials is still *sub judice.*

Of course, in an Hg treatment, *iodin therapy* also comes into question. Undoubtedly it should be resorted to in many cases, and in some it is certainly beneficial. The administration of KI (or NaI) may be combined with that of Hg, or the drugs may be used alternately for from one to three months, 1–4 grams or more daily, before or after a tonic. The use of iodin has been greatly facilitated by the introduction into treatment of *iodipin,* which is best given in the form of subcutaneous injections (10 and 25 per cent.), and is usually well borne.

Of other antisyphilitic methods—Zittmann's treatment, *roob laffecteur,* diaphoresis, etc.—not much is to be said. Occasional trials of these have been unsatisfactory to me.[1] So much for the antisyphilitic treatment of tabes.

Now the question arises: How are the cases of tabes without preceding syphilis to be treated? Considering that some authors regard all tabes as due to syphilis, and the impossibility in many cases of positively excluding a syphilitic infection (because, from the nature of the case, the infection is not unlikely, at all events can neither be disproven nor demonstrated) it may be asked: Should, or may, these cases also be placed under antisyphilitic treatment? Some authors answer this question in the affirmative, and advise at least a trial with mercury and iodin; certainly this is very common in practice, and although we risk little with such an attempt yet I do not concur in it; every physician must, in the individual case, answer this question for himself; here cytodiagnosis may help us to a decision. If we believe that lymphocytosis of the lumbar fluid is positive evidence of the syphilitic nature of the infection, and if in beginning tabes *without* demonstrable syphilis we find a typical lymphocytosis, this is a very clear indication for syphilitic treatment.

I shall briefly detail such a case which I recently observed: A man, aged 35, had suffered for 3 years from lancinating pains, ptosis, and bladder weakness, to which typical *tabes incipiens,* with all its objective symptoms (without ataxia) was added. He *never had chancre or syphilis,* but had one attack of gonorrhea. His children were healthy. When examined *no signs of syphilis could be found* (merely slight cicatrices

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with warm baths and complete rest. The method recently advised by O. Rosenthal ("Therapie der Syphilis," 1904) is worthy of notice. He reports a number of conspicuous cures and of arrested cases under the following treatment: daily *inunctions* of 4 to 10 grams with 4 to 6 weeks' *rest in bed* (very important), also sweating, daily bathing, and injections of iodipin. The treatment is to be *repeated* after 3 months, then after an interval of 9 months. In the second year there are to be two treatments, and in each of the two following years one treatment. The diet is to be carefully regulated, but should always consist of roborant foods; no alcohol, no tobacco!

[1] Whether, when demonstrable lymphocytosis indicates a beginning tabes, this should be treated with *intradural injections* (of preparations of iodin, mercury, or the like) I dare not decide, but the question certainly deserves consideration.

at the angle of the mouth which had no significance as the patient was an inveterate smoker). Lumbar puncture revealed *high-graded lymphocytosis*. I regarded this as a clear indication for treatment with Hg, which gave most excellent results.

It will rarely be necessary to combat *other causal agents* than syphilis. The causes which the patient may mention are too remote, and are past counteracting; there remains, therefore, merely the question of removing exciting or permaent deleterious factors: the effects of cold, of over-exertion, of living or working in damp rooms, of sexual excesses, of stimulation, the abuse of alcohol and of tobacco, excitement, emotion, mental strain, of baths either too cold or too hot, etc.; with the regulation of these much will be attained.

For those who regard tabes as a late form of syphilis—i.e., an actual syphilitic disease—and if they desire to do justice to the *indicatio causalis*, the *indicatio morbi* coincides with the specific treatment just outlined. Self-evident as is this view to-day, nevertheless it has not been proven; specific treatment has its limits, and frequently yields only unsatisfactory results. There is still room for a non-specific treatment of tabes; at all events, the cases of tabes without a preceding syphilitic history should be directly treated, and in all cases the *indicatio morbi* should be fulfilled. Its purpose is to compensate for the degenerative processes going on in the posterior columns and other parts of the nervous system; at least to remove the *functional disturbances caused by these.* This is its most important object, although, in the strict sense of the word, a "pathologico-anatomical cure" is probably never attained; it is practically satisfactory to the patients if the disturbed functions are sufficiently compensated, and their complete activity restored. It is quite immaterial to them whether gray degeneration still remains or whether there are irrelevant symptoms which nowise disturb them—such as reflex pupillary rigidity, and the absence of the patella tendon reflex.

The methods which nature provides to attain this goal vary. Nutritive disturbances in the nervous elements may be counteracted by an invigorated and improved metabolism, by the stimulation of the circulation of blood and of the lymph, perhaps also by the elimination of toxic products; but the slumbering powers of vicarious and collateral tracts may also be awakened, and may be utilized as substitutes for the lost functions.

The *general improvement of the nutrition, the stimulation of metabolism, as well as of the functions of the diseased nerve tracts, relief of the circulation* in these tracts, and kindred alterations may produce *a change of chemism in the diseased nerve tracts*; the stimulation and *exercise of vicarious and compensatory functions* will be supplementary.

There are many methods for this purpose. Before enumerating and considering these, a few introductory remarks are in order concerning the *general dietetics* and the *mode of life* of the tabetic.

Generally speaking, tabetics must live a quiet, regular life, eliminating over-exertion, excitement, and excesses as far as possible. "Live as if you were an old man, simply, abstemiously, quietly, and regularly!" is the advice that I give to all patients in the first stage of the affection; later this is compulsory, but in the stage of onset it is profitable, although often difficult, to follow this direction.

Therefore simplicity of life, with moderation in eating and drinking, in smoking, in social intercourse, in sexual and other pleasures, with proper alter-

nations of work and rest, moderation in work, and sufficient recreation is important; simple pleasures and cheerful companionship are most desirable, but all excitement, emotion, and over-exertion are to be prohibited; an outdoor life in the summer,[1] a residence near the woods or in a mountainous region, and in the winter a warm climate, are to be recommended. Care should be taken to avoid excessive bodily exertion and over-fatigue; hence sports (hunting, mountain climbing, bicycling, and tennis) are to be prohibited or carefully regulated. In the first stage, the pre-ataxic, we may allow the gymnastic exercises, especially in the room, rowing, moderate bicycling, and even horseback riding if not in excess.

Naturally we must closely consider the individual conditions, and, especially in recent and progressive cases, must be extremely cautious; in the late or stationary stages, the patients should practise regular muscle exercises, and movements and gymnastics in the open air (see Frenkel's method).

Edinger's theory of the development of tabes is of great importance, but very perplexing. Were it generally accepted we would advise for most tabetics, especially at the onset of the affection, in exacerbations, or in relapses, complete bodily rest or even rest in bed. But, of course, this cannot always be done; for a robust tabetic, who for years and decades may perhaps still be active, will not at the beginning of the disease submit to such a method. But for those forms in which ataxia progresses rapidly, with motor debilitative conditions and extreme emaciation, absolute rest is strongly to be recommended. I believe that the benefit of this method cannot be doubted, but in other forms of the disease it may do the patient harm by making him more flaccid and less energetic, by increasing the hypotonia of the muscles and the ataxia; therefore in these it should not be continued. In regard to this it would be well to collect extensive reports, as Edinger did. Here, as everywhere, we must carefully individualize.

Among the various remedies and curative methods, *drugs* must be first mentioned, but little can be said in their praise. There has been but slight advance or additional clearness of indications in the past 30 years, and no record of prompt and positive results. Nevertheless, in many cases their value cannot be doubted, and, with the endless duration of the disease, they certainly cannot be dispensed with.

Silver nitrate still enjoys the highest reputation—its results in a certain number of cases are incontestable; it should be given for a long time, even months; or, with long intervals, may be continued for years (in pill form, 3 to 5 centigrams daily, preferably with extract of nux vomica) until from 10 to 12 grams of the remedy have been used. Inunctions of unguentum argent. colloid. Credé, need further trial (Determann).

Ergot which was almost exclusively employed by Charcot (2 or 3 times daily for a few weeks, then resumed after an interval, etc.) has fallen into disuse; its affinity for the posterior columns (Tuczek's ergotin tabes!) makes its administration seem unwise, although its effect in small doses cannot be denied. I chiefly employ this drug (or, better, ergotin) in cases of extreme weakness of the bladder.

[1] The treatment advised and elaborated by Determann combines hygienic, physical, and dietetic methods; "treatment with rest in the open air" certainly merits consideration.

Even before syphilis was recognized as the main cause of tabes, *potassium iodid* was praised by many, particularly for the lancinating pain. Nowadays its effects do not seem to be remarkable. We have already referred to the use of KI, NaI, and *iodipin.* The *salts of bromin* have also been recommended, but I have never observed their directly curative effect in tabes. *Arsenic* also has been much praised; undoubtedly this is useful in various diseases, particularly those of the nervous system. I have used it often, apparently with very good effect; but I have never decided whether it acts simply as a tonic and blood-forming remedy, as a nervine, or whether it has a directly curative influence upon tabes; the first theory appears to be the most likely. I administer it in the usual forms, either as arsenious acid, as Fowler's solution (generally in combination with tincture of nux vomica), as Levico water, or, lately, as cacodylate by subcutaneous injection. After abundant experience I have formed a very favorable opinion of its tonic and alterative effects in the last mentioned form.

The same may be said of *strychnin* which I have frequently used, and which undoubtedly has some effect in tabes, although this action is difficult to explain. Whether it directly affects the chemism of the spinal cord tracts, or is merely a general nervine, stomachic, or tonic, I shall not attempt to decide; but it is certainly beneficial (the subcutaneous injection of 2 to 10 milligrams, or the administration of the various preparations of nux vomica, either tincture or extract, and preferably combined with other remedies, such as arsenic, silver nitrate, wine of condurango, or other *tonics*).

Among these I lay stress upon the preparations of iron, which, combined with arsenic, quinin, or strychnin, are undoubtedly valuable for their general tonic effect by which they restore the powers of the organism. I frequently employ with benefit my so-called tonic pill (see formula below [1]), and alternately arsenic and the cacodylates. They form a very satisfactory after-treatment to Hg and iodin cures, and are well borne and highly praised by the patients.

Of the numerous other remedies employed in tabes—the double chlorid of gold and sodium, quinin, antipyrin, phosphorus, and barium chlorid—nothing special can be stated; experience alone will show whether the *glycerophosphates,* the phosphorus-containing *lecithin preparations,* and *phytin,* which have lately been advised, give better results.

The opotherapy of Brown-Séquard, treatment with organic extracts (of the brain and spinal cord, and of the testicles, also with spermin-Poehl, etc.), has found but few proselytes. I must, however, admit that I have seen a few cases in which injections of spermin apparently had a favorable and tonic effect; therefore this should not be entirely tabooed in the treatment of this tedious disease.

Balneotherapy has a prominent place in the treatment of tabes. The views

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[1] ℞ Ferr. lactic., Extr. chinæ aqu. } āā 4.0–5.0
Extr. nuc. vom. spir 0.5–0.8
Extr. gentian .... q. s.
M. f. pil. No. 100.
One or two three times daily after meals.
The formula may be modified according to individual indications.

in regard to this and its indications have changed but little in the last 25 years. The exact action of baths, by means of their temperature and the salt and CO_2 they contain, is not quite clear, but there is no doubt of their effect upon the circulation and respiration, the skin, the centripetal nerve tracts, the general metabolism and nutrition, upon appetite and sleep, etc., although this is difficult to explain. To-day baths certainly belong to the therapeutic measures which are indispensable in tabes.

The thermal salt baths of Nauheim and Rehme-Oeynhausen, which are rich in CO_2, are in the first rank. I have had abundant experience with these, and in many cases their beneficial effect is striking. But, when unsuitable, especially when forced, they may do harm, therefore we must exercise discretion in using them, and must strictly individualize. It is the duty of the spa physician, who fully understands the waters, to prescribe the exact temperature and duration of the bath, its amount of gas, and the kind of movement (douche baths, etc.); baths of medium or low temperature, containing a *moderate* amount of CO_2 and with a moderate motion of the water, appear to be the best adapted to all tabetics except those who suffer from severe attacks of pain, or who have areas upon the trunk which are hyperesthetic to cold, and whose nervous systems are very irritable.

Those *iron baths* which are rich in CO_2, and some of which also contain salt (Schwalbach, Franzensbad, Tarasp-Schuls, St. Moritz, and others), have a similar effect and are resorted to for similar reasons, particularly when we desire to combine their tonic effect with mountain treatment. *Peat baths* have been sometimes praised, but the indications for their use are difficult to define, hence they are rarely ordered in tabes.

Springs at *varying temperatures* (Schlangenbad, Ragaz, Wildbad, Gastein, Teplitz, Wiesbaden, Baden-Baden, etc.) were formerly recommended; but baths at high temperature (above 33° C.) have proved rather injurious to the tabetic, and hold no important place in the therapy. In a few irritable and erethetic cases with great pain, hyperesthesia, and different forms of crises, etc., they appear to have been beneficial when cautiously given at a somewhat reduced temperature, when of brief duration, and not too frequently repeated.

The same is true of *sulphur springs* (Aix-la-Chapelle, Nenndorf, Baden near Vienna, Baden in Aargau, etc.); they are highly advocated as auxiliary remedies during mercurial treatment, but we should be careful not to permit too high a temperature.

The *springs of La Malou,* ordered exclusively in France, appear to possess no special advantages.

Steam, air, and *electric light baths* can be used in tabes only with the greatest care, and are better omitted.

On the contrary, the benefits from *hydrotherapy* are especially marked. The generally strengthening and refreshing effect of the water, its action upon the skin, the sensory nerves, the circulation, and the respiration, upon metabolism, upon the general nutrition, etc., sufficiently account for the benefit which tabetics experience under this treatment. I have had abundant experience with hydrotherapy, and I consider it just as valuable as a treatment in Nauheim or Rehme; much depends upon the mode of application and its individual adaptation. All highly irritating procedures which cause a sudden

shock (very cold baths, douches, ablutions, or packs) are to be strictly avoided, as well as the Kneipp treatment; as a rule, the milder processes, carefully and gradually increased, such as *tempered sitz baths* (from 30° to 20° C.), simple *ablutions, tepid full baths, local spongings, as of the back, feet,* etc., are preferable. Much depends upon the choice of an institution; sanatoria in wooded or mountainous regions are the best, and the treatment should be by a specialist. Under a suitable *régime* all tabetics bear this treatment well. Here and there a certain intolerance is noted in anemic or irritable persons who show a poor vasomotor reaction, hyperesthesia to cold, etc.

In some cases *river bathing* is permissible in midsummer, while *sea bathing,* especially when the temperature of the water is low and the surf strong, must be resorted to very cautiously; nevertheless, they are sometimes beneficial.

Electrotherapy in tabes, as practised and advised by R. Remak, has under the weight of recent opinion and, it appears to me, unjust criticism, come under the ban. Naturally we do not share Remak's illusions; nevertheless, although electrotherapy in tabes has often been unproductive of good, in many cases it undoubtedly has led to improvement, amelioration, or even the arrest of the affection—in rare cases the results were quite remarkable. This is absolutely certain, and in the interest of tabetics I regret that the skepticism of most authors concerning the action of electricity has curtailed the use of this remedy. Since the influence of suggestion is so powerful, it might be well to treat the tabetic by hypnotic suggestion; but concerning this remedy no successful results have been published.

Of course this treatment must be carried out scientifically by a physician who fully understands the conditions, and is specially versed in the method. But this principle is frequently overlooked; hence the many unsuccessful results which have helped to displace electric treatment. Treatment by the patient himself, by his relatives, or by nurses should be absolutely forbidden. As a rule this prohibition amounts to nothing, and in extremely mild cases we may be obliged to permit persons who have been carefully instructed to carry it out.

For details concerning the probable effects of electricity (stimulation of function, of the circulation, of nutrition, of the nerve elements, thereby checking degeneration), the strength of current to be employed, and the methods of application, I must refer the reader to text-books on electrotherapy; here I shall briefly discuss the most important indications.

The *galvanic current* is of prime importance. In a longitudinal (columnar) disease of the spinal cord, such as tabes, the longitudinal application of the current is most practicable. Both poles should be placed on the vertebral column, the large electrode (ascending or descending current or these alternately), the stabile current with the electrodes successively and directly covering the different areas of the spinal cord; or the method, which I prefer, of simultaneous galvanization of the sympathetic (the cathode upon the cervical sympathetic, the anode at different points along the thoracic and lumbar vertebral column, stabile, applied to each side for one or two minutes, then longitudinally to the back) or the application of either electrode to the sternum, the other to the back, successively covering the various portions of the spinal cord, stabile, in recent cases the anode, if of long standing preferably the cathode, or even both poles alternately may be applied to the back. Where the

spinous processes are specially sensitive, and there is girdle pain, intercostal neuralgia, hyperesthesias of the trunk, or lancinating pain, this "polar" treatment of the back (of the nerve roots, intervertebral ganglia, etc.) is recommended.

In addition, peripheral galvanization of the extremities and their nerve trunks may be useful (anode to the lumbar enlargement, cathode labile along the entire course of the nerves in the leg, the same to the upper extremities).

The use of the large electrode with only a moderately strong current, the thorough moistening of the skin, the smooth and skilful management of the electrodes, and the not too-prolonged administration, are all enjoined.

The *faradic current* usually serves a symptomatic purpose; the faradic brush which Rumpf applies systematically to the skin of the trunk and the extremities with a moderately strong current, for from 5 to 20 minutes daily, perhaps favorably influences the regeneration of the sensory neurons by stimulating their functions; Rumpf has successfully used this method with a simultaneous inunction treatment, and I also can recommend it; especially in cases in which there are marked hypesthesia and extreme and lancinating pain.

Static electricity, high tension currents, in the *magnetic field,* and other modern methods which are much employed and praised, have as yet no warrantable scientific foundation.

The application of *derivatives to the skin* is still advised, and in some cases is undoubtedly useful. I employ these in different ways, especially when there is pain in the back, a girdle sensation with localized lancinating pains, gastric and anovesical crises. Occasionally I use them in a more permanent way by keeping up a counter-irritation on the back, and thus favorably influence the entire process. This action, although of very mysterious nature, is not to be denied.

"Pointes de feu" (a small thermo-cautery applied over an area about the size of the hand, making 30 to 50 small superficial burns in an ascending row immediately over the vertebræ, applied every 6 to 8 days to the entire back, and continued for months), used and particularly recommended by French physicians, are most suitable for this purpose; so also are *fly blisters,* and *painting* with weak or strong tincture of *iodin* over the vertebral column. I have repeatedly applied *mercurial plaster* to the vertebral column for a long time, also *alcoholic solution of veratrin, spirits of camphor,* or similar rubefacients.

Finally, we must mention the highly lauded and sometimes extreme *surgical or orthopedic curative measures* which have recently been proposed in tabes. In spite of the exaggeration with which they have been introduced into practice, a slightly beneficial effect cannot be denied them.

Massage has been much resorted to in tabes, and certainly has a beneficial effect upon the process as a whole; by stimulating the metabolism of the peripheral nerves, etc., it assists the circulation in the skin and muscles, stimulates the sensory tracts, increases the metabolism, the general nutrition, and the muscular activity. All the various movements of German and Swedish massage may be useful, provided they are not carried to an exhausting extreme. I have certainly seen great benefit from it, especially in poorly nourished, emaciated persons with weak muscles, loss of energy, sensory disturb-

ances, conspicuous ataxia, a dry, flaccid skin, etc., and for these cases it may always be recommended.

Gymnastic exercise is also valuable as a general strengthening measure, assisting the muscular functions and the muscular activity, etc.; it has received a great impetus through Frenkel's "reeducation method," to which we shall revert when describing the symptomatic treatment of ataxia. That this treatment may also favorably affect the general process is obvious. But with gymnastics and massage, as with all other remedial measures, we must strictly individualize.

Recently but little has been reported of *nerve stretching*, formerly so much discussed, especially of *operative* nerve stretching. After a few apparently favorable results, we were led to perform a number of such operations, and during one of these both sciatics and both crural nerves were stretched. After a few cases had terminated fatally, and many had proved unsuccessful, the method was abandoned; Benedikt's opinion that it is a mistake not to perform it has failed to prevent the method from falling into disuse. In the rare cases of very stubborn, localized, tabetic neuralgia affecting various nerve regions (sciatic crises, severe intercostal neuralgias, etc.) the method may be useful, for we cannot deny the possibility of an effect upon the spinal ganglia and the posterior roots.

For these and similar cases, however, *bloodless nerve stretching* will suffice, and is certainly less dangerous. v. Corval's method of performing it is to have the patient lie perfectly flat with legs extended, then to flex the legs so strongly at the hip-joint that both feet approximate the head, this position to be maintained for some time; or, according to Hegar, the patient is to sit upon a table with the lower extremities extended, then the head and chest are to be strongly bent forward ("stretching of the spinal cord"). The milder method of Blondel is daily to approximate the flexed knee to the chin, and hold it firmly there for five minutes by tying a bandage around the neck of the patient and also around the popliteal space; Bonuzzi has the patient lie flat upon a bed with his head supported, fastens a towel around the ankles, then by flexing the vertebral column forward as far as possible the legs are drawn so high above the head that the knees are brought to the frontal region and even further. This movement is very painful, and not devoid of danger; only the hypotonia of the tabetic enables him to perform it at all.

All of these methods are occasionally productive of benefit, especially for the lancinating pains; they relieve, partly by stretching the nerves, partly by their effect upon the lower half of the vertebral column, perhaps also by stretching the posterior roots of the nerves and spinal ganglia. Hence, whenever suitable, they may be cautiously used.

The method introduced by Motschutkowsky, and eagerly adopted by Charcot's disciples, that of suspension of the patient, also depends upon its effect on the vertebral column. Sayre's apparatus is used, the patient being suspended by the chin and nape of the neck while supported at the axilla, and is held in this position for from one to five minutes; this is done once daily or less often. The weight of the body stretches it, and elongates the vertebral column; perhaps it also produces tension of the meninges and nerve roots, and frees the nerve trunks.

Sprimon's suspension apparatus is more simple and gives better results,

The patient is seated, and weights of varying size (50 to 150 pounds) are attached to his elbows, his chin, and the nape of his neck; he is then slowly lifted. This method is susceptible of various modifications, is never injurious, and may be continued for a longer time—from 10 to 20 minutes. Simple surgical extension of the vertebral column upon the oblique bed (as in caries of the vertebræ) may also be resorted to.

No satisfactory explanation has yet been offered of the undoubtedly favorable results of this process; certainly, in spite of suspension, some decided successes in tabes must be admitted, for the disease as a whole, as well as many of its individual symptoms—the lancinating pains, the girdle and armor sensations, the disturbance of muscular sensation and the ataxia, the bladder and sexual weakness, etc.—have shown decided amelioration. I can report some favorable results with this method, but, in many cases, it has been unsuccessful; carried out according to the original method I do not consider it free from danger; very heavy patients, or those with arteriosclerosis, disease of the heart, or cerebral disorders, etc., should not be subjected to it.

For slight but permanent suspension, extension of the vertebral column by a well made Hessing *brace corset* is useful. This furnishes a support and removes weight from the vertebral column; in fact appears to give a certain relief to many a patient. Bladder disturbances, pain, and paresthesia are lessened, and patients acquire a firmer station and gait. But the permanent results are by no means so brilliant as Hessing assumes; his versatility, as shown by the invention of such an apparatus, probably depends upon his ignorance of the pathology of the spinal cord, and is exaggerated by the *naïveté* of his prognosis; actual cure is impossible, and I have seen a number of cases in which the patients soon objected to the corset, and refused to wear it. Nevertheless for many a corpulent and nervous tabetic it forms a good palliative remedy.

An important part of the treatment of tabes remains to be discussed. This includes the almost endless *symptomatic indications*, which, as well as the treatment of the fundamental disease, call for the attention of the physician; when the malady is of long duration, and even after all hope of improvement is gone, these become more and more prominent. Above all, the treatment of the most typical and specially important symptom of tabes—ataxia—has lately been accurately studied and developed to a high degree, and we will now thoroughly discuss this as well as less noteworthy conditions.

One of the earliest indications is given when the *lancinating pains* become at all severe, and—as in "tabes dolorosa"—these soon may become unbearable. In the milder forms the patient frequently regards them as ordinary "rheumatic pains," he scarcely notices them, and does not seek professional aid, so that the affection is often recognized too late.

For these pains numerous remedies are at our command; only a few of these have a certain effect, and the reaction in individual cases is very different, hence many remedies must be resorted to. In the most severe cases the patients suffer greatly, and only the injection of large quantities of morphin affords relief.

At first, mild, external remedies should be tried; among these are warm or cold compresses, the application of cotton saturated with chloroform or ether to the painful and hyperesthetic areas, ethyl chlorid or ether sprays,

sinapisms, inunctions with oil of chloroform, or with veratrin dissolved in alcohol or in an ointment, the application of various blisters, of emplastrum opiatum or emplastrum belladonnæ, etc., also counter-irritation with strong solutions of iodin, and the use of vesicators or *pointes de feu* on the vertebral column or over specially affected root regions. *Electricity* often has a favorable effect, either the stabile application of the anode or cathode to the painful area and to the affected root regions of the cord, or strong faradization of the skin (moist, or with the faradic brush). *Friction and brushing of the skin*, strong *pressure* (the tying on of leaden plates, etc.), *massage, suspension, bloodless nerve stretching*, may also be very beneficial.

Various *internal remedies* are, however, much more active; among these are quinin, the salts of iodin and bromin, gelsemium, and especially the numerous anodynes which have almost all been recently employed for the pain of tabes, and are very active. Chief among these is antifebrin (0.3–0.5 per dose), then antipyrin (0.5–1.0), phenacetin (0.5–1.0), salipyrin, lactophenin, aspirin (0.5–1.0), exalgin, migrainin, pyramidon (0.25–0.75), etc. As in migraine, the effect of these remedies varies in different persons; hence they must be carefully tried in each individual case. Sometimes different ones may be combined with small doses of narcotics (codein, atropin, morphin, heroin) and thus a more powerful effect be secured. Lately I have often seen excellent results from a so-called "mixed powder" (phenacetin 0.6, antipyrin 0.3, codein 0.04), and this may be used in different combinations. In severe cases, the doses must be large, and rapidly increased. In the worst forms, which are accompanied by severe paroxysms of lancinating pain, lasting for days, only morphin will relieve the sufferer; yet this calls up the threatening specter of morphinism, therefore great care is necessary! In each individual case we must decide how far it is safe to go. For myself, an existence made tolerable by the use of morphin is infinitely to be preferred to unmitigated torture.[1]

Much more difficult, however, is the treatment of the various *tabetic crises*, especially those of the *stomach* and *intestine*, which are usually intractable and cause the patient great misery. These are best treated by complete rest and a strict diet (almost complete abstention from food, nourishment being by means of enemata and salt water infusions). The lancinating pain must be relieved by anodyne remedies, including morphin; warmth, cold, derivatives, *pointes de feu*, and vibration massage may also be of use. Sometimes electricity is effective (the galvanic current, a large electrode being applied to the epigastrium and abdomen, to the celiac plexus and the mesenteric plexus, to the splanchnic nerves, etc.; or faradization and the faradic brush); last of all we resort to the hypodermic syringe. Recently some results have been noted from lumbar puncture. During the periods free from attacks, forced feeding should be employed as far as possible. For the *laryngeal crises*, inhalations of chloroform or ether, spraying with cocain, galvanization of the cervical cord and of the pneumogastric and sympathetic nerves, narcotics, bromin,

[1] The remarkable observation of Stembo (*Neurol. Centralbl.*, 1904, p. 303), that after 28 injections of antirabic medulla emulsion in a case of tabes dolorosa the pains entirely disappeared, must be mentioned as a curiosity! French authors, Oberthur and Bousquet, have lately recommended subcutaneous injections of *sodium nitrite* (1–10 cgm.) for the pains as well as for other tabetic symptoms.

etc., may all be tried. For the *anovesical* and *clitoral crises,* local applications, sedatives, the salts of bromin, codein, heroin, and morphin are recommended.

In the cases with *paresthesia* and *anesthesia* electricity by means of the faradic brush, or the labile application of the galvanic cathode, etc., forms the best remedy. Friction, massage, irritative inunctions (veratrin with spiritus formicarum), may also be beneficial.

We will rarely succeed in aborting the progressive blindness caused by *atrophy of the optic nerve,* although in a few instances energetic treatment with mercury and potassium iodid has been known to arrest the affection for some years. Many authors, as well as I, have seen such cases. The statement that this treatment increases the visual disturbance is unwarranted; nevertheless successes are rare. Besides prolonged galvanic treatment of the eyes, subcutaneous injections of strychnin, and the application of derivatives to the nape of the neck, etc., are advisable.

In so far as they do not yield to the treatment of the underlying affection, the *paralyses* which occur in the course of tabes, the various paralyses of the ocular muscles, the atrophic paralyses of the extremities, the paralyses of the muscles of the tongue, of the muscles of mastication, of the larynx, the bulbar paralyses, etc., should be treated according to the usual methods—either with the electric current, injections of strychnin, baths, massage, or exercises.

More difficult is the treatment of the chief symptom of the second stage of tabes—the *ataxia;* this merits a detailed description, particularly because the results recently attained have in so many cases been exceedingly gratifying. We must not forget that we are here chiefly treating merely a symptom, although one of the most important and distressing, and that its removal conduces but little to the improvement or cure of the underlying affection. In this respect many authorities greatly exaggerate the treatment of ataxia, which they make too prominent. We might almost believe that there is absolutely no other rational treatment of tabes than the treatment of the ataxia; i. e., the treatment of a symptom which sometimes appears only 5, 10, or even 20 years, after the onset of the affection!

Sometimes ataxia appears early, and in a few weeks or months *rapidly develops* to an extreme degree, perhaps in consequence of over-exertion, sexual excess, trauma, or the like. In such cases I advise complete and prolonged rest; the patients should remain in bed for weeks, certainly should be almost always in the recumbent posture, and should avoid all use of their legs. Under this treatment the ataxia may rapidly recede, and when this has occurred and the condition has become stationary the true treatment of ataxia should begin. Of course, we must never allow the debility and hypotonia of the muscles, the loss of energy of the patient, etc., to reach an extreme degree.

The conditions are different in chronic cases of *slowly progressive* ataxia, or when this has already developed. In these cases particularly, a decided advance in treatment has been brought about by means of Frenkel's "reeducation method." It is greatly to Frenkel's credit to have formulated this "gymnastic treatment" of ataxia, and to have established it on such a sound theoretic and practical basis.

In 1876 v. Leyden expressed the opinion that ataxia might in part be compensated for by developing and strengthening the muscles; here, it seems to me, he had especially in mind the increase of power in the muscles.

Frenkel's method, on the contrary, consists in gradually restoring by *practice* the use of the coördinating muscles, especially to lessen the immoderate muscular contractions which form such a prominent feature of ataxia. But to Leyden belongs the honor of having correctly appreciated Frenkel's method when first reported in 1889–90, for having combined it with his own idea of a possible "compensation for ataxia," and for having called the attention of the scientific world to the method. He recognized that Frenkel had brought a new factor—exercise—into the therapy of this disease.

Certainly Leyden and his followers, Goldscheider, Gutmann, Jacob, and others, paved the way for the general recognition in Germany of Frenkel's practice therapy; while in France, following Hirschberg's comprehensive researches, it was the School of the Salpêtrière which, under Raymond's direction, developed and broadened the method. I cannot here detail the history of this method.

"Practice therapy" consists in *learning by systematic practice coördinated and proper movements in place of ataxic ones*; as this is really the practice of coördinated movements which have been lost, the designation chosen by Raymond, "rééducation des mouvements," appears to be most fitting.

The tabetics can to some extent control their ataxia, and are able to compensate for it, by the energetic exercise of their muscles, by concentrating their attention on their movements, and by using their eyes to control them, etc.; we know this from the mere observation of any ataxic. As a rule, however, the patient succeeds only imperfectly, and here practice in gymnastics is valuable since it enables him to attain better results, for he is trained to perform coördinated movements with the least possible muscular exertion.

Theories regarding the results of practice therapy are naturally based on our views of the nature of the tabetic coördinative disturbance. We have previously discussed these, and have shown them to be very complicated, and by no means thoroughly understood; but no matter what our opinions regarding ataxia, the benefits from exercises are readily apparent. We note daily that the fineness of our cutaneous and muscular sensations, as well as those of the special senses, may be greatly increased by practice, and also that by practice the fineness and precision of our most complicated movements may be extraordinarily increased. Therefore this depends either upon practice or an increase of the susceptibility to centripetal stimulations, so that "the regulating central apparatus contents itself with a minimum of sensory impressions" (Frenkel), or upon a vicarious sensory stimulation which is still retained (that is, a greater implication of the sense of feeling, and especially of the sense of sight); and, in regard to a new and changed exercise of the coördinative centers and conduction tracts, of the practice of a more rigid control, of a proper distribution, and the more accurately gauged strength of the individual centrifugal (motor) stimulations. The principle is this; that by frequently repeated, perfectly understood, and well controlled movements, the patient's mind being concentrated upon them, both centripetal and centrifugal stimulations are reawakened, refined, rearranged, or conducted into new tracts, and this reestablishes coördination. Therefore neither the strength of the muscles nor the activity induced by practice is the main condition (although these are not unimportant); but, by the production of sensory control and a compensatory sense of sight, a greater sensitiveness and pre-

cision, and especially the proper distribution and strength of individual muscular activities in the production of complicated movements, is attained; here too slight, as well as too great, exertion is referred to its proper source. Hence, the essential point is practice for the acquirement of an easier and improved coördination of movements.

That this presupposes a fine method, a strictly systematic process, which should be constantly under the direction and control of the physician, has been shown by the development of practice therapy. After determining the exact nature of the motor disturbance, we plan all the details of the individual movements. Naturally this plan will vary according to the severity of the case and the degree of ataxia; we must proceed gradually from the milder to the more severe exercises, from the use of individual muscles to that of a larger number of muscle groups; in the severer cases, exercises in the recumbent posture and while sitting should precede those while standing and walking; exercises in maintaining the equilibrium and in balancing the body require special attention. Leg exercises are more important, and usually more necessary, than those of the arms; both are based on the same principles, although the methods and aids are different. It is well to utilize the intelligence of the patients, as well as their understanding of the processes in coordination of movements; this enables them to comprehend what the reeducation method requires of them, and often proves of great service to the physician.

Above all, *the patient must avoid over-fatigue,* chiefly for the reason that in tabetics "the sensation of fatigue" is usually lacking; therefore they are specially liable to over-exertion, and this is most injurious. This necessitates the supervision of a physician or of a well-trained nurse; the practice of these exercises can rarely be left to the patient. Certainly from time to time it should be superintended by the physician. Since this treatment must be carried out very energetically as well as carefully, and since, as a rule, no decided benefit can be seen until weeks or months have elapsed, it calls for great patience on the part of both the physician and patient. For the latter constant watchfulness, and for the physician a periodic supervision are necessary if good results are to be secured.

The scope of this article does not permit me to detail individual methods. I can give merely an outline of the process, and must refer the reader to excellent manuals in regard to these gymnastic exercises; above all, to the explicit work of Frenkel,[1] to the monographs of Leyden,[2] of Goldscheider,[3] of Jacob,[4] of Hirschberg,[5] of Gräupner,[6] of Stintzing,[7] and especially to that of O. Förs-

[1] "Die Behandlung der tabischen Ataxie mit Hilfe der Uebung." F. C. W. Vogel, Leipzig, 1900.

[2] "Die Tabes dorsalis." Berlin, Urban & Schwarzenberg, 1901.

[3] "Anleitung zue Uebungsbehandlung der Ataxie." 2. Aufl., Leipzig, Gg. Thieme, 1904.

[4] "Physikalische Therapie der spinalen Erkrankungen." "Handb. d. physik. Therapie," Leipzig, Gg. Thieme.

[5] "Traitement mécan. de l'ataxie locomotrice." *Bull. génér. de Thérap.*, 1893, 30. Janv.

[6] "Die Behandlung der Gangstörung bei der Tabes mittels der Uebungstherapie." *Allg. med. Centralztg.*, 1898, Nr. 38.

[7] "Behandl. der Erkrankungen des Rückenmarkes," etc.; "Handb. d. Therapie innerer Krankheiten von Pentzoldt und Stintzing," 3. Aufl., V, 1903.

ter[1] which gives all the details. These contain accurate descriptions and plans, and various more or less ingenious apparatus are described, so that any physician who desires to study the subject will here find the necessary directions. There is nothing to prevent him from carrying out this treatment, but he cannot do it without carefully studying the method. If unable to do this, he should send his patients to a specialist, or to one of the institutions which are now everywhere established for the carrying out of exercise therapy.

In the legs only the simplest exercises performed in a slow, measured way on command are at first possible. These consist of the flexion and extension, the abduction and adduction, the elevation and lowering of all the joints, the touching of definite points or of articles such as ten-pins with the tip of the foot, the performing of lateral and oblique movements, describing circles and other figures in the air, drawing figures upon pasteboard or tracing them upon the floor (circles, spirals, squares or other angular figures, numerals, stars, etc.), catching with the tip of the foot balls or coins which are moved to and fro, and the like. Then follow exercises in standing, rising and sitting down, in knee flexion, first with, then without, support and the aid of the eyes. Finally there is practice in standing and walking, sometimes by means of a walking frame or other support made for this purpose, sometimes with the aid of figures drawn upon the floor, or especially constructed linoleum, etc. Then follow exercises in standing with the feet widely separated, afterward more closely approximated, finally with the feet close together; then standing upon one foot, upon the heels, upon the toes, at first with support and open eyes, then with the eyes closed; lastly in walking with the support of apparatus, the help of the physician, a stick, etc., taking carefully measured steps forward and backward, walking in a straight line, then so as to describe certain figures, placing one foot immediately in front of the other, etc.

All of these exercises necessitate the closest attention on the part of the patient, and, therefore, the supervision of a physician; at first they are very exhausting, and should be practised only one to three times daily, beginning with a few of them which are practised five or ten times, later twenty to forty times, then gradually extending the exercises to manifold combinations with intervals of rest; these periods of practice should not be longer than from 15 to 30 minutes, and at the conclusion of the exercise mild massage of the muscles should be given to relieve the sensation of fatigue (Hirschberg).

When there is ataxia of the arms, finer and more complicated exercises are necessary, and for these Frenkel has proposed ingenious apparatus and methods. These comprise at first flexion, extension, and spreading movements of all the joints of the hands and fingers, and touching the tips of the fingers with the thumb; then more complicated movements, such as the tracing of lines, figures, and circles upon paper, using a pencil in a groove or upon an edge, touching objects held before the patient, catching balls which are in pendulous motion, playing ball; upon command touching a series of grooves in a board, sticking corks in holes, assorting coins, counting money, shuffling cards, playing the piano; finally, writing and drawing. These patients must also be taught the finer movements of the hand necessary in daily life, such as those in dressing and at table, with attention and control.

[1] "Die Physiologie und Pathologie der Coördination," Jena, 1902.

This treatment by exercise is very beneficial to many tabetics; we might say to nearly all who are ataxic. The earlier it is begun the better, and the more energetic, self-reliant, and hopeful the tabetics are, the greater the chances of success; but there are certain *contraindications.* When the ataxia is very recent and acute, and the patients are debilitated, weak in muscle, and have lost energy, when arthropathies, muscular atrophy, gastric or other crises are present, when the affection has reached the third stage, when there is atrophy of the optic nerve, advanced arteriosclerosis, valvular lesion, or cerebral disease, this treatment is never in place.

There can be no doubt that in many cases of ataxia this practice therapy brings about distinct improvement; the results are sometimes astonishing. Evidently suggestion and the removal of a purely psychical inhibition to coordination play a certain rôle. This, however, does not lessen the beneficial effect of the treatment. The general condition of the patient may be decidedly improved, as well as the hypotonia of the muscles and the flaccidity of the joints. But I am under the impression that in our enthusiasm regarding exercise treatment the importance of our results has been exaggerated, as is the case with many therapeutic innovations. In not a few instances the benefits are slight or not at all apparent, in many cases they are only temporary, the ataxic loses patience and courage, he discontinues the exercises, loses control of the individual movements, and, finally, improvement ceases. The next ten years will show what permanent results may be expected from the reeducation method in tabes.

Frenkel and v. Leyden have also advised exercise treatment as the best *prophylaxis* for ataxia. It should be instituted in the pre-ataxic stage, at all events as soon as ataxia appears, so as to conserve for the patient as long as possible his natural coördination, to enable him better to control and more accurately to perform movements, to guarantee to him a greater precision, and to show the effect of these movements. In the first stage, when there is but little disturbance of movement, it is difficult to induce the patient to undertake this tedious treatment.

The question in how far the exercises can be combined with other curative methods—for instance, with baths, hydrotherapy, galvanism, massage, or internal remedies—must be decided according to the circumstances of each individual case. As a rule, there is no objection to the use of tonics, change of climate, or a mild water treatment, and electricity is in most cases well borne. Massage properly given is beneficial for the exhausted muscles after exercise. We should be very cautious about recommending a simultaneous spa treatment. Certainly treatment at Nauheim and Rehme, especially the more powerful methods (Sprudel baths and Sprudel needle baths), are contraindicated. They exhaust the patient, and, moreover, it is unwise to combine them with exercise treatment; in my experience this is frequently injurious to the patient. The same is true of the use of Hg and iodin. I must, therefore, issue a warning against attempting too much in this condition. For practice therapy, fresh and recuperative powers are necessary.

Bladder and sexual disturbances frequently demand treatment; except for a possible catarrh the former consist chiefly of deranged innervation and debility of the detrusor and sphincter muscles; they are frequently benefited by the use of electric currents applied according to well-known methods (but

not intravesical!); ergot and strychnin are also advised for weakness of the detrusor, and the latter is a valuable remedy for lack of power in the sphincter.

With extreme weakness of the detrusor and partial retention of the urine, we should first attempt to relieve the bladder by manual compression from the abdomen, which is usually successful; this should be followed by the early employment of the catheter, but with the greatest care in order to guard against cystitis. By this process we may often prevent or stop the very unpleasant dribbling of urine. Massage of the bladder (bimanually from the rectum and abdomen) is sometimes beneficial, as well as treatment by suspension.

Sexual disturbance in those rare cases which, in the initial stages, show increased sexual irritability and libido or frequent pollutions must be energetically treated with large doses of bromin, camphor, lupulin, or heroin (0.01 or 0.02 given at evening), cool sitz baths, a regulated diet, and the avoidance of all sexual excitement.

In the much more common cases of diminished or lost potency it is usually advisable not to restore the sexual activity of the patient; nevertheless in some cases this may be attempted—and, as I have often noted, with success; by suitable hydriatic applications and electricity, by the careful use of nux vomica, of tonic pills, of preparations of arsenic, and by suspension, which, in this class of cases, gives astonishing results. I must urge caution in the use of yohimbin by tabetics.

For the obstinate constipation almost always present, the usual dietetic and physical methods, also drugs, are available. It is better to withhold the last as long as possible, and at first to employ only physical remedies (massage, hydrotherapy, faradism, enemata) with a regulated diet. The later stages always give abundant opportunity for the use of purgatives.

Muscular atrophy is a trophic disturbance which must usually be combated, and electricity, massage, injections of strychnin, friction, and all kinds of gymnastic exercises, are useful. The arthropathies so commonly noted are not amenable to treatment. They are progressive; but in their first stages may sometimes be favorably influenced or arrested. The most successful treatment is by complete rest of the joint, mild compression, counter-irritation with iodin, inunctions of iodin vasogen, or ichthyol ointment, and, occasionally, various surgical measures (puncture of the hydrarthrosis, resection, etc.). As a rule, such surgical treatment is unsatisfactory; then nothing remains but orthopedic relief by suitable apparatus. These often help the patient for a long time.

Bed-sores are to be treated according to the well-known surgical principles (cleanliness, avoidance of pressure, antisepsis, etc.); the same applies to the perforating ulcer of the foot, mal perforant, which is occasionally observed, and in which, according to my experience, treatment by mercury and iodin combined with suitable antiseptic dressings, etc., is often very beneficial. Chipault has recently treated most successfully several cases of this complication by stretching the nerves of the leg.

In conclusion, a word concerning the psychical treatment of tabes. In a severe disease of such prolonged duration, this is naturally of the highest importance. It is only wise and humane to conceal from the patient as long as possible the gravity of his affection, but we must impress him with the

importance of submitting to the necessary treatment, and of carrying it out persistently. We need not mention locomotor ataxia or tabes. I usually tell the patient that he is suffering from an irritation of the nerve roots of the back, or of the membranes of the spinal cord, and that by suitable treatment this may be greatly relieved, but I let him understand—particularly if he is easy-going—that, under some circumstances, the affection becomes very much aggravated.

We must always seek to inspire him with the hope of improvement and recovery, with confidence in his physician, and in the mode of treatment pursued. Variation in the treatment, regular but light exercise, harmless enjoyment, change of residence and scene, the stimulation of varied interests, driving, pleasant company, the theatre, music, etc., often serve to keep up the courage of the patient. In tabes little is to be expected of true suggestion, especially of hypnotic suggestion. But, without this, a skilful, tactful physician will be able to exercise a certain authority over the unfortunate sufferer.

Following this almost endless enumeration of remedies and methods in tabes, a few words are permissible concerning the general plan of treatment. This to some extent is shown by the foregoing presentation. But it must be modified according to the individual circumstances, external conditions, the position, mode of life, and the social, pecuniary, and family relations of the patient.

Therefore the individual indications are to be considered carefully, and no means necessary to the desired end should be neglected. The long duration of the affection and its many complications make it our duty carefully to choose the method of treatment, bearing in mind the change of seasons; a variation in the methods may serve to reawaken hope in the patient. The psychical elements of the case demand close consideration. The uncertainty of success, the difference in the reaction of individual patients, justifies us in trying various methods of treatment. Their results will shape the further process; treatment productive of benefit should, naturally, be continued; that ineffectual should be discarded.

Under any circumstances, we must caution the patient against over-exertion. Different methods are not to be resorted to indiscriminately or employed simultaneously, although all combinations are permissible.

The treatment, at least in patients of the better classes, can often be carried out in spas or other institutions for the treatment of nervous diseases; here much depends upon the choice of the place and the physician. For the lower classes, treatment in clinics and in hospitals is preferable to that in their own homes.

In the first stage of the disease, and especially at the onset, we must seek a causal indication; should this necessitate antisyphilitic treatment, it must be carefully but energetically undertaken, and, under some circumstances, must be repeated after intervals; these pauses can be utilized for treatment by tonics, a change of air, a bath cure, or electricity. A choice between hydrotherapy and thermal salt or other baths will depend upon the circumstances and the season of the year. In winter galvanism, the administration of various drugs, the application of derivatives, suspension, perhaps also slight friction or artificial CO_2 baths, may be resorted to. For wealthy patients we must consider the advantages of change of climate. In any case the patient's mode

of life should be strictly regulated, and every attention should be paid to the symptomatic treatment of the individual case.

In the second or ataxic stage specific and tonic treatment may alternate. But, as a rule, the practice of exercises should supplement these, and form the chief treatment. In addition, we may try any other curative measures: in a warm climate treatment by water and baths, in winter electricity, massage, suspension, and drugs. Careful regulation of the diet, so as to improve the general nutrition, and the consideration of the psychical condition of the patient are necessary, besides meeting the symptomatic indications which now fill a wide field.

In the last stage, the hopeless patient who seeks relief should be spared exhausting treatment, journeys to spas, and expensive special treatment, and we should advise only the most necessary change of scene. Massage, electricity, and tonics may here be of benefit. Our chief reliance must now be upon symptomatic therapy and the psychical treatment of the sufferer. In the latter sense it may occasionally be desirable to institute a new mode of treatment, so as to arouse hope in the patient and divert his mind. This may be more or less effective, and even in the last stages the tactful and humane physician may be able to give such relief that to his patient he appears to be a helpful and sympathetic friend.

MULTIPLE SCLEROSIS

By E. REDLICH, Vienna

Multiple sclerosis is in many respects of especial interest. In the first place it is one of the most common affections of the central nervous system; but the cases of multiple sclerosis in which, considering all the circumstances, the diagnosis can be made with certainty are far outnumbered by those in which the differential diagnosis may be perplexing. And, moreover, the clinical aspect of multiple sclerosis may vary considerably; this variation, which often furnishes surprising clinical pictures and impresses its stamp upon the course of the disease, necessitates an explanation; hence, from clinical observation and the pathological anatomy of multiple sclerosis interesting questions may arise which involve the general pathology of the nervous system.

Cruveilhier, in his celebrated Atlas, was probably the first to describe anatomical preparations in cases of this disease and to furnish illustrations. Frerichs, Leyden, Rindfleisch, Schüle, then Vulpian and others, reported additional cases. Charcot, with his peculiar intuitive faculty, must be credited with having separated the mass of symptoms presented in the peculiar symptom-complex, and clearly defined the typical clinical picture of multiple sclerosis. Above all, it became possible by this means to diagnosticate multiple sclerosis from paralysis agitans with which it had been commonly confounded; Charcot's contributions to our knowledge of multiple sclerosis naturally extend much further; for, at the same time, he gave us an insight into the pathology of this disease and its definite histological landmarks; for example, the retention in the sclerotic foci of the axis cylinder, this being fundamentally necessary for our understanding of the affection. Since Charcot described multiple sclerosis we have acquired additional knowledge of it, details of which will not now be given. But one point is of special significance. The clinical picture, delineated by Charcot, is certainly typical of many cases but not of all, and certainly not of all stages of multiple sclerosis, the course of which may be protracted for many years. Subsequently other authors, among whom were Oppenheim and Bruns, showed that other groups of symptoms might frequently be found in multiple sclerosis, perhaps as often as Charcot's picture, that the so-called "formes frustes" of Charcot have an important place in the clinical description of multiple sclerosis, and that among these pictures, which often vary so decidedly, there are many very different from the Charcot type, yet which must be regarded as typical cases of multiple sclerosis. But of this later.

ETIOLOGY

On considering the distribution of the disease it may be stated that the sexes are almost equally affected. Charcot reported that the female sex showed a special predilection, and this was confirmed by Berlin (among 39 of the latter's cases 26 were women). More recent statistics, however, do not coincide. For instance, Lent had, among 51 cases, 37 men and 14 women; Krafft-Ebing had, in 100 cases, 58 men and 42 women; 12 of my 23 cases were men. Frankl had, among 206 cases, 140 men and 66 women; Probst, in 58 cases, 34 men and 24 women; Bruns-Stölting, among 38 cases, 13 men and 25 women. This does not show a preponderance of the female sex, but, on the contrary, of the male. I must emphasize that multiple sclerosis, as a rule, occurs somewhat more frequently among the poorer classes than the well-to-do.

The question of its *age incidence* is more interesting. Charcot stated that the disease is more common in the young, which succeeding authors have confirmed. This is especially significant from the circumstance that in multiple sclerosis which has persisted for years the history often shows that its onset dated back many years.

Since, therefore, the majority of cases of multiple sclerosis are between 20 and 30 years of age when they come under observation, we may certainly assume that in many cases the onset was much earlier, even in childhood. In spite of the difficulty in diagnosticating multiple sclerosis in childhood, it may be positively maintained that typical multiple sclerosis does occur in children.

Marie, Nolda, Unger, Frankl-Hochwart (8 cases with onset between the first and tenth years of life), and others have reported such cases; Zenker, Schüle, Humphrey, and Eichhorst have confirmed this by anatomical findings. But it must not be forgotten that multiple sclerosis is also observed in later life, hence the statement of Charcot that the affection rarely occurs after the fortieth year is erroneous, but was apparently due to accidental circumstances. Cases at fifty years of age, and often after sixty, have been described, among them an entire series which were proven by autopsy to be typical multiple sclerosis, cases in which the history removed all doubt that the disease had developed during later life.

After determining the general etiologic limit, we should ascertain the actual cause of the disease; and here we encounter difficulty. First, an important question must be decided. Are we dealing in multiple sclerosis with an *exogenous* or an *endogenous* disease, i. e., is multiple sclerosis due to external cause in persons previously entirely healthy, in whom there might be at most a certain predisposition, or are such factors unnecessary? Is it a faulty predisposition which produces a pathologic degeneration of the central nervous system, and does this, without external lesion, perhaps from internal cause or merely functional use, cause multiple sclerosis? The majority of authors incline to the first opinion, but the theory of the endogenous nature of multiple sclerosis has also found prominent supporters who base it upon the circumstance that numerous cases of multiple sclerosis are observed in which the etiologic factors known to us, even allowing the greatest latitude, are inoperative.

First, from the anatomical standpoint of Ziegler and Jutzler, congenital

anomalies with areas of unusually close neuroglia have been thought to be responsible for the development of sclerotic foci. Strümpell is decidedly of the opinion that multiple sclerosis is an endogenous disease. He observed the concurrence of hydromyelia, a congenital anomaly of the spinal cord, with multiple sclerosis, and this led him to suspect its endogenous origin, a view which was further confirmed by the appearance of the disease in childhood and by the persistence of the axis cylinders, which in his opinion is not the case in exogenous diseases. Although Hoffmann also recently inclines to this theory, I must confess that I do not consider it well founded.

The factors mentioned by Strümpell are soon discussed. The coincidence of hydromyelia and multiple sclerosis is exceptional, therefore of no significance. Hydromyelia is found in every other disease, even in persons with sound nerves, just as frequently as in multiple sclerosis; furthermore, it is not true that axis cylinders do not occur in exogenous diseases. It is sufficient to call attention to the periaxial neuritis of Gombault in experimental lead poisoning, in which affection the axis cylinders remain perfectly intact and the medullary sheath alone is diseased. Multiple sclerosis in childhood may readily be due to other factors (see later).

The assumption of an endogenous cause of a disease, a reference of it to congenital factors, must be based upon very weighty reasons, especially clinical, if it is to constitute an etiologic explanation; for the anatomical view of a "predisposition" to the affection under discussion has little foundation. Clinical experience, therefore, in the great majority of cases, is opposed to the view of an endogenous origin of multiple sclerosis.

Those affections in which, considering all that is known, we feel justified in assuming an endogenous origin often prove, in the absence of all other causes, to be of hereditary, family nature, or, at least, we recognize a strong hereditary predisposition in the affected individuals. This is not the case in multiple sclerosis. We have an example of direct heredity in an interesting report by Eichhorst of a mother and child in whom the existence of multiple sclerosis was verified by anatomical investigation. Without wishing to detract from the value of this observation, it seems proper to state that in the mother the puerperium played a rôle in the causation and development of the disease, a factor which we also encounter in the etiology of multiple sclerosis. In the mother, therefore, multiple sclerosis was probably of exogenous origin. In a second report of hereditary multiple sclerosis affecting a mother and child, and described by Ella, the cases were only clinically observed; the cases of Cestan and Guillain, in which two children in one family showed the symptoms of multiple sclerosis, were not confirmed by the necropsy.

The same is true of other isolated reports of the family appearance of multiple sclerosis. Other authors unanimously admit that hereditary predisposition plays no special rôle in the etiology of multiple sclerosis, i. e., as a rule, persons affected by the disease show no marked predisposition, and these alone come seriously into question.

In reviewing the other factors considered with more or less justice to be the causes of multiple sclerosis, it is evident that in the course of time views have greatly altered, that much which played a prominent rôle in earlier literature has to-day been relegated to the background, and much has been emphasized without this change having shaped our views. The *psychical fac-*

tors formerly enumerated, fright, excitement, etc., may be disregarded. We must admit that multiple sclerosis, heretofore latent, may appear after psychical emotion or psychical trauma, but we can hardly ascribe to these factors an actual causative rôle. The influence of *exposure to cold* is different. In earlier literature this played a significant, and even the main, rôle in the etiology of multiple sclerosis, as will appear to any one who studies the works of the early writers; with them contracting a cold was one of the most important etiologic factors. All this is changed to-day; refrigeration has more and more been replaced by causes which could be better substantiated. But although this is true, and it is to-day impossible to determine the precise importance of exposure to cold and dampness, and thus make these a clear pathological formula, yet it must be conceded that these factors have an undoubted etiologic significance in multiple sclerosis. Among recent authors, Krafft-Ebing strongly maintains this. Other authors are more conservative, and believe it to be true in a small number of cases; the majority are unanimous in admitting this factor. Every author of experience has known cases of previously healthy persons in whom, after a more or less severe chilling or wetting, for example, by falling into water or into snow, or being thoroughly drenched by a heavy rain, etc., the symptoms of multiple sclerosis appeared, or they subsequently developed after indefinite symptoms. In other cases it is the same factor working in a more chronic way, for example, by living in damp, draughty rooms, etc.; this is apparently the only cause that can be determined.

Under the stimulation of a report by Marie—Leyden, Kahler and Pick had even earlier reported the same—another etiological factor assumed prominence, that is, the influence of preceding *infectious disease*. The fact that multiple sclerosis occurred more frequently in youth than in later life was utilized to show the etiologic dependence of the malady on infectious diseases, for the number of infections occurring in youth or infancy is much greater than in adult life. Thus the argument used by Strümpell to prove the endogenous nature of multiple sclerosis, probably has another more correct application. Recently the etiologic importance of preceding infectious diseases received a certain setback because of the skepticism of some prominent authors, among whom we must mention Krafft-Ebing, Strümpell and Hoffmann, who have absolutely denied that this is a cause.

Let us investigate the facts. We have long been familiar with the appearance of multiple sclerosis after enteric fever. It has also developed after variola, pneumonia, erysipelas, diphtheria, measles, scarlatina, dysentery, cholera, influenza, angina tonsillaris, rheumatism, etc. There are also reports, especially recent ones, of the relation of multiple sclerosis to malaria. For example, Spiller cites the case of a sailor, aged 40, who presented the symptoms of multiple sclerosis. Anatomical examination revealed the presence of numerous sclerotic foci in the central nervous system, and the capillaries of the brain were filled with the estivo-autumnal parasite of malaria.

The number of cases which individual authors have described as occurring after infectious diseases varies widely. This is due to the circumstance that, only in the rarest cases, can we demonstrate an immediate connection between a preceding infectious disease and the appearance of multiple sclerosis; this was true of a case I described in which, after an angina tonsillaris, probably

of diphtheritic nature, the typical symptoms of post-diphtheritic polyneuritis first developed, and, after some years, multiple sclerosis became manifest. This case finds its confirmation in one on which Henschen held an autopsy, and in which multiple sclerosis and polyneuritis could be demonstrated as following diphtheria. In other cases, relatively soon after the disappearance of the acute infectious disease, an indefinite, pathologic, nervous stage sets in, the persons convalescing from the infectious disease are not restored to perfect health, and subsequently the picture of multiple sclerosis develops. In other cases there is an intervening period, lasting even years, of apparently perfect health between the infectious disease through which they have passed and the appearance of multiple sclerosis, so that we are forced to doubt their etiologic connection. Still, the last mentioned circumstance is not a positive proof of the contrary. It may be stated that multiple sclerosis has a long period of latency. Nothing prevents us from assuming that this is the case because the onset of multiple sclerosis with its vague symptoms is masked by the symptoms pertaining to convalescence from acute infections.

If, on the other hand, we take the view that multiple sclerosis is not the direct consequence of an immigration of microörganisms into the central nervous system, and their local propagation, many factors make it difficult to estimate the influence of infectious disease in the etiology. We may adhere to the importance of this factor in multiple sclerosis although unable to state in how many cases it becomes operative; the number is probably by no means small. In the first sense, we may utilize the fact that an already existing multiple sclerosis may, after an intercurrent infectious disease, sometimes show distinct aggravation and an increase of symptoms.

It is universally admitted that, in contrast with other diseases of the central nervous system, syphilis plays a slight, if any, rôle in the etiology of multiple sclerosis. Aside from the fact that the picture of syphilis resembles multiple sclerosis, there are but few cases in which, with any degree of probability, hereditary or acquired syphilis may be considered an etiologic factor in its development.

A number of cases have been observed in which multiple sclerosis occurred after pregnancy and the puerperal state, and even in afebrile conditions without infection. We have already mentioned a case of Eichhorst's as belonging to this category. Oppenheim, Balint, and others mention the same etiologic factor; and I have also observed such cases. The fact, several times noted, that symptoms of multiple sclerosis become more intense after the puerperal period must be here mentioned. How this influence is to be explained, whether by the foregoing alterations in metabolism, by the return of the genitalia, etc., to their normal condition, or whether toxic products are the cause, cannot be determined; analogous conditions, in a certain sense proofs, are found in the appearance of polyneuritis and various psychoses during pregnancy and the puerperium. This fact leads us to propound another question: Are *intoxications* an etiologic factor in multiple sclerosis? Of the acute poisons we can, in this respect, mention only isolated cases of carbonic oxid gas poisoning which, as we know to-day, under some circumstances promotes the development of other serious changes in the central nervous system (softening, etc.). Chronic alcoholism, certainly, has no special influence. But this is not the case with other chronic intoxications, particularly the *metallic poisons.*

Oppenheim called especial attention to chronic metallic poisoning contracted in an occupation. Here he mentioned lead, copper, verdigris, zinc, and anilin dyes, and he recently reported an interesting case of multiple sclerosis (which I also had an opportunity of seeing) in which the poisoning due to zinc in dyed stockings (traces of zinc were found in the urine) had produced nephritis, and gradually multiple sclerosis.

Recently Embden, and subsequently Jaksch, described a peculiar clinical picture appearing among workers in manganese (Embden calls it a *manganese dioxid, grinder's disease*) which symptomatologically closely resembled multiple sclerosis. Embden, as well as Jaksch, differentiates this affection from multiple sclerosis, although both of these authors assume multiple changes in the central nervous system to be the anatomical foundation. As no autopsy reports are yet available, this point cannot be decided. Nevertheless, from our present standpoint, this affection is not without importance, and may be said to favor Oppenheim's view, for we cannot deny that his opinion has been seriously opposed. In the majority of cases there is certainly no question of chronic intoxication due to occupation for the disease occurs in the young who have not yet been compelled to work or in persons in walks in life which exclude all possibility of such chronic intoxication. Therefore, the number of cases in which this etiology may be considered is certainly not large, but its possibility must be admitted.

We must now mention a deleterious factor which, as was shown by recent authors, among them Kiewlicz, Jutzler, and Mendel, may not rarely produce multiple sclerosis, and this fact is *trauma.* But we must be very cautious in assuming trauma to be the etiologic factor. It is evidently such if a previously healthy individual after severe trauma, for example, a fall from a great height, is not restored to perfect health, but gradually develops all the symptoms of multiple sclerosis. I remember a case that was very characteristic. A previously healthy girl, while on a raft and in consequence of the raft's colliding with another vessel, sustained a fall upon the buttocks. Certain sequels remained, and years later the unmistakable picture of multiple sclerosis developed. As an index of this genesis of the affection, symptoms referable to the lower portion of the spinal cord, such as paralysis of the bladder and rectum and paresis and ataxia of the lower extremities, were very conspicuous. Cases of trauma to the vertebral column in consequence of a fall or blow upon the back or the head should be considered in a similar light. On the other hand, there are cases in which the relation of the symptoms is less clear, either because the trauma was slight or because multiple sclerosis developed too long after. Here decision is very difficult; even after a careful weighing of all the circumstances it can be made only with the greatest reserve. But the etiologic influence of trauma upon the development of multiple sclerosis in a previously healthy individual is in a certain number of cases undoubted, and this is very important in considering accident cases. In differentiating these supposedly traumatic cases we must bear in mind the fact that trauma may greatly aggravate already existing symptoms of multiple sclerosis.

I have now stated the chief and positive points upon which we may base a more or less well-founded assumption of the etiology of multiple sclerosis. In the main these are the same factors to which we attribute many other organic diseases of the nervous system. Why, in one case, their effect is so

severe as to produce an organic, progressive, nervous affection, while in many other cases there are no serious consequences, why multiple sclerosis arises and not another form of organic disease, we are unable to say. But this is not true of multiple sclerosis alone. As a rule, our etiologic diagnosis does not actually correspond with the real condition; an unknown quantity, perhaps several such, frequently remains.

As I stated in the introduction, in a number of cases of multiple sclerosis we are unable to find any etiologic factor, anything which we may with some degree of probability assume to be the cause of the disease. It is to be hoped that the researches of the future will bridge this gap in our knowledge of the malady.

SYMPTOMATOLOGY

We shall now discuss the uncommonly rich symptomatology of multiple sclerosis, and shall first describe the symptoms in regard to their frequency and importance, subsequently their grouping, and the course which characterizes its typical forms.

Among the symptoms emphasized by Charcot because of their frequency we first find *tremor* and *nystagmus.* It is at once evident that these belong together. Charcot aptly described the tremor of multiple sclerosis as an *intention tremor,* i. e., it does not appear during rest but with intended movements. The tremor is most distinct in the upper extremity, particularly in the hands; for example, the arms hang down and are completely at rest, but as soon as the patient attempts to grasp any object a tremor occurs which during the time the motion is being made increases decidedly and is sometimes an actual shaking. The tremor is even more distinct when the patient tries to hold any heavy object, for example, a vessel full of water, when the tremor finally becomes so marked that the patient spills some of the water. It is particularly noticeable in the hands while writing. Specimens of handwriting reveal distinctly this course, gradually increasing tremor, and here it must be mentioned that because of a simultaneous ataxic disturbance the writing is very irregular. The lower extremities not infrequently show this tremor, as well as the musculature of the trunk and the head. Here also its intentional character is generally marked; i. e., when the patient, who has been lying quietly upon his back, rises or walks a coarse, usually increasing, tremor develops, and Oppenheim has characteristically designated this as waddling. Since even standing still requires the continuous innervation of many muscles which serve to maintain the erect posture, here also there is usually a decided tremor or waddling. Oppenheim has reported that reflex movements, for example, the extension of the knee-joint after percussion of the tendon of the quadriceps tendon, sometimes produce tremor. It must be added that an intention tremor is often more fully developed upon one side than upon the other, and may even be unilateral, an observation which is further borne out by the hemiplegic mode of development of multiple sclerosis (which will later be explained) as well as by the fact that in rare cases the intentional type of the tremor is not well marked, but there are certain transitional stages which lead to a more continuous type of tremor.

Intention tremor is a common symptom of multiple sclerosis. Of course,

it is not observed in all stages of the disease; it may be safely said that it is found, at least periodically, in from 50 to 60 per cent. of the cases.

Quite analogous to intention tremor is *nystagmus*. This similarity is in many cases at once apparent, for the oscillation of the eyes, designated nystagmus, does not appear when the eyes are quiet, but occurs only upon turning them laterally or upward. If the patient rolls the eyes to the extreme left, there is sometimes only a to and fro motion while in this position, and it is soon arrested; in other cases, however, this motion ceases only when the eyes have returned to their former position. The same oscillation is seen when the patient looks up or down. In other cases *rotatory nystagmus* occurs, the rapid oscillations succeeding each other in a varying excursion. Here we may mention a movement described by Kunn, in which two or three lightning-like, horizontal twitchings appear when the patient attempts to gaze steadily at any object. The same author refers to tremor of the ciliary muscle—certain disturbances of sight when looking at close objects.

In the frequent cases in which horizontal or rotatory nystagmus occurs when the eyes are directed forward, we have a condition analogous to tremor on standing still, for even when the glance is directed straight forward a certain continuous muscular innervation is necessary.

The majority of authors estimate that nystagmus, as a conspicuous but by no means typical symptom of multiple sclerosis, occurs in about one-half of all cases.

To the motor disturbances we must now add others, first, those which occur in the extremities. Charcot called attention to the frequency of *spastic pareses* in the lower extremities; and subsequent investigations have confirmed their importance. In multiple sclerosis, at least in some stages, more frequently in the later but sometimes rather early, there are more or less well developed pareses, the nature and appearance of which of course vary; when the lower extremities are attacked there may be all degrees, from slight implication of motility to the most profound paralysis. Often the sides are not uniformly affected; one leg may be more involved than the other, or the affection may be confined to one leg alone. While, therefore, in the milder cases, the patients note only a weakness of the affected leg, an increased lassitude and fatigue so that the legs become stiff on walking for a short time, in severe cases the power of locomotion decreases more and more, the gait becomes remarkably spastic-paretic in character, and the footsteps are short and dragging; finally the paralysis becomes complete and the patient is bedridden. The paretic legs, as a rule, exhibit distinct, very often conspicuous, spasms and contractures, and even passive movements meet with resistance; the tendon reflexes are greatly increased; where the implication is not uniform they are usually most increased upon the side most markedly affected; we also find patellar and foot clonus. With severe spasm we frequently note Babinski's toe-reflex. Well developed flaccid paralysis is seldom seen. Loss of the tendon reflexes is very rare. Marked atrophy is scarcely ever observed and moderate atrophy only exceptionally.

Paralysis of the lower extremities often develops gradually, and increases in the course of years. If the disease is not steadily progressive, well developed pareses may disappear or, at least, their intensity is lessened. But a more acute onset of paralysis in the lower extremities has been noted, even in rela-

tively early stages, and in this complication improvement or complete disappearance of the paralysis is sometimes observed.

To the motor disturbances due to spastic paresis of the legs, in quite a number of cases *ataxic symptoms* are added. I must call especial attention to this combination of spastic paresis and increased ataxia as a common symptom in multiple sclerosis. As a rule the ataxia resembles the cerebellar form. There is a swaying, particularly upon walking, a deviation from a straight line which may increase to actual waddling, but is less unsteady than in tabes dorsalis. In these cases, the patient while standing feels very insecure, especially when the space on which he stands is reduced by his placing his feet in juxtaposition when, without closing his eyes (unlike the case in tabes dorsalis), there is a marked increase in the swaying. With unilateral paresis we occasionally note that the ataxia also is chiefly one-sided. In some cases ataxic phenomena are observed without especially marked paresis. It must be added that in isolated cases v. Frankl-Hochwart noted a gait which is usually typical of paralysis agitans, namely, retropulsion and propulsion, i. e., a backward and forward movement of the patient which, after a corresponding intention or after a slight push, causes him to break into a run either backward or forward.

Paresis of the upper extremity is less prominent among the symptoms of multiple sclerosis, although it is by no means rare, and is more often unilateral than bilateral. In the cases of common hemiplegic type there are pareses in the arms and legs of the same side. I must emphasize a not unusual distribution of the paralysis—paraparesis, or paraplegia, of the legs and paresis of one arm; sometimes this develops from the hemiplegic type in such a way that the more marked affection of the leg corresponds to a paresis of the arm upon the same side. In severe cases there are usually spastic symptoms in the upper extremities; contractures develop and the tendon reflexes are increased. What has been stated of the appearance and course of paralysis of the lower extremities is also true of that of the upper extremities.

We have already mentioned that the addition of ataxia to the usual intention tremor causes an impairment in the use of the arms; but it is not always easy here to discriminate between tremor and ataxia. Some authors, for example, Strümpell—in my opinion erroneously—have gone so far as to consider intention tremor and ataxia identical. Oppenheim has occasionally observed an acute hemiataxia of the upper and lower extremities.

Marked atrophy of the upper extremity is also rare. It is most often found in the small muscles of the hand, and was described by Charcot. Some cases have been reported which resembled the picture of amyotrophic lateral sclerosis; Charcot mentions a case which simulated spinal muscular atrophy.

Among the *spinal paralytic phenomena, disturbances* of the *bladder* must be mentioned. These were formerly declared to be rare, but we know to-day that they may be very common in multiple sclerosis. Frankl-Hochwart and Zuckerkandl noted them in four-fifths of the cases, especially with paraplegia of the legs. As in other pareses, these may be temporary paralytic conditions which appear and rapidly disappear, or, especially in the later stages, such disturbances may persist. As a rule, there is dysuria, difficult and prolonged urination, sometimes with strangury and intact sensibility of the bladder; in other cases there is incontinence of urine with a constant dribbling, particularly in the terminal stages of the disease. Impairment of the activity of the

rectum is not very common, being usually seen only in the form of chronic constipation.

In comparison with the motor disturbances, *sensory disturbances* are less important. Charcot's opinion that these are usually absent has not been confirmed, as we know, by the investigations of Oppenheim, Freund and others; but these sensory disturbances are rarely intense or conspicuous, and they can sometimes be discovered only after thorough and repeated investigations; often they are only transitory, existing for weeks and months, and then disappearing. Sensory disturbances may appear either with the late or the early symptoms. The patients complain of paresthesia, burning, formication, numbness in one or the other leg (the periphery being less frequently attacked), more rarely there is pain which, however, is not lancinating.

Examination not infrequently discloses objective disturbances of sensibility, such as a uniformly diminished sensation to all stimuli. In the later stages, particularly when the paraplegia is severe, there is in some cases total anesthesia of the legs. In isolated cases disturbances of thermal sensibility preponderate.

Here and there the sensory and motor disturbances are distributed according to the type of Brown-Séquard's paralysis. We sometimes find a combination of multiple sclerosis and hysteria which may naturally produce extensive sensory disturbances, a point to which we will revert when discussing the differential diagnosis.

The *cutaneous reflexes* are rarely altered; the occurrence of Babinski's sign has been previously mentioned. *Vasomotor* and *trophic disturbances* of the skin are rare or may be wholly absent. Bed-sores occur only in the terminal stages of the disease in patients long bedridden.

Disseminated foci distributed over the spinal cord and brain are characteristic of multiple sclerosis. It, therefore, becomes necessary to describe the *cerebral disturbances*, especially those prominent in the symptom-complex of the affection under discussion.

On account of their frequency and diagnostic importance, symptoms referable to *the optic nerve* must primarily be considered.

In his reports Charcot called attention to the importance of changes in the optic nerve and their clinical characteristics; Uhthoff, with his pupil, Lübbers, has lately investigated this subject, and given us very comprehensive reports. Among other authors who have done much to increase our knowledge of these disturbances I must mention Oppenheim, Frank, Bruns, and Stölting. Statistics reveal their importance. Uhthoff demonstrated ophthalmoscopic changes in the optic nerve in 45 per cent. of all his cases; Bruns and Stölting report disorders of vision as the first symptom of multiple sclerosis in 30 per cent. of their cases.

In this description we shall differentiate between demonstrable ophthalmoscopic changes in the optic nerve and disorders of vision; these by no means always correspond.

First, in regard to the ophthalmoscopic appearance of the papilla, in well marked cases there is sometimes a slight general atrophy of the optic nerve. Frequently this is not symmetrically distributed, but the *temporal* half is pale and distinctly atrophied. This temporal pallor of the papilla is the most characteristic of the ophthalmoscopic findings in multiple sclerosis. Total atrophy,

on the other hand, is only exceptionally demonstrated. But in some cases described by Uhthoff, Lübbers, Oppenheim, and lately by Bruns and Stölting, there was typical optic neuritis, even true choked disc.

We must now mention a point of special significance. The ophthalmoscopic changes may take place very early; for example, an atrophy of the optic nerve or neuritis may wholly or almost entirely disappear, and some time subsequently, in isolated cases even after years, the other symptoms of multiple sclerosis may develop. Between the appearance of changes in the optic nerve, which sometimes may be most acute, and the actual onset of the disease there may be a *stage of latency* of varying duration, even years. In other cases, the optic nerve becomes affected at a relatively late stage and shows a slow but progressive advance. These alterations are more frequently unilateral than bilateral, that is, they are more conspicuous on one side than on the other.

What is true of the ophthalmoscopic changes is also true of the disturbances in sight of which the patients complain; these may come on very early and acutely and lead to total blindness, and then gradually improve, or they may form a late symptom that sometimes, although rarely, shows steady progression. The disturbances in sight are not always due to distinct ophthalmoscopic alterations, and, *vice versa*, notwithstanding evident pallor, even atrophy of the papilla, vision may be very clear. As a rule, however, disturbances in sight correspond with the ophthalmoscopic picture. In the early stages central scotoma, such as we observe in retrobulbar toxic neuritis, is relatively common. The scotomata are rarely total, more frequently they relate only to colors, particularly green and red. In other cases, on the contrary, there is a limitation of the field of vision, rarely concentric, usually irregular. Sometimes a diminution of central acuity of vision is combined with anomalous extension of the visual field. Permanent total blindness is very rare.

Not quite so common as disordered vision but, nevertheless, frequent enough, are disturbances in the *innervation of the muscles of the eye*. Paralysis of the muscles of the eye is sometimes an early but transitory symptom, so that we not infrequently hear the patient with multiple sclerosis state that he long ago had double vision. Often there is partial paralysis of the oculomotor nerve, as a rule unilateral, a paralysis of one abducens, etc. Sometimes there is so-called *associated* paralysis of the muscles of the eye, for example, paresis of the oblique muscles or the muscles of convergence. In isolated cases extensive permanent ophthalmoplegia has been observed. Disturbed *innervation of the pupils*, above all, inequality of the pupils, anisocoria, may be transient or permanent; myosis or mydriasis is sometimes observed. On the other hand, disordered reflex activity of the pupil is very rare; occasionally there is a sluggish reaction to light. Uhthoff once found reflex rigidity of the pupil, twice the reaction of convergence was absent. Probst and Pini have noted an absence of pupillary reaction. As this finding is very rare, it is questionable whether this was not due to complications.

We have previously referred to the frequency of nystagmus, which is due to a disturbance of the innervation of the muscles of the eye.

Isolated paralysis of other cranial nerves than the facial is rare; this is prone to occur in the hemiplegic forms of multiple sclerosis, and has all the characteristics of cerebral facial paralysis (predominant implication of the middle and lower branches) and is relatively common. Facial paralysis impli-

cating all the branches, sometimes combined with simultaneous paralysis of the abducens and crossed with paresis of the extremities (pons!), has occasionally been observed. Spasms in the course of the facial nerve have been described. Oppenheim reported a case with symptoms referable to the fifth nerve (as the first evidence of a developing multiple sclerosis there was persistent neuralgia of the fifth nerve and a sclerotic focus at its point of exit). Affections of the auditory nerve, for example, nervous deafness, are very rare.

We must more minutely consider the disturbances of speech in multiple sclerosis. In Charcot's classic description they occupy a prominent position; above all "scanning speech," i. e., a slow, measured way of speaking in which the different syllables are quite distinct from each other, as in the so-called scanning of verses is significant. This form of speech is very frequent, but not invariably present, nor at every stage. In some cases the speech is exceedingly slow, bradyphasia, and at the same time it is hoarse, low, monotonous, without modulation, or it may be stuttering or explosive. In addition to these speech disturbances there are in some cases rare but significant symptoms of a dysarthritic nature; words are not clearly articulated, some particularly difficult sounds are indistinct; the speech can scarcely be understood; it resembles that observed in progressive bulbar or pseudobulbar paralysis. Sometimes the movements of the tongue are impeded, and there are cases which to a certain degree resemble bulbar paralysis. In these, speech is very indistinct and almost incomprehensible, the lips move slowly, the expression of the face is flaccid, the tongue is markedly implicated, deglutition is difficult (fluids regurgitate through the nose), and the retching reflex is diminished or absent. Paresis of the tensors of the vocal cords was observed by Oppenheim. Increased salivation is noted as well as disturbance of the action of the pneumogastric nerve, such as irregular and accelerated action of the heart, oppressed and jerky respiration, etc. A conspicuous feature of progressive bulbar paralysis is absent in these rare cases of multiple sclerosis, namely, atrophy of the implicated muscles; slight emaciation, for example, of the tongue, has been observed.

We must now discuss some general cerebral disturbances which are quite common in multiple sclerosis, for example, headache. This, as a rule, is neither persistent nor particularly severe. There are, however, cases in which, in the early stages or even as the first symptom, uncommonly severe headache, such as we find, for instance, in tumor of the brain; this, however, may subsequently cease. This headache may be accompanied by severe vertigo, and vertigo alone is often experienced in multiple sclerosis. There is also syncope, but loss of consciousness with epileptiform convulsions is more rare.

Apoplectiform attacks are specially interesting. Sometimes they are early symptoms, or it may happen that they introduce the disease. They may disappear very rapidly, or for a few days cause paralysis of a monoplegic, hemiplegic, or paraplegic character. In some of these cases—for example, when of hemiplegic type—these cerebral paralyses are somewhat more intense and may be persistent. Such apoplectic attacks with paralysis may occur in the further course, even in the late stages of the disease.

Finally, we must mention the psychical disturbances; rarely well developed, they are more common than was formerly believed, and, as has lately been proven, even severe psychoses may be due to multiple sclerosis. A very

frequent symptom—not of psychic nature but, nevertheless, conveying a strange impression—has been designated as spasmodic laughter or crying, for, on the slightest cause, sometimes even without any (for example, when stimulated by the laughter of another person, echo laughter), the patient laughs immoderately. Perhaps the opposite symptom, spasmodic crying, is more often noted. When the patient describes his symptoms to the physician, and, finally, when he is addressed in any way, he cries, and the tears roll down his cheeks. He cries like a child. It takes some time to pacify him and on the slightest cause he begins anew. This curious spasmodic laughter and crying is also seen in other affections, in hemiplegia, in bulbar paralysis, etc. We shall not attempt to explain it, because, in my opinion, this is impossible. We may regard these symptoms as expressive of a loss in inhibition, of a lack of self-control, hence a certain psychical influence cannot be denied. In some cases, perhaps even in the majority, the psychical state remains intact to the end. In others, there is a slight impairment of intellection and memory, a certain veiling and obtuseness of the psychical personality, a predominant childishness, occasionally an unmistakable euphoria, or, at least, the patient takes an optimistic view of his own condition; sometimes he is apathetic, or, as Oppenheim states, shows a mania for joking. Sometimes the psychic disturbances are severe; there may be a marked impairment of intelligence, even to dementia or confusion, excitement with hallucinations, etc. These are relatively rare and their explanation must be sought in the occurrence of multiple foci in the cortex of the cerebrum.

In isolated cases, the coincidence of multiple sclerosis with true progressive paralysis has been described, and the difficulty of this diagnosis will be revealed when we discuss the differential diagnosis of multiple sclerosis from progressive paralysis.

We have discussed the various symptoms of multiple sclerosis, and will now study their grouping in individual cases.

The symptom-complex described by Charcot is the typical form; those running another course are the *formes frustes*, but, as we have already mentioned, the latter are actually the most common, Charcot's complex being noted only in a minority of cases. I repeat, a combination of intention tremor and nystagmus with disturbances of speech, not necessarily always of scanning character, in which there are quite invariably or at least in the later stages, spastic paretic symptoms on the part of the lower extremity, must be regarded as the classical type. To this are frequently added affections of the optic nerve with disordered vision and transitory, but rarely permanent, paralysis of the muscles of the eye. Paresis, especially of the lower extremities, not rarely accompanied by ataxic symptoms, may be observed.

In other cases there is neither nystagmus nor intention tremor, and we find merely paralytic symptoms in the lower extremities combined with spastic phenomena, disturbances of the bladder, occasionally also sensory disturbances; in a word, the picture of transverse interruption of the spinal cord. In rare instances, the symptoms of spastic paresis combined with ataxic disturbances are limited for years to only one of the lower extremities. These have led to the differentiation of a purely spinal form of multiple sclerosis, which is especially favored by Leyden. In some of these cases symptoms of a cerebral nature for a certain time supervene or precede; this is particularly true of

changes in the optic nerve. Oppenheim and Frank have shown that multiple sclerosis may run its course under the picture of spastic spinal paralysis, a circumstance which will be more minutely discussed under spastic spinal paralysis, but in these cases atrophy of the optic nerve or typical temporal pallor of the papilla may point to the existence of multiple sclerosis.

It has been mentioned that in isolated cases atrophy may be added to these spastic symptoms in the upper and lower extremities, and the picture of amyotrophic lateral sclerosis thus be simulated. In my experience the so-called hemiplegic type of multiple sclerosis, hemiplegia of spinal origin with unilateral paralysis of the extremity, or of cerebral nature involving the facial nerve, and in which the upper and lower extremity upon the same side are involved and the symptoms may appear acutely, apoplectiform, are quite frequent. Occasionally we observe a crossed paralysis of the extremity and of the cranial nerve (facial, abducens), and here for a long time, perhaps even permanently, there may be neither intention tremor nor nystagmus, or at most but slight tremor of the paretic upper extremity. Subsequently disturbances of the bladder are added, or paresis of the other side, or bilateral hemi-paresis with predominant implication of the legs, or changes in the optic nerve may supervene, and finally, nystagmus, typical disturbances of speech, or dysarthria. Cases with bulbar paralytic disturbance, with or without other typical symptoms of multiple sclerosis, have been described.

We would digress too far were we to enumerate all the aspects which multiple sclerosis, particularly in the stage of onset, may assume. The foregoing should warn us again schematism. It should be borne in mind that the symptoms of multiple sclerosis may vary greatly, and in all doubtful cases its possibility must be considered.

COURSE

We will consider briefly the course of multiple sclerosis. As a rule, it is an extremely chronic disease, under some circumstances lasting for years, even ten to twenty; but we have several times stated that in the great majority of cases it is by no means invariably and steadily progressive, as are other chronic diseases, in the sense that the symptoms appear gradually and gradually become aggravated. On the contrary, acute periods of aggravation frequently alternate with periods of improvement. This variation in the intensity of the symptoms, these remissions, sometimes lasting for one or two years and longer, which may even simulate perfect recovery, are among the typical features of multiple sclerosis. An acute onset, apparently when in perfect health, severe symptoms followed by periods of aggravation and improvement, and a stage with relapses, are typical of multiple sclerosis. Under the consideration of the etiology we mentioned that external agencies, such as cold, trauma, infections and the puerperium, may aggravate the malady. In other cases the cause cannot be determined. A more profound paralysis finally ensues, and the patient may be bedridden for years.

In opposition to this exceedingly chronic course we have recently seen *subacute* cases which are, on the whole, more rapid; if the symptoms begin acutely, slight amelioration is rapidly succeeded by aggravation, so that the disease is fatal in a relatively short time, about two years.

Death is often the result of intercurrent diseases; more rarely, but particularly when the paralysis is severe and the patients are bedridden, death is due to bed-sores, phlegmons, cystitis, etc.

PATHOLOGY

We must now investigate the pathological anatomy in multiple sclerosis, and try to explain the symptoms by these pathological findings. The presence of numerous disseminated sclerotic foci in the brain and spinal cord is characteristic—the existence of a pure spinal form or a pure cerebral form must be considered doubtful—and they vary in magnitude from the size of a pin's head or lentil to that of a filbert. By the confluence of several foci large sclerotic plaques may develop, the form of which is rarely circular but usually irregular. In comparison with normal tissue the foci of multiple sclerosis are elevated so that in fresh sections they usually appear to be slightly elevated if recent, but contracted if of long standing. As a rule they show a gray or grayish red color. The number, seat and distribution of the foci in individual cases is subject to great variation, although even here there is a certain standard. In the optic nerve and chiasm the foci are frequently of varying extent but implication of the optic nerve is common; the cortex of the cerebrum is rarely attacked although, in opposition to former views, in many cases the presence of numerous foci has been demonstrated. Foci are occasionally noted also in the cortex of the cerebellum. The centrum semiovale, especially the area surrounding the lateral ventricles, is often the seat of extensive foci, while they are less common and less extensive in the trunk ganglia. The pons and medulla oblongata are the preferred seats, and here the foci may fill the greater part of the transverse section, covering extensive areas, and equally implicating the white and gray substance. In such cases the sections are often smaller and contracted, they are coarse in texture, and sometimes appear slightly transparent. In the spinal cord there are usually numerous foci, the form and extent of which vary greatly; some are the size of a pin-head or a millet-seed, some the size of a pea or even larger. Sometimes the entire transverse section or the greater part of it is implicated, in which case the circumference of the spinal cord is usually decreased in lesions of long standing. The longitudinal extent of the foci also varies. Large areas of the spinal cord may show diffuse changes. Occasionally there is a special implication of the cortical regions. Foci may be found in the anterior and posterior roots of the cord, while they are quite rare in the peripheral nerves; it has even been doubted whether they are ever found there.

While macroscopically and in preparations the foci are, as a rule, sharply defined, under minute microscopic investigation the normal tissue appears to be only slightly involved in the sclerotic focus.

I shall not minutely discuss the histologic examination, especially as the findings are not always the same. Some prominent points should be emphasized. A characteristic of ordinary foci is a marked increase of glia tissue, especially of the glia fibers, which appear as a dense film composed of parallel or interwoven fibrillæ. We usually find within this dense glia tissue—in suitable preparations also in longitudinal sections—naked axis cylinders or axis cylinders whose medullary sheath has been greatly diminished. The axis cyl-

inders themselves may appear unchanged; in other cases they reveal slight histologic alterations. Some authors maintain a regeneration of axis cylinders in multiple sclerosis, but these have probably been confounded with glia fibers.

This retention of the axis cylinder—described by Charcot and Schultze—is very characteristic of multiple sclerosis. Although we find naked axis cylinders in other processes, in these they form an exceptional occurrence, and not the rule as in multiple sclerosis. As the axis cylinder represents the most important constituent of the nerve fiber, this explains the circumstance that even extensive foci of multiple sclerosis, for example, in the pons, the spinal cord, etc., may show no secondary degenerations. Nevertheless, there are exceptions to this rule. For instance, we may maintain that with intense sclerotic foci a certain number of axis cylinders perish; this is most apparent in the cases running a subacute course, where the secondary degeneration may even be marked.

Let us first consider the ordinary picture of multiple sclerosis in which we know that even extensive foci usually show histologically less damage than might be expected. This is apparent also when we examine foci of the gray substance; here, upon minute examination, we are often astonished to find absolutely or comparatively intact ganglion cells in the foci of apparently severe sclerosis. It is true that some have suffered considerable damage. It must be remembered that, among the symptoms, we mentioned the relative rarity of muscular atrophy; this finds its anatomical correlation in the above circumstances.

The vessels in the foci of multiple sclerosis frequently show change. They are often dilated, their walls infiltrated or much thickened, the tissue around them somewhat indurated. But this finding in the vessels is by no means invariable, and the occurrence of such changed vessels in the center of foci cannot be considered characteristic of anatomical changes in multiple sclerosis, as is maintained by some authors.

The histologic picture of cases with subacute course differs somewhat from this description; here histologically we usually perceive the signs of acute degeneration, such as medullary granules and fatty granular cells; glia proliferation, as a rule, is but slight; swollen axis cylinders are numerous; in fact, the true sclerotic character of the process is but slightly if at all apparent. To this we must add that the nerve fibers are sometimes completely destroyed in circumscribed areas so that empty spaces develop, or spaces filled with fatty granular cells, and give to the tissue a so-called vesicular appearance. Almost invariably we find changes in the vessels, which are dilated, their walls often infiltrated, the adventitious sheaths filled with fatty granule cells. The same, or a similar histological picture, is commonly seen in the periphery of old foci, and forms the anatomical sign of subacute and acute aggravations of the process with which we are already familiar from the clinical picture. Corresponding to acute foci in those areas in which nerve fibers have to a large extent actually disappeared, we sometimes find secondary degeneration, either ascending or descending. The meninges, as a rule, remain uninvolved, and only occasionally have slight inflammatory processes been described therein.

According to the valuable researches of Uhthoff and Lübbers, focal anatomical changes also occur in the optic nerve. Histologically they resemble those found in multiple sclerosis; they consist of marked proliferation of the inter-

stitial tissue of the internal optic nerve sheath as well as of the finer elements; in some areas there are marked contraction, disappearance of the medullary sheaths, and the persistence of axis cylinders; sometimes, however, there is complete atrophy of the nerve fibers with severe vascular changes and, as a cause of dilatation and increase of the same, small celled infiltration, thickening of the walls, etc. The focal character of the changes, the persistence of the axis cylinders, and the consequent absence of extensive secondary degeneration, differentiates this process from the atrophy of the optic nerve which is common in tabes dorsalis.

In the *general pathology* of multiple sclerosis much might be said of the nature of the pathologic process, but since the views of eminent authorities are by no means unanimous and are still under warm discussion, we shall be brief. First is the question, What is the nature of the fundamental process and where does it arise, or, differently expressed, are we dealing with an *inflammatory or non-inflammatory* process, and is it of *parenchymatous* or *interstitial* form? Are the changes in the nerve fibers of primary, and the proliferation of the supporting tissue of secondary nature, or *vice versa?* In answer to the first question, multiple sclerosis has been included among the inflammatory processes; Leyden, for example, regards it as a form of chronic myelitis. Based upon clinical factors the acute appearance of some cases, the paroxysmal progress of others, the transition of various clinical pictures into multiple sclerosis, etc., transitions from acute disseminated myelitis or from disseminated encephalomyelitis, have been regarded as multiple sclerosis, and this has led us to adopt the view of its inflammatory genesis. Additional arguments have been found in the histologic picture of multiple sclerosis, especially in the not infrequent and severe vascular changes and infiltrations surrounding these. Others, however, have pointed out that the positive inflammatory processes of the central nervous system in many points resemble other pictures than those of multiple sclerosis, that vascular changes in themselves prove nothing, that anatomical chronic inflammatory processes of the central nervous system are exceedingly difficult to define. This question, however, involves a second, namely, Whence does the process arise? Charcot and many others assumed the primary condition to be an inflammatory process in the supporting structure which leads to proliferation, and that from this, therefore indirectly, the parenchymatous portion of the central nervous system is damaged. Opposed to this is the view of a primary damage to the nervous parenchyma, especially to the medullary sheath and by the consequent diminution of the resistance of the tissue, the glia is stimulated to proliferation. I cannot discuss the pros and cons of these views nor shall I consider, for example, the disturbance of the lymphatic circulation which, according to Borst, constitutes the primary pathologic factor.

Let us attempt, nevertheless, to gain some insight into the pathologic condition. Evidently the circulation plays a rôle in the production of the changes. Infectious toxic products, which we have learned to recognize as among the etiologic factors of multiple sclerosis, which to some extent may also be the consequence of exposure to cold, may damage the circulation after trauma—that trauma may damage the vessels and the circulation we know from other experiences—and are probably the cause of the pathologic changes. The nervous parenchyma, being the most sensitive tissue, first shows reac-

tion by the disappearance of the medullary sheath; irritated by this, but perhaps also because the same irritation which caused the degenerative processes in the parenchyma acts as a more powerful irritant to the supporting tissue, proliferation of the glia follows. Since, as we have seen, the circulation plays a causative rôle in these disturbances, it does not surprise us to find histological changes in this structure also. The varying pictures then depend upon the greater or less intensity of the deleterious factor, upon its especially injurious nature or its especially rapid entrance into the central nervous system, and enable us to understand why in one case there is merely slight parenchymatous damage with scarcely noticeable glia proliferation, and in another severe degeneration of the nerve fibers with histologic pictures which otherwise would be designated as inflammatory processes. Possibly there may be transitions of multiple sclerosis into inflammatory processes, for purely degenerative and inflammatory processes are exceedingly difficult to distinguish from each other—much more so than is commonly supposed. In the last mentioned form of multiple sclerosis there is apparently an extreme effect, whereas usually slight damage of the parenchyma is characteristic—hence the persistence of the axis cylinders and ganglion cells.

Let us briefly review the symptoms of multiple sclerosis from the standpoint of pathological anatomy. Much that we have learned to recognize loses its conspicuous character. We can readily understand that numerous foci may be present without generating special symptoms and that there may be a prolonged period of latency if the foci of multiple sclerosis leave intact the chief constituents of the functionating substance, the ganglion cells and nerve fibers. Hence the disproportion so often observed between the clinical and anatomical findings is to a certain degree only an apparent one. The acute exacerbations of the disease are explained by the more rapid advance of the anatomical process, due apparently to disturbances in the circulation of the blood and lymph which produce a greater derangement of function than actual degeneration. With the cessation of these initial phenomena and the persistence of but slight anatomical changes, the clinical symptoms also markedly decline. The remissions of the more chronic cases may be similarly explained, while the regeneration of axis cylinders which have perished need not be considered, this process having by no means been proven although some authors use it in explanation.

The frequency of spastic paretic symptoms, of disturbances of the bladder, etc., briefly, of symptoms which belong to the ordinary picture of a transverse interruption of the spinal cord, is accounted for by the remarkable changes which the extensive foci in the spinal cord undergo. Ataxic symptoms are not always referable to the same cause; they are seldom due to spinal foci which extend to the posterior columns; much more often they are produced by foci in the [illegible] which reach the [illegible], etc. The [illegible] cases, in particular, have a spinal seat, and when the [illegible] [illegible] [illegible] [illegible] disturbances in speech [illegible] [illegible] of the muscles of the eye, and disturbances in sight [illegible] due to foci situated in the [illegible] nerve and [illegible]

But I cannot [illegible] possibilities which may explain

the symptoms of multiple sclerosis. On account of the numerous aspects of the clinical and anatomical picture, in the majority of cases a satisfactory explanation can only be arrived at after a minute comparison of the anatomical findings with the clinical picture of the individual case. At the same time, it is extremely difficult to elaborate an anatomical foundation for the classical symptoms of multiple sclerosis, especially for nystagmus and the intention tremor. Every attempt at localization, i. e., the reference of these symptoms to definite localities, and their implication, is beset with difficulties, since the symptoms may be absent in spite of the undoubted affection of the localities. Is the damage in such cases not sufficiently severe? We do not know. Perhaps, as in nystagmus, we are dealing with a symptom which is susceptible to many explanations, and this enhances the perplexity. Probably there is also an indirect damage to the motor and coördinating tracts in question, and these symptoms are, to a certain extent, merely the expression of a general weakness of the systems which come under consideration.

DIFFERENTIAL DIAGNOSIS

We now come to one of the most important and interesting chapters, namely, the differential diagnosis of multiple sclerosis. Many cerebral and spinal processes must be considered in a differentio-diagnostic respect, as well as a number of so-called neuroses. In some cases all our diagnostic power must be brought to bear upon the case and yet it may be impossible to come to a decision. On the contrary, this must be based upon the subsequent course of the affection.

To be emphasized as especially characteristic of multiple sclerosis are the following: The coincidence of cerebral and spinal symptoms, occasionally a distribution which directly points to numerous foci in which there have been for years remissions and acute exacerbations. We will now consider in detail the differential diagnosis.

If we find the picture portrayed by Charcot: nystagmus, intention tremor, disturbances of speech, spastic paretic symptoms in the extremities, perhaps combined also with ataxia, disturbances of the bladder, characteristic affections of the optic nerve and a typical course, the diagnosis will generally not be difficult. But even here we must be cautious. Westphal, subsequently Strümpell, v. Frankl-Hochwart and others have shown that in a small number of cases there may be a condition analogous to Charcot's picture, while the autopsy reveals no characteristic changes nor any anatomical alterations which indicate multiple sclerosis. These cases, described as *pseudo-sclerosis*, as a rule begin early, about the tenth year or a little later; heredity appears to have some influence, and even a family predisposition has been observed. Strümpell believes that hereditary syphilis may perhaps play a rôle. There are invariably disturbances of motility, less frequently paralysis, at most only in the latest stages, while spastic phenomena, even marked contractures and spastic gait, are common; a remarkable slowness in movement is also sometimes noted. The tendon reflexes are exaggerated. Usually there is tremor, particularly of the upper extremities, which does not, however, closely resemble intention tremor but is slower, much coarser, and may terminate in profound jactitation. Disturbances of speech are prone to appear, above all

slowness of speech, and also dysarthric affections. On the other hand, nystagmus has rarely been observed; there are neither changes in the optic nerve nor sensory disturbances. Pain or slight paresthesia is occasionally mentioned. Bladder affections are rare. A common, perhaps an invariable symptom, is psychical disturbance, which sometimes increases to actual dementia. Spasmodic laughing and crying have also been observed. Attacks of syncope and apoplexy are relatively frequent, and sometimes leave paralysis which lasts for several days; epileptiform attacks have several times been noted. As a rule, these cases of pseudo-sclerosis are of long duration, ten years or, under some circumstances, much longer. Remissions may occur.

Thus a clinical picture is presented which bears a close resemblance to multiple sclerosis, but certain differences may be discerned: for instance, the paretic symptoms are less developed, there is usually no actual paralysis, the tremor is not absolutely typical, bladder disturbances are rare, there is no change in the optic nerve or paralysis of other cerebral nerves; on the other hand, psychical disturbances, etc., are more prominent. Moreover, we must admit that if the cases described as pseudo-sclerosis be compared we see no uniform picture; some of these force upon us the suspicion voiced by Strümpell that we are dealing with juvenile progressive paralysis. We must not forget that some of these cases originated at a time when the clinical picture of juvenile paralysis was less clearly portrayed than it is to-day.

I have stated that the anatomical findings in these cases apparently indicate normal conditions, and histological investigation is of little importance. But it would be a great mistake if, for this reason, we were to consider these cases hysterical, as French authors have done. Strümpell, Gerhardt, Fränkl-Hochwart are probably correct in assuming that these cases [illegible]

[illegible]

which lead to actual dementia. The course is protracted throughout years, usually, however, without remissions, and steadily progressive.

Here there is an unmistakable resemblance to the symptom-complex of multiple sclerosis, and this, according to Gerhardt, is particularly marked in children. In fact, the differential diagnosis is extremely difficult. The absence of nystagmus, the prominent psychical disturbances, the severe disorders of speech, etc., may in some cases, but not in all, form a contrast to multiple sclerosis.

In so-called pseudo-sclerosis and diffuse sclerosis we are dealing with rare affections which are therefore perhaps of less practical importance. We must now, however, differentiate multiple sclerosis from a number of common diseases. Starting from the fully developed picture of multiple sclerosis, we first consider *progressive paralysis.* In progressive paralysis there is a conspicuous tremor which does not always resemble intention tremor, very commonly there are also spastic paretic and ataxic symptoms, particularly in combination with tabes. Disturbances of the bladder, alterations in speech, changes in the optic nerve, paralysis of the muscles of the eye, apoplectic attacks with consequent paralytic phenomena, etc., are noted. On the other hand, dementia is usually and early prominent in progressive paralysis and increases to an extreme degree; as a rule, even in the early stages, other psychical disturbances are added (maniacal state or depression), while psychical disturbances are relatively rare in multiple sclerosis, and, if at all, usually occur late. The disturbances of speech in progressive paralysis are not of the same character as those occurring in multiple sclerosis. Optic atrophy, if present, resembles the tabetic form. There is reflex pupillary rigidity, a symptom not occurring in multiple sclerosis, but exceedingly common in progressive paralysis. The course of progressive paralysis is more rapid than that of multiple sclerosis. Frequently there is a history of syphilis so that, as a rule, a positive differential diagnosis is possible.

In childhood cases of apparent multiple sclerosis must be distinguished from the infinitely more numerous cases of *cerebral diplegia.* In the latter disease the symptoms are often congenital, or they appear very early. Epileptic attacks are frequent at the onset; as a rule, we find no nystagmus, no disturbances of speech, certainly no typical ones, no changes in the optic nerve, nor affections of the bladder, etc. The symptoms are usually stationary, or regressive rather than progressive.

Acute disseminated encephalitis or *encephalo-myelitis,* which must be compared with sub-acute cases of multiple sclerosis, develops acutely, usually with febrile symptoms, and subsequently shows remissions which, contrary to the condition in multiple sclerosis, are not, as a rule, succeeded by exacerbations. Nystagmus and affections of the optic nerve, certainly atrophy, are absent. Transitional stages in the clinical picture have already been mentioned. As multiple sclerosis may be apoplectiform in type, and leave more or less permanent hemiplegia, we must also exclude the ordinary causes of such condition, *softening and hemorrhage.* The latter, as a rule, occur in elderly persons or in the young who suffer from a disease of the heart, from nephritis or syphilis. As a rule, after the first symptoms have disappeared, the disease is stationary, except when renewed attacks occur. Tremor is usually on the hemi-paretic side. There is neither nystagmus, bladder disturbance, nor

change in the optic nerve, etc. Gerhardt calls attention to the fact that, under some circumstances, multiple softening may produce a clinical picture resembling that of multiple sclerosis in which advanced age, demonstrable arteriosclerosis or nephritis, a moderate degree of nystagmus, intention tremor, and marked prominence of bulbar symptoms (?) favor softening.

Westphal and others have described cases in which multiple sclerosis was simulated by *brain tumors*; tumors of the cerebellum or the posterior cranial fossa may, under some circumstances, cause spastic paretic and ataxic symptoms, as well as apoplectiform attacks, affections of the sight, changes in the optic nerve, nystagmus, and disturbances of speech. As a rule, however, the differential diagnosis is not difficult. But it must be borne in mind that there are cases, such as Bruns and Stölting described, in which multiple sclerosis appeared acutely with severe headache, vomiting, loss of consciousness, optic neuritis, and even choked disc, and we can readily understand that in this stage no differential diagnosis can be made. Only the subsequent course, the regression of the threatening symptoms, and the later appearance of other typical features, will disclose the nature of the case.

The differential diagnosis of multiple sclerosis from cerebrospinal *syphilis* is of great importance particularly in regard to treatment. There may be a clinical similarity because in both maladies numerous disseminated foci may form in the brain and spinal cord. Among the common symptoms relating to the brain are disturbances in the optic nerve, apoplectiform attacks with ensuing paralysis, occasional disturbances of speech, spastic paretic phenomena in the extremities, derangements of the bladder and sensation. On the other hand, in multiple sclerosis pains in the back and vertebral column are usually absent, and in syphilis there is neither nystagmus, intention tremor nor scanning speech. Spontaneous remissions occur in syphilis, but these are rarely of such long duration as in the case of multiple sclerosis. In doubtful cases the results of an antisyphilitic treatment will frequently, although not invariably, decide the case—for syphilis of the central nervous system may prove refractory under this form of treatment.

Among the so-called neuroses we must first consider *paralysis agitans*; a certain similarity is at once evident; Charcot was the first to differentiate these affections positively by their clinical course. As a rule, their differences are greater than their points of resemblance, for paralysis agitans usually occurs in elderly persons, multiple sclerosis in the more youthful. The tremor of multiple sclerosis is intentional, that of paralysis agitans continuous. In the latter affection there are peculiar states of tension and contraction, but no true paretic and ataxic symptoms, no marked disturbances of speech and of the bladder, no intense sensory disturbances, above all, no changes in the optic nerve, no nystagmus, and no paralysis of the muscles of the eye. The course is also different: In paralysis agitans it is steadily progressive, in multiple sclerosis there are acute exacerbations and succeeding remissions. Apoplectic insults with succeeding paralysis are common in multiple sclerosis. In paralysis agitans, as a rule, the numerous attacks of vertigo and syncope leave no well marked paralysis. Of course, these distinguishing features may not always be so well developed. Tremor may be absent in multiple sclerosis, and in the early stages of paralysis agitans intention may distinctly increase the tremor. Paralysis agitans may occur in relatively young persons, multiple

sclerosis in the aged. Hence, in imperfectly developed cases of multiple sclerosis there are certain, although by no means insurmountable, obstacles to the diagnosis.

The differential diagnosis from *hysteria* may be very perplexing, and here grave errors have occasionally been made. Oppenheim warns us that beginning multiple sclerosis may readily be mistaken for hysteria. *Vice versa,* hysteria may, under some circumstances, produce a picture resembling multiple sclerosis. In such cases there is tremor (often resembling intention tremor), spasm and paresis—of course, I refer here only to the so-called *pseudo-spastic tremor* after trauma—increase of tendon reflexes, ataxia or pseudo-ataxia with pseudo-Romberg symptoms, disturbances of sight and of sensation, diplopia, and even bladder disturbances. As a rule, a differentiation can be made; sensory disturbances are usually much more conspicuous in hysteria than in multiple sclerosis, often are unilateral, and the sensory functions are implicated. In hysterical disturbances of sight the concentric limitation of the field of vision is characteristic (in multiple sclerosis central scotomata or irregular contractions of the field of vision are predominant); diplopia is monocular, bladder disturbances are transitory, etc.; above all, in hysteria the demonstrable ophthalmic changes of the optic nerve as well as actual paralysis of the muscles of the eye, the nystagmus, the severe disturbances of speech, the bulbar symptoms and spastic phenomena are, as a rule, of different nature. Babinski's sign is usually absent. The appearance of typical, hysterical, spasmodic attacks or the peculiar psychical condition of many hysterical patients may clear up the case; but the possibility must always be borne in mind that multiple sclerosis may be combined with hysteria, in which case the difficulty of diagnosis is multiplied.

The *formes frustes* of multiple sclerosis which have been described may for other reasons give rise to differentio-diagnostic problems. Without discussing all the possibilities, a few important points may be emphasized. We have seen that multiple sclerosis may produce symptoms which are usually attributed to a transverse interruption of the spinal cord; therefore, spastic paresis of the lower extremities with sensory disturbances, affections of the bladder, etc. In such instances we must consider all of the affections which lead to transverse interruptions of the spinal cord, namely, caries of the vertebral column with compression of the spinal cord, syphilis of the spinal cord, chronic myelitis, tumors of the spinal cord, even syringomyelia. Minute examination to determine whether other symptoms of multiple sclerosis are present—for example, those of cerebral nature such as nystagmus, above all, an implication of the optic nerve or phenomena in the upper extremities (intention tremor), or ataxia—will furnish data for the differential diagnosis. The same is true when the symptoms of spastic spinal paralysis resemble the clinical picture under discussion. Aside from the fact that the latter affection is rare, the diagnosis, as was shown by Oppenheim, Frank and others, can only be made by exclusion and the ophthalmoscopic finding, and sometimes the subsequent course of the disease will alone permit a decision. The same is true when multiple sclerosis appears under the guise of amyotrophic lateral sclerosis. In spastic ataxic paresis, some forms of combined system disease will be considered; the presence or appearance of severe cere-

bral symptoms will be decisive. Other spinal processes, such as tabes, Friedreich's disease, etc., need only be mentioned. They may, under some circumstances, be considered, but, as a rule, they are readily excluded.

PROGNOSIS

Little remains to be said of the prognosis. Multiple sclerosis is a chronic, incurable malady. In isolated cases cures have been reported, for example, by Charcot and Oppenheim, but without doubting the reports of these experts, such cures are certainly rare. A cessation of the process, remarkable remissions lasting even for years certainly occur, and this not even infrequently, but, finally, new phenomena appear.

TREATMENT

In contrast with our perhaps too lengthy discussion of the pathology and diagnosis of multiple sclerosis, in the treatment of the disease we may be brief. So far as the disappearance of the symptoms or the complete arrest of the process is concerned, there is unfortunately no actual treatment. Nor can we speak, except to a very limited degree, of the prophylaxis of multiple sclerosis, especially in those cases in which it follows injuries or these favor its advance, for example, exposure to cold, trauma, excessive exertion, the puerperal state, intoxications, etc., all of which are preventable.

Patients with severe paralytic symptoms, particularly acute exacerbations, should have prolonged rest in bed. In the latter case, mild antiphlogistic remedies (ice coils to the back, and preparations of the salicylates, etc., internally) may be tried. Among other remedies specially indicated in the early stages are mild hydriatic procedures, for example, lukewarm baths (32° C.), perhaps with the addition of salt, or artificial carbonic acid baths; in the initial stage half baths (at a temperature of 31° C. and reduced to 29° or 28° C.) or moist packs. Indifferent baths, or salt and carbonic acid baths, are not injurious, but the temperature should never exceed 32° to 34° C. In addition to these electricity, galvanization of the spinal cord and the extremities, is to be employed and light massage may be carefully tried. In stationary conditions in which ataxic phenomena are predominant, exercises such as Frenkel employed in tabes may be practised. As we are dealing with a chronic affection lasting for years, these curative measures are not to be used continuously, but should be employed from time to time according to the urgency and intensity of the symptoms, at one time this remedy, at another that, or their combination and then cessation. Some drugs may be given internally for a few weeks, for example, sodium iodid in moderate doses (1.5–2.0), which is given in all chronic spinal processes with appreciable or positive results; silver nitrate and ergotin, formerly much in vogue for the treatment of spinal processes, may be used but it must be with care; their effect is more than doubtful. Roborants may be tried, preparations of arsenic and quinin, nux vomica, etc., which sometimes favorably influence the general condition and, perhaps indirectly, the spinal process. In all of these methods of treatment we must bear in mind the very frequent spontaneous remissions. If one of these occurs during the progress of such treatment, it will naturally

appear that the therapy is effective, but a new advance of the process will show the treatment to have been completely without result. In the terminal stages of the disease, when the patients are permanently bedridden and there is paralysis of the bladder, there is danger of cystitis and bed-sores, and the most careful nursing is necessary as well as all the precautions with which we are familiar in the treatment of other severe cerebral or spinal processes.

SYRINGOMYELIA

BY FR. SCHULTZE, BONN

A patient, a young man, presented upon inspection a peculiar anomaly, ***marked emaciation of the muscles of the hand.*** All of the interossei muscles of the left hand and the hypothenar surface were flattened and there was also thenar atrophy. The same changes had taken place in the right hand, but to a less extent.

There was a *claw* position of the fingers, this being more marked in the left than in the right hand. The terminal phalanges were abnormal, and were laterally displaced toward the mid-phalanges; the nails were somewhat defective. In various areas of the skin were elevated cicatrices and indurations left by previous injuries.

While the color of the hands was, in the main, normal or only slightly bluish (livid), the index finger of the right hand, in spite of the warmth of the lecture room, was ghastly pale.

Upon examining the arms of the patient, a decrease in the musculature of the forearm, upper arm, and shoulders was noted.

On testing the muscular power of the hand and fingers a more decided loss was observed. The fingers could not be fully extended, although some of them more so than others. Adduction and abduction were abnormal; perfect apposition of the thumb and little finger was impossible and more marked in the left than in the right hand.

In this case a condition of muscle atrophy existed. According to the report of the patient, this had come on gradually, first attacking the left and then the right hand, and then involving the forearm. We have to consider that form of ***progressive muscular wasting*** which we designate *progressive muscular atrophy.*

This euphonious title sounds diagnostic, yet it is not. Just as little as jaundice, dropsy, or fever prefigure a satisfactory diagnosis, so little does the designation "progressive muscular atrophy" define the condition.

In each individual case we must investigate the cause of the disease. It may be a *primary* affection of only the muscular fibers, a so-called primary myopathy, a primary muscular dystrophy, or a *secondary* change.

These disturbances might at first be assumed to have been the result of chronic arthritic changes; but in such conditions certain muscles pertaining to the joint affected are invariably diseased; for example, the muscles of the hands and fingers after disease of the knuckles. Secondly, they might have been the consequences of primary disease of the *nervous apparatus.* In this case we must consider the peripheral nervous system and the medulla spinalis including the root bundle. After changes in the brain this double atrophy of the muscles of the hand and arm is not observed.

Under all circumstances, therefore, the entire nervous system must be investigated.

In regard to *sensation,* we note that tactile sensation in the hands was by no means obliterated, but was *decreased* to such an extent that the touch of dull and sharp pointed instruments was no longer distinguished. This was most marked in the course of the left ulnar nerve.

Much more conspicuous is the fact that in the hands *sensibility to pain* was nearly or wholly lost, as well as in the arms and the upper part of the trunk, and that it was markedly *decreased* in the neck.

This was, as a rule, true also of the *temperature sense, especially in regard to heat,* which was almost absent in the skin of the hands, and decreased in the upper extremity,

in the trunk, and in the neck. Behind the ears and near the angle of the jaw cold was frequently felt as heat, and lower down thermic variations could not be differentiated.

On palpation of the peripheral nerves which were susceptible to this method, and the brachial plexus, no thickening nor tumor could be discovered nor was there any abnormal sensation of pressure.

The *vertebral column* was found to deviate markedly to the right. In the main there were no recognizable sensations upon inspection or palpation. The spinous processes and their surroundings showed no abnormal sensitiveness to pressure.

Fig. 174.—A Man, Aged 25, Suffering from Syringomyelia.

Examination of the *functions of the brain* revealed no marked change. The sensory, the central, and the peripheral apparatus belonging to the individual cranial nerves functioned normally.

The palpebral fissures were equally wide, the eyeball neither protruding nor too much sunken. Both pupils were somewhat contracted, the right less so than the left, but in refraction, accommodation and convergence they were normal.

Except a mild, not distinctly pathologic nystagmus when the glance was directed to the right, no abnormal change was discovered in the *eye-muscles*.

The motor portion of the trigeminal and facial nerves was intact.

Sensation in the fifth nerve, to delicate tests as well as upon mere contact, was not everywhere normal. Dull and pointed objects were commonly confounded; sharpness

was distinctly appreciated only in the upper part of the forehead, being less acute in the lower trifacial branches.

Deep pricking with a needle was described as painful, but not slighter pricks. Pricking through a fold of the skin below the chin was not felt at all.

If a test-tube containing *boiling* water was held against the skin, pain was at once experienced above the orbital border, but not below this region; the patient felt it to be warm, and reported that the sensation of "heat" decreased in distinctness from above downward.

Fig. 175.—Syringomyelia (A man, aged 25).

Heat upon the mucous membrane of the lips, the mouth, and the tongue, also on the skin of the nose and on the eyelids was distinctly felt. This area of distinct sensations was bounded by a line drawn from the external commissure of the eyelid to the alæ of the nose. Upon the *hairy scalp* cold and heat were distinctly appreciated.

In the distribution of the hypoglossal nerve no disturbance could be demonstrated, which is also true in general of the course of the pneumogastric. The *right vocal cord* could be adducted, but no longer abducted, hence there was a right-sided paralysis of the recurrent laryngeal, especially in the region of the posterior cricoarytenoid muscle. No abnormality was detected in the course of the spinal accessory nerve.

Besides the changes in sensation mentioned, examination of the *spinal nerves* showed that the muscular power of the lower arm as well as of the upper arm, the shoulders, the trunk, and lower extremities was almost normal. *Electric* examination of the muscles of the hand revealed complete DeR in both primary external interossei. In the remaining interossei, and in the hypothenar eminences, there was a partial DeR.

The *reflexes* were as follows: While the tendon and periosteal reflexes were absent in the arm and hand, they were active in the lower extremities but not pathologically increased.

There was neither foot nor patellar clonus; on tapping the tibia there were no contractions of the quadriceps. The plantar reflexes were normal; the *reflexes of the abdominal walls* were produced only by stroking the skin over the lower part of the abdomen.

There were no disturbances of coördination, tremor, or other motor irritative phenomena. The gait was unchanged. The bladder, the rectum, and the genital apparatus acted normally.

In addition, a careful testing of *sensation* in the trunk and extremities showed that the anterior line of thermo-anesthesia extended to the seventh rib in the mammillary line, while sensibility to heat increased to the navel, that is, hot water was felt as warm.

Severe irritative pain from firm pinching was not felt above the eighth rib, but was evoked lower down. Still lower down sharpness and dulness could not be distinguished.

There was complete analgesia in *the back* from the middle of the neck down to the eleventh rib on the right, and to the tenth rib on the left side.

Examination of the other organs of the body elicited no recognizable anomaly. With the exception of the finger-joints which were stiff, the joints as well as the bones, tendons, and ligaments were normal.

This patient was of medium size, of strong bony frame, the muscles and fatty tissue showing a moderate degree of nutrition. The skin and mucous membranes were pale.

There were no cicatrices upon the penis; a few cubital and axillary glands were somewhat larger than normal.

The *history* showed that the parents of the patient died early, of unknown diseases, and that two of his brothers were healthy. Whether his birth was normal, he did not know. As a boy he was always well. In 1892, during an adventurous life in Dahomey with the French mercenaries, he suffered severely from fever with jaundice. In 1894, in Tongking, he had tearing pains in both arms with distinct *muscular twitching,* so that not infrequently his gun fell out of his hand. This peculiar twitching usually came on after he had been resting for some time. It lasted from two to three minutes, often awakened him from sleep, and was not accompanied by loss of consciousness. *Two or three months later* deformity of the fingers gradually appeared, and he noted that he *could touch hot substances* without experiencing inconvenience, but that blisters followed. He had to resign from the service, and returned to Europe in October, 1895.

The deformity of the fingers steadily increased; and for nine months he was *frequently* affected by *whitlow.*

The legs remained normal; the patient could walk for hours without fatigue. No other disturbances than those mentioned were ever observed.

In this patient we were not dealing with a progressive muscular atrophy alone, but there were simultaneously *peculiar sensory* and *trophic disturbances,* so that the *nervous system* was unquestionably affected. The stiffness in the joints was only to be regarded as an accompanying symptom.

What was the nature of this nervous disturbance, and where its seat?

Any one familiar with the symptomatology and etiology of *multiple neuritis* or *polyneuritis,* which this case resembles, would say that in this patient none of its known causes was operative. Neither alcoholism, lead poisoning, nor the effects of arsenic could be demonstrated. And it would be extremely arbitrary to assume refrigeration as a cause, or a rheumatic origin in the same sense as though an acute or chronic arthritis or muscular rheumatism had preceded or accompanied the disease. There had been no trauma, no syphilitic infection could be determined, nor had their been excessive use of the arms.

Above all, in ordinary polyneuritis, no matter of what nature, the *localization of the paralysis* is different. In most cases the nerves of the lower extremities are simultaneously implicated, or even earlier and more markedly; in lead paralysis the region of the radial nerve suffers most, but was here exempt. Notwithstanding the sensory disturbance in our case, there was no sensitiveness to pressure upon the peripheral nerve trunks, while in polyneuritis and other forms of neuritis there is none of the peculiar sensory disturbance which was here present. In polyneuritis the sensations conveyed by the individual cutaneous nerves are distributed much more uniformly. Finally, the limitation of the analgesic area and the zone of temperature sensation of the skin in peripheral neuritis is very different from that in the case described.

Only one form of neuritis could be thought of, the variety occurring in leprosy, i. e., in that form of leprosy designated by the term *nervous leprosy,* because it particularly implicates the nerves and not the skin.

As our patient had lived in countries where this disease occurs he might easily have acquired it.

Cutaneous leprosy was out of the question. This affection, as a rule, is first localized in the face, which in this patient was entirely devoid of the characteristic nodules and abnormal pigmentation. The eyebrows were perfect, and there were none of those white and dark areas upon the skin of the trunk and extremities which are so typical of leprosy, as well as circumscribed anesthesia in certain areas.

Perhaps the state of the hands resembled leprosy, since in leprous neuritis there is an atrophy of the muscles, and trophic disturbances and vasomotor changes appear, even the dissociated sensory paralyses which we detected in the patient under consideration and which were formerly called partial sensory paralyses may occur.

But there was no swelling of individual portions of the peripheral nerves such as occurs in leprosy, particularly of the ulnar nerve in the vicinity of the olecranon which may be felt in this area. Sometimes, I must admit, the diagnosis of such a thickening is very perplexing; it may, perhaps, be the result of chronic inflammation and ulceration in the hands and forearms without leprosy. In this case, however, no such change was present.

Concerning other symptoms it must be stated that *unilateral paralysis of the recurrent laryngeal nerve* without implication of the skin of the face and of the other nerves of the head does not occur in leprosy except when other causes accidentally produce paralysis of the larynx. Moreover, contractures of the muscles of the arm such as occurred in our patient is rarely observed in leprosy. Finally, the bacillus of leprosy was not found in the nasal mucus, and, according to recent investigations, this microörganism is frequently, if not invariably, present in leprosy.

Taken all in all, no diagnosis of leprosy could be made in this case, and as there were no reasons for assuming a bilateral plexus paralysis, the question arose, What form of spinal disease is here present?

The peripheral nerve fibers and nerve bundles extending to the spinal cord pass through the intervertebral spaces and the membranes of the spinal cord before they reach the medulla spinalis. Therefore, diseases of the cervical vertebræ and the ligamentous apparatus belonging thereto, as well as diseases of the membranes, may exert pressure upon the passing roots or cause inflammation therein.

In this patient there were none of the symptoms which appear in chronic and progressive diseases of this nature.

Inflammation and neoplasms of the vertebræ and the membranes of the spinal cord, even in the beginning, usually produce local and radiating pain, and stiffness of the *neck*, with difficulty in moving it. Although the pain is not always severe it is invariably present in those cases in which anesthesia subsequently appears. In our patient, there was no pain, nor was there rigidity and inhibition of motion in the cervical vertebral column, therefore we were forced to consider whether the *white substance* of the spinal cord was diseased, and whether *chronic myelitis* of the cervical enlargement might be present, since, according to experience, in these affections the white substance usually is implicated.

Since, however, these are accompanied by changes in the lower extremities,

particularly *spastic paresis as well as changes in the function of the bladder,* and as these were absent in our patient, no alternative was left us but to regard the *central gray substance* as the seat of the disease. Isolated chronic inflammation never occurs in the anterior and posterior portions of this structure without the formation of cavities—that is, it has never yet been demonstrated; and since even tumors rarely form in this substance without producing cavities, we were certainly dealing with a chronic, progressive, and extensive cavity formation with destructive processes; briefly, with *progressive syringomyelia,* or with a *central tumor formation* which did not produce pressure upon the white substance from within.

While refraining from an immediate differentiation of these two pathologic conditions, I wish to state that *syringomyelia* is by no means a rare affection of the spinal cord.

We must now discuss the nature of this peculiar spinal change, and we will most readily understand the clinical symptoms if we begin with the pathological anatomy, and then utilize our knowledge of physiology in considering the functions of the spinal cord.

The name "*syringomyelia*" (i. e., a flute-like spinal cord, from ἡ σύριγξ, the flute) was applied to this affection by a French physician, Ollivier, who wrote a monograph upon diseases of the spinal cord in the early part of the preceding century. In fact we not rarely find throughout the entire extent of the medulla spinalis a space in which a penholder or lead-pencil might easily be introduced, and which contains a watery fluid.

Such a curious and conspicuous change was of course observed long before the time of Ollivier, apparently first by Etienne at the end of the sixteenth century, later by Morgagni, Portal, Andral, Nonat and others. But prior to about the seventh decade of the last century, these cases were regarded as mere curiosities.

In explaining its occurrence, writers of about the year 1800 assumed the condition to be due to an abnormal development of the central canal which in children, as is well known, extends the entire length of the spinal cord from above downward, while in later life it is partially or wholly occluded or even obliterated.

It is quite conceivable that this canal might, under some circumstances, remain open; if, for instance, abnormal dilatation of the cerebral ventricles and the consequent pressure of the fluid they contain dilates the central canal in the early stages of its development. This canal communicates directly with the fourth ventricle.

Undoubtedly there is such a hydromyelia, but it is usually a stationary condition which does not produce progressive clinical symptoms. It is uncertain whether or not, from this abnormal condition, increasingly destructive processes may at the same time develop and, like *ordinary progressive syringomyelia,* destroy the anterior and posterior gray substance and also the white.

If we examine transverse sections of a syringomyelitic spinal cord we at once observe that, particularly in the cervical portion, there is a great but varying loss of substance, chiefly of the gray. Throughout the whole of the posterior horns and the commissures there are empty spaces which separate the entire anterior and lateral portions of the spinal cord substance from the posterior columns. Minute microscopic investigation sometimes reveals around

these spaces a thin layer of glia fibers, in other cases simultaneously such a great accumulation of glia cells are produced that they cause displacement phenomena (so-called *gliosis* or *primary gliosis*) or an extensive tumor formation (*gliomatosis, gliomata*).

The central canal is often situated *in front* of the cavities and spaces although commonly filled with those ependymal cells which are quite frequently found in the normal cord of adults.

Therefore, in such cases we can exclude simple dilatation of a *normally* situated central canal in the walls and surroundings of which destructive and proliferative processes have been generated, and other morbid states must be considered.

In the first place minute anatomical investigations have demonstrated that several central canals may develop and be situated behind each other, and that consequently an extensive diverticulum of the central canal may be formed similar to that observed in the esophagus.

A number of cases make it seem extremely likely that syringomyelitic processes may readily develop from such diverticula and auxiliary cavities. In

FIG. 176.—SYRINGOMYELIA OF THE CERVICAL ENLARGEMENT. The white substance is stained black by Weigert's stain. The entire gray substance is changed, destroyed, or permeated with cavities. Even in the posterior horn, only a part of its contour is normally retained.

other instances no such anomalies can be found, and we must assume that syringomyelia may develop also in a normal spinal cord. Nevertheless, we must finally inquire how these anomalies of development arise, and whether in the growing cord, at birth or later, other disturbances may not naturally develop which finally lead to progressive syringomyelia. The cavities and spaces are not necessarily the *primary* condition but may arise *secondarily*. Thus it is possible that tumors of a gliomatous nature may develop from the ependyma or from other parts of the glia substance and gradually break up in the center, thus causing these secondary cavities sooner or later to com-

municate with the normal or abnormally situated central canal. Moreover, it has been proven that chronic *pachymeningitic* and *leptomeningitic processes* may here and there occur, and be combined with the formation of an elongated central cavity. We know, too, that *hemorrhages into the spinal cord*, such as appear after *trauma*, are most often found in those areas in which the cavities

FIG. 177.—TRANSVERSE SECTION THROUGH THE DORSAL PORTION OF THE SPINAL CORD. Tumor-like accumulations of glia around a central cavity (gliosis).

FIG. 178.—TRANSVERSE SECTION THROUGH THE DORSAL PORTION OF THE SPINAL CORD (from the same case as Fig. 177). There is no central cavity, but only rarefaction of the greatly proliferated central glia masses.

of syringomyelia are localized. The interesting fact has been observed that in infants whose bodies had been subjected to marked pressure and compression because of hard labor circumscribed hemorrhages were found in the spinal cord and the medulla oblongata in the same areas in which cavities form.

It is noteworthy, too, that the blood-vessels in the surroundings of the cavities are often greatly thickened, and obliteration as well as thrombosis may appear.

We may, therefore, conclude that in some cases elongated tumors first appear followed by central destruction, that in others *hemorrhages* first occur and subsequently, as in ordinary cerebral hemorrhage, cavities and cysts form. We must still explain how progressive syringomyelia develops from these cavities. Here we encounter the same difficulty as in the case of hydromyelia and the other anomalies of development we have named.

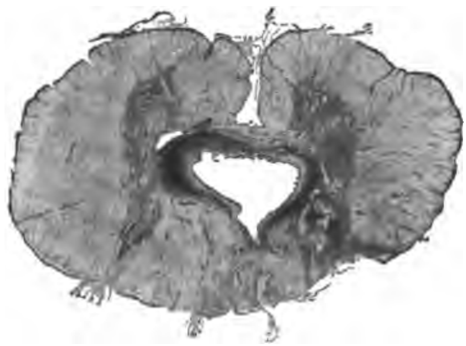

FIG. 179.—TRANSVERSE SECTION OF A DORSAL PORTION OF THE LUMBAR CORD (from the same case as Fig. 177). A larger central cavity with a marked glia ring.

In other cases, acute or chronic inflammatory processes may be combined with necrosis and loss of substance so that a "*myélite cavitaire*," as some French authors designate it, develops. These cavities are not found in the usual acute and chronic forms of myelitis even when progressive processes are present, as in the case of multiple sclerosis. Except in the pachymeningitic

processes just mentioned, elongated cavities do not form in syphilitic affections, although I observed a case of this kind in which a moderately extensive syringomyelia was present as well as chronic myelitic changes in the lumbar cord without pachymeningitis.

If in such cases we assume thrombotic changes or vascular obliteration from a non-inflammatory cause, the increased length of the canals and spaces is difficult to explain, since the arteries and veins which penetrate the gray substance have only a short course and we must therefore assume that numerous adjacent areas, without exempting isolated diseased ones, have been successively implicated. Acute poliomyelitis is often widely distributed so that here also the involvement of many areas takes place; but, notwithstanding great vascular changes no cavities are formed.

When we state, in addition, that in certain cases of brain tumor or compression of the spinal cord pressure from the central canal was regarded as the cause of cavity formation, it is evident that every effort has been made to explain the development of these processes.

It is, however, apparent from the very complete and accurate description of these conditions that we are not at present justified in assuming for *syringomyelia a uniform mode of development.* Congenital anomalies of development, either disturbances occurring at birth or in the course of extra-uterine life, here play a rôle. Tumors, hemorrhage, and necrosis, inflammatory and degenerative processes, as well as peculiar, localized, vascular changes, must be considered.

All of these have the common property of localization in the *central* portion of the spinal cord and a tendency to progression.

The latter condition is most difficult to understand. In the case of tumors of great or less extent, therefore in gliomatosis, in formation of glioma, or in primary central gliosis, advancing growth is at this time as impossible to explain as that of tumors in general. If it be due to inflammatory or degenerative processes, the understanding of its tendency to progress as well as many other advancing pathologic conditions in the central nervous system and in other organs is lacking.

It is remarkable that these by no means invariably accompany arteriosclerotic processes in other organs, or even in the brain. Therefore, they are not typical of a disease of advancing age, as is arteriosclerosis, but they usually develop in youth; hence early disturbances in the formation of the spinal cord, and injuries occurring prior to this, must also be considered.

The conclusion is obvious that in many cases in which the first signs of syringomyelia appear during the course of infectious disease, for example, enteric fever, the microörganisms or toxins which are generated in such affections accumulate in abnormal diverticula of the central canal, and thence set up pathologic processes.

Whether this be true or not, the condition and localization of the syringomyelitic changes which have been described may be deduced from other well known physiologic facts and many of the symptoms which necessarily arise in this affection.

Before attempting this, I must state that, besides affecting the gray substance and the posterior columns, the proliferative and destructive processes going on around the spaces and cavities may also implicate the lateral columns,

for they are either exposed to a marked lateral pressure from the proliferated glia masses or are directly implicated in the chronic degeneration.

Frequently the entire spinal cord is permeated by cavities and spaces; sometimes, however, the cervical and dorsal portions alone may be implicated, while syringomyelia limited solely to the lumbar portion is very rare. Usually the segments of the cervical enlargement, particularly the lower ones, are most markedly affected.

It is significant that not rarely the medulla oblongata also is implicated in this peculiar cavity formation, that, therefore, a *syringobulbia* develops. This anomaly is usually unilateral at the height of the fourth ventricle in the region of the nucleus of the pneumogastric, and thence passes anteriorly and laterally. In other cases the destruction is bilateral but asymmetric. The destruction terminates on the floor of the fourth ventricle above the nucleus of the vagus and the striæ acousticæ; its localization may be clearly understood from the illustrations appended to this article.

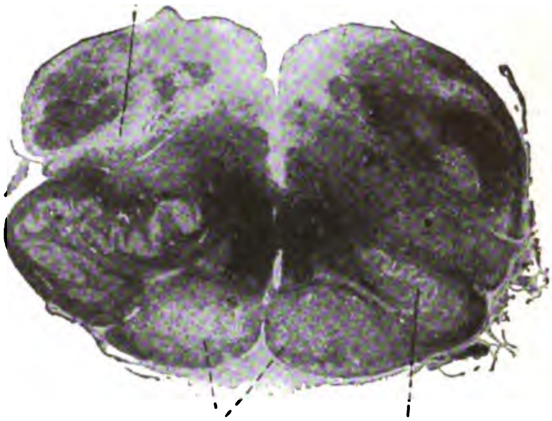

FIG. 180.—TRANSVERSE SECTION THROUGH THE MEDULLA OBLONGATA IN A CASE OF SYRINGOBULBIA. (After A. Westphal.)

FIG. 181.—SYRINGOBULBIA. (After A. Westphal.)

It is remarkable that in syringobulbia the spaces and cavities within the bulbus of the medulla oblongata are usually surrounded only by scant accumulations of glia, and the vessels show, as a rule, unusually thickened walls.

If we recall our physiologic knowledge of the function of the spinal cord, these anatomical findings furnish a simple explanation of many of the symptoms of syringomyelia.

The gradual disappearance of the large motor *ganglion cells in the anterior gray substance* causes the disintegration and, finally, the destruction of their nerve fibers and muscle bundles. As the lower portion of the cervical en-

largement is frequently first attacked, the muscles of the hand atrophy first, since the ganglion cells which innervate these are situated in this region.

As, moreover, so far as we at present know, the motor ganglion cells quite irregularly, each for itself but by no means symmetrically, atrophy in groups which belong functionally together, it is to be expected that in the larger muscles isolated bundles may be alone diseased, and in fact this is not rarely the case.

The destruction of the *posterior gray substance* inhibits the conduction of the *sense of pain*, as was shown by the well known experiments of Moritz Schiff in animals. The conduction of the sensation of touch, which often remains intact for a comparatively long time, is explained by the circumstance that the majority of the fibers of the posterior column frequently remain exempt, and to these fibers the conduction of tactile sense is chiefly ascribed.

If these are markedly implicated from the onset, the consequent disturbances keep pace with the limitation of other sensory functions; partial sensory paralysis is absent.

We are still ignorant as to the areas of the transverse section in which the nerve tracts which conduct temperature sensations are situated.

Trophic disturbances are usually attributed to destruction of definite areas of the transverse portion of the spinal cord.

Diseases of the gray substance must be considered the cause of these, although perhaps not a direct one. At all events, we know that such sequelæ do not occur, as a rule, in chronic myelitic processes which chiefly affect the white substance. Neither is the gray substance of the posterior horns normal in tabes dorsalis, in which similar changes occur.

If the disease in any way attacks the *lateral columns*, symptoms referable to the lateral column appear; above all, spastic paresis or paralysis of the extremities, especially the legs.

If the nuclei of the *pneumogastric* or *hypoglossal nerves*, or the fibers belonging to them in the bulb of the medulla oblongata, are attacked, derangements of deglutition, of the innervation of the larynx, and of cardiac and respiratory functions are produced. If the cavity formation in the gray substance extends as far as the *ascending trigeminal roots*, the same sensory disturbances occur in the area of distribution of this nerve as in those of the extremities.

If there is an extensive *tumor formation*, a gliomatosis, of course more or less decided pressure symptoms may result.

SYMPTOMATOLOGY

The foregoing enables us to understand that, owing to the irregular and varying distribution of the syringomyelitic processes, the *grouping of the symptoms* of this disease must necessarily vary.

Some *typical forms* may be differentiated. First, the most common, the so-called *classical* form, a case of which I have described.

In this variety, the symptoms of gradually increasing muscular atrophy, paralysis, and sensory disturbances, usually with partial sensory paralysis, are combined with trophic disturbances.

As subdivisions and special *types* we observe first a *cervical form*, as in the patient whose history I quoted, next, if syringobulbia develop, a *bulbo-*

medullary type, finally, the much more rare "*dorso-lumbar*" and "*sacrolumbar*" types.

In some cases the *motor* symptoms may be so prominent as to form the picture of chronic progressive muscular atrophy or of so-called amyotrophic lateral sclerosis.

In other cases *sensory* disturbances predominate, and there is either extensive analgesia, even resembling hysteria, or, at the same time, irritative symptoms develop like those of tabes dorsalis.

Finally, there are forms in which marked *trophic* disturbances appear. If whitlows and phlegmons develop, particularly upon the upper extremities, with analgesia, we have before us the so-called "Morvan's disease."

Deformation, if prominent, produces the *mutilating* form which bears a great resemblance to lepra mutilans.

Osseous and *arthritic changes* lead to the variety which Schlesinger calls the osteo-arthritic form; this sometimes causes a change in the hands and forearms similar to that in acromegalia. But the most common form is that in which progressive muscular atrophy of the hands and arms is combined with sensory disturbances and bulbar symptoms, as in the case which was detailed.

In all of these forms we usually find a variety of symptoms, and these we will now consider.

Beginning with *motor disturbances*, we note that the *irritative symptoms* of this disease are usually not at all prominent; certainly they more readily escape observation than debility and paralysis. *Fibrillary muscular contractions* are frequent, even with a sufficiently warm external temperature and during rest; contractions which also occur in other slowly progressive diseases of the great motor ganglion cells.

Tremor may occur, particularly in the hands, and, if increased by voluntary movement, as is usually the case, it resembles the tremor in multiple sclerosis.

More rare are *choreic* contractions which also appear during rest, and are chiefly confined to the upper extremities. Sometimes, although rarely, they are observed in muscles supplied by the bulbar nerves. I shall never forget a patient with this symptom whom I first saw in Friedreich's Clinic in Heidelberg. He held a handkerchief in his mouth continually, and only with the greatest difficulty could be persuaded to remove it. When he did so, against his will his tongue protruded, it trembled violently, and painful jerkings of the lower jaw pressed it against the upper teeth so that it was bitten. In order to prevent this, the patient had had all his teeth extracted. There were also irregular clonic and tonic contractions in the trapezius, and subsequently also in other muscles of the shoulder.

Since such contractions are only occasionally present we are forced to assume an especial predisposition of the patient to irritative conditions of this kind, but a certain relation to syringomyelia and syringobulbia present can scarcely be denied.

Tonic spasms may be persistent or occur in acute attacks. Contrary to the clonic spasms, they are much more common in the lower extremities; in rare cases they are distributed throughout the body, so that general rigidity and opisthotonos may appear. Laryngospasms also occur and may threaten life.

It is self-evident that with spastic paresis in the legs we frequently find also a *permanent rigidity* of the corresponding muscles.

Attention has only recently been called to peculiar *myotonic* disturbances occasionally found in syringomyelia. These fail to present all of the characteristics of true Thomsen's disease, myotonia congenita, particularly the simultaneous disturbance of almost all of the muscles of the body. But in some cases we note the same long-continued rigidity of individual muscles to the effect of cold as in the last named disease; also the same inability to relax the muscles which have been voluntarily contracted, particularly after a preceding long rest. We find the same peculiar and persistent pitting of the muscles after palpation which occurs in Thomsen's disease, and to some extent even the peculiar myotonic electrical reaction, which I cannot here describe. But these disturbances are found only in those muscles whose activity has been altered by the syringomyelitic process.

The *motor symptoms of weakness and paralysis, which occur with and without muscular atrophy*, are of much more significance in the clinical picture.

They usually develop slowly, particularly in the hands, and the appearance of weakness and atrophy is coincident. Sometimes, however, particularly in the legs, and when there is softening of the spinal cord substance following thrombosis, vascular constrictions, or hemorrhages, paralysis may come on very suddenly. This paresis of the arms is usually flaccid, of the legs spastic, except when associated with lumbar syringomyelia and destruction of the gray anterior horns in the lumbar enlargement. Spastic contractures of the muscles of the arm, the leg and the trunk rarely exist simultaneously.

The *localization of the atrophy and paralysis* varies greatly with the differing localization of the spinal changes. *Muscular atrophy* with debility is usually first noted in the *small muscles of the hand*, and shows a special predilection for the interossei primi externi. This, however, is generally by no means symmetrical.

If the ulnar region is first implicated, the *claw-hand* develops; if the median tract, which is more rarely the case, the *ape's hand*. When the various muscles are simultaneously implicated the disturbances are, as a rule, simultaneous. If, subsequently, the flexor muscles of the forearm become atrophic, the so-called *preacher's hand* may develop, and the claw-hand, on account of the preponderance of the extensors, is at the same time flexed dorsally. Usually, however, because of the atrophic weakness of the forearm, the extensor muscles are also implicated, or the atrophy, passing by the muscles of the arm, leaps from the hands to the muscles of the shoulder.

In other cases the disease is from the onset confined to the muscles of the shoulder, thence it passes to the muscles of the upper extremities and to the trunk. Here the muscle in its entirety is not always attacked, but it is often only partially involved, and most frequently the extensive area of the trapezius. This leaping of the disease from one muscle to another and its asymmetry are almost characteristic.

Atrophy of the muscles of the leg is rarer, and there is no definite rule for the development and progress of the disease. Pes equino-varus and pes calcaneus have been observed, but here the localization of the atrophy was very different and it involved also the muscles of the hip.

Hence it is evident that the most varied disturbances in the function of

individual members may arise, but these cannot here be detailed. It can only be stated that as spastic paresis is most common in the leg, so the gait typical of this condition is most frequently observed, while the ataxic, even the staggering, gait is more rare. Distinct ataxia has rarely been observed in the arms and hands. The disordered gait usually develops gradually; often very early on one side. Sometimes, however, disturbances in gait are sudden or quickly increase in severity.

The atrophies and paralyses in the course of the *bulbar nerves* are very interesting. Rarely is there paralysis of the motor branch of the trigeminal nerve on one side only. Paralysis of the muscles of the eyes, especially of the abducens, is uncommon, but more frequent is *paralysis of the facial nerve,* which is almost always unilateral, and generally affects only the branches supplying the mouth, although the upper branches also may be implicated. Like paralysis of the legs this usually develops slowly, yet may sometimes come on rapidly; the electric contractility of the muscles supplied by the nerve shows the same changes as that of the muscles of the hand.

Hemiatrophy of the tongue, in which one-half of the tongue becomes so thin that grooves develop, is more common. The *fibrillary contractions* are usually well developed. The absence of the power of motion, as a rule, causes the patient no great inconvenience.

Often there is unilateral *paralysis of the muscles of the palate and larynx.* Deglutition then becomes so difficult that it is almost impossible to swallow a bolus, and ingested foods are regurgitated through the nose. Examination of the velum of the palate discloses the defective elevation of one-half upon phonation, also the retraction of the uvula to the healthy side and fibrillary contractions.

In the *larynx* we can determine only unilateral *paralysis of the recurrent laryngeal nerve,* which at first involves merely the abductors of the vocal cord, but finally becomes complete. Sometimes the paralyzed vocal cord is decidedly atrophic, and the voice becomes rougher and hoarser. Paralysis of the vocal cord may come on very gradually, or it may occur suddenly and rapidly become worse.

In rare cases the paralysis is bilateral. Unilateral paralysis is often combined with paralysis of the palate and esophagus of the same side; if there is simultaneously hemiatrophy of the tongue, this is also found upon the same side; therefore we have the picture of a *unilateral lower bulbar paralysis.* In such cases the voice is not only hoarse but has a nasal tone, and speech may be difficult; now and then it is slow and scanning as in multiple sclerosis, a symptom the cause of which is still unknown.

It is not remarkable that, under such circumstances, *cardiac activity* should sometimes be disturbed by the changes in the nucleus of the pneumogastric nerve. *Tachycardia* has not infrequently been observed, more rarely *bradycardia. Dyspnea* may follow these disturbances, which in different cases must be ascribed to various causes.

Dyspnea may arise from paralysis of the posterior cricoarytenoid muscles and may readily be recognized by the accompanying stridor; it may be due to increasing atrophy of the muscles of inspiration and expiration, and is then steadily progressive and increased by the slightest exertion. It may occur paroxysmally without any changes of this kind, owing to sudden paralysis of

the respiratory muscles. In this case, if syringobulbia soon becomes marked, an early and fatal termination may be looked for.

The results of *electric examination* of the nerves and muscles vary. With a slow progress and prolonged duration of atrophy in the muscles, direct and indirect irritability is more or less diminished, and DeR rarely occurs, most rarely that complete form associated with rapid atrophy. This is partly due to the fact that the atrophied muscle masses frequently contain some muscle fibers which still react well.

Mechanical irritability corresponds with this; upon tapping the muscles we rarely note distinctly sluggish contractions.

The *sensory disturbances* are of especial significance.

Here *irritative phenomena* are less important than paralytic symptoms; nevertheless, they are very prominent. Especially in regard to the sense of temperature, *paresthesias* appear in the form of *burning*, of *hot*, and of *cold sensations*. Ordinary tactile paresthesias appear to be rare, which corresponds with the fact that paralytic symptoms also are chiefly confined to the realm of temperature sensations and are less often found in the tactile.

Pain may be of a tearing and boring as well as of a burning character. It is rarely so marked as to cause suffering. Usually it is so persistent that rheumatism is simulated. In some cases, however, pain is of the same *lancinating* character as in tabes. Here and there a girdle sensation is complained of.

The *seat* of the paresthesia and pain corresponds with the extent of the spinal and bulbar substance implicated. These symptoms are most frequently found in the arms and the upper parts of the trunk, more rarely in the legs and the region of the fifth nerve, sometimes even in the occipital region. They may appear quite early as well as late, and they may be combined for a long time with other symptoms of the disease.

The peculiar, *sensory, paralytic symptoms* are much more constant; the discovery of these about 1880, together with the atrophy, first made the diagnosis of syringomyelia possible.

Partial sensory paralysis or *dissociated "syringomyelitic"* sensory paralysis, as this condition has been called is often noted, although it occurs also in other diseases of the nervous system. This was present in my patient. While tactile sense is normal or shows only slight impairment, pain and temperature sense are markedly decreased. It is erroneous, however, to assume that the tactile sense is normal throughout the entire course of the disease. Sometimes it is but slightly impaired, and it is wholly absent in at most only a relatively small part of the cutaneous surface.

The *sense of pain*, corresponding with the anatomical seat of disturbance, is most frequently diminished or absent in the hands and arms, in the upper portion of the trunk, in the skin as well as in the deeper areas, and in the joints and bones, although neither simultaneously nor symmetrically.

If syringobulbia appears concurrently, the scalp and mucous membranes of the head are implicated. Analgesia or hypalgesia in the legs is rare. The specific pressure pain in the testicle may disappear while, as Schlesinger has demonstrated, pain is almost invariably evoked by pressure over the eye-ball.

The time of development of hypalgesia and analgesia conforms to no rule. It usually appears in the early stages of the affection, and generally is coin-

cident with the implication of the temperature sense and the development of muscular atrophy.

The study of the *disturbance of the sense of pain and its distribution,* as undertaken in the last few years by Laehr, Schlesinger, Brissaud and others, is highly interesting. Although these investigations have not yet been concluded, it is evident that very different areas are involved.

It might be supposed that these would correspond with the areas implicated in diseases of the peripheral nerves. But this is not the case. On the contrary, the analgesic areas are the same as those frequently found in diseases of the posterior nerve roots and the *spinal cord segments* belonging thereto, and consequently they are of a *segmental type.*

The localization of the hypalgesic and analgesic areas is as follows: They appear on the trunk as a series of transverse zones, extending horizontally and parallel with each other to the median line, while on the extremities they are noted as bands which extend continuously upward and parallel with the longitudinal axis of the limbs until they reach the trunk, passing thence to the median line. Here and there the analgesic bands assume a spiral form.

In the *face* and on the head the hypalgesic zones are of peculiar appearance. If higher segments of the cervical portion of the cord be affected the limitations are invariably found to be higher, usually as irregular arches with the concavity toward the front. As a rule, the upper half is parallel with the edges of the hair, and the lower with the border of the lower jaw, so that anteriorly the nose and its immediate surroundings generally remain uninvolved.

This is graphically portrayed by the diagrams here inserted.

Fig. 182.—The Various Limits of the Zones of Sensory Disturbance upon the Head. (After v. Soelder and Schlesinger.)

Secondly, a *rare form* of hypalgesic and analgesic disturbance is that in which the zones correspond with the affection of individual members or their parts, in which therefore the hands and arms are chiefly and uniformly attacked, and the zones disappear while passing upward spirally. On the trunk sensory defects may appear in "waistcoat-like" forms. If the whole trunk has become analgesic a shirt-like analgesia is produced, or, more correctly, a jacket-like analgesia, since the limit is usually not so low down.

These zones of analgesia correspond with those found in *hysteria,* but they differ by their greater persistence; in syringomyelia the intensity of the hypalgesia may also vary. But other symptoms of hysteria are absent. Of course, in some of the cases hysteria may be associated with syringomyelia as with other organic diseases.

How this member-like limited analgesia may be derived from anatomical changes is still not clear, in spite of ingenious hypotheses. But, inversely, it seems to throw some light upon hysterical analgesia and its nature, a point which I cannot here discuss.

Thirdly, when there are extensive changes in the posterior gray substance and the long sensory tracts, a sensory paralysis of the Brown-Séquard type may exist, also a bilateral form involving the entire trunk and the legs, as well as the segmental type in the arms, perhaps also in the face. In the Brown-Séquard variety, besides the partial sensory paralysis which is usually very conspicuous upon one side of the body, there is simultaneously a marked motor paralysis upon the other side.

The *mucous membranes* most frequently affected are those of the pharynx and oral cavity, especially their posterior portions; more rarely the conjunctivæ as well as the bladder, urethra and rectum. Analgesia may reach its highest degree in any of these areas.

It has been stated that these disturbances may vary in intensity. In complete analgesia they are, however, rarely observed and are not of a decided nature.

The localization of derangement of the *temperature sense* resembles that of the pain sense, although its distribution and degree do not exactly corre-

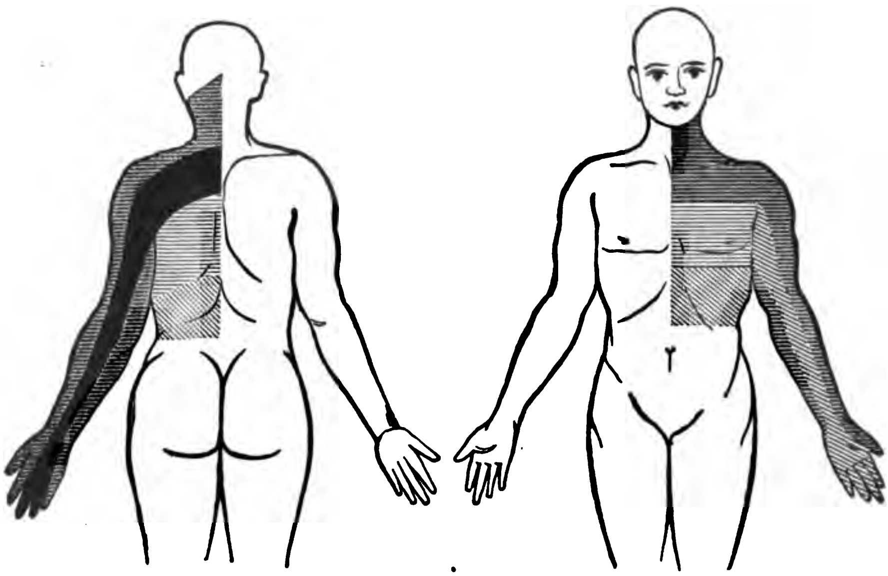

Fig. 183.—Disturbance of the Pain Sense, Segmentally Limited. (After Schlesinger and Hahn). The darkest areas correspond to the zones of complete analgesia, the lighter to varying but slighter degrees of disturbance.

spond. Like disturbance of pain sense, this may occur early and rapidly lead to injuries, particularly to burns.

The temperature sense may be diminished in any degree, and this disturbance may vary in that the sense for heat or for cold only may be impaired, or that the sensations may vary in degree of impairment. There may be a

variation in the rapidity of conduction of impressions for heat and cold as well as an inversion of these sensations, so that heat is mistaken for cold and *vice versa,* or cold and heat are appreciated only as coolness and warmth. A delayed temperature sense has been observed in rare cases.

The disturbance of temperature sense is almost invariably combined with disturbance of the sense of pain; but it may occur earlier, and in some cases is said to be the only permanent defect in sensation.

The *mucous membranes* and the *deeper areas* may be implicated just as in the disturbance of sense of pain.

Much less important is disturbance of other kinds of sensibility; for instance, anomalies of the so-called *muscular sense* and *sense of position.*

In the lower extremities the sensation for active and passive movements, for weight and resistance, may be more or less impaired, owing to the fact that localized changes gradually take place in the posterior columns in syringomyelia just as in ordinary tabes, or because an actual combination of these diseases exists. *Ataxia* may then be observed which, unlike that in tabes, sometimes affects one leg only. It may, however, occasionally attack both an arm and leg of the same side.

Bilateral ataxia of the arms alone has never been observed. But the cerebellar gait as well as Romberg's phenomenon has been noted.

The *pressure sense* may be disturbed. Schlesinger made tests with a special instrument (*algesimeter* of Max Herz and Hermann Schlesinger) and found that the sensibility of the skin to pressure exerted upon it was sometimes diminished or absent, while the tactile sense was retained. The pressure sense of the skin also varies from that of the deeper tissues; therefore in this respect also the pain and temperature senses may be analogous.

Under these circumstances it does not appear remarkable that any diminution of sensation in the deep as well as in the superficial parts, as well as the so-called *stereognostic* sense, may here and there be lost, so that the patients are no longer able to distinguish the differing substance of articles placed in their hands.

In conclusion, the peculiar sensation produced in the bones by placing a vibrating tuning-fork upon them may be absent; this is neither remarkable nor, at present, of especial signifiance.

Much more important is the *third group* of symptoms, *trophic disturbances,* which are found in great variety upon the *skin* as well as in the *bones,* the *joints,* and *other parts* of the body.

Many abnormalities are observed, especially in the hands, where they resemble those due to neuritic changes, but in neuritis they are never so extensive, so regular nor so varied as in syringomyelia.

In this affection the hands may be swollen, the skin abnormally red, and erythromelalgia may be simulated; in this, however, there is also severe pain. At other times the skin may be very red but smooth and firm, so that we call it "glossy skin"; changes similar to those of scleroderma and sclerodactylia may appear, but the skin is firmer and more tense. Often it becomes cyanotic, and shows changes resembling those which take place in the beginning of Raynaud's disease from deficient arterial nutrition of the finger-tips. Marinescu has described a special "main succulente." In this condition the whole or a part of the dorsal surface of the hand is swollen yet does not pit upon

deep pressure with the finger. The fingers are likewise affected, and are swollen into a spindle-shape; the skin is smooth and glistening, and when cold is reddish blue. In other cases there is edema.

Vesicles and eczema are observed, the latter, especially, being chronic. The vesicles resemble those from burns and not rarely are due to this cause. But, like *pemphigus vesicles* they usually develop from an unknown cause, they do not itch, and they usually desquamate without leaving scars.

Deep fissures, especially in the transverse folds of the skin, the so-called rhagades, are very conspicuous and slow to heal, as are also *ulcerations, cicatrices* and *indurations* of all kinds and origins. Sometimes the patients do not know how these have arisen. Sometimes the parts have been burned, crushed or bruised without any sensation of pain having developed, and consequently their treatment has been neglected.

Whitlows are not so rare, as in my case, and a number of these may occur in close succession. Unlike the ordinary form in which they are very painful, they run their course painlessly; after Morvan they have been designated "panaris analgésique" and when they occur with "paréso-analgésie" of the upper extremity they have been considered a special type of syringomyelia, since they have never been actually observed except in this disease. Being painless, they are frequently neglected, hence there are often severe sequels, more often than in the ordinary form; sometimes the nail and a part of the bone are permanently destroyed, the fingers are shortened and their tips clubbed.

Deep *suppuration* in the hand also may generate necrotic processes and cause stiffening of the joints and loss of substance; marked deformities resembling those of leprosy may occur and to these the term of "syringomyelia mutilans" (Hoffmann) has been applied.

Arthritic inflammation and non-suppurative inflammation of a tendon are also frequent; in some cases a thickening of the entire volume of the hand may make the condition simulate acromegalia. I recently saw a case of this kind in which a different diagnosis had been made, apparently because the popular nomenclature for this condition had led to an erroneous conception. The other symptoms of Pierre Marie's disease, particularly the changes in the head, were absent, and the classical features of syringomyelia present.

Other parts of the body as well as the hands may be attacked by *trophic disturbances*; the upper arm, the upper part of the trunk, or even the face and the legs. Erythema may appear temporarily on the *skin*; not infrequently we note the so-called *writer's skin,* sometimes spinal *edema* develops and is almost wholly confined to the arms, being unilateral, and lasts hours or days. Urticaria is more common, in the form artificially produced by scratching as well as that which is spontaneous. It differs from other varieties of urticaria in developing only upon the analgesic and anesthetic areas of the skin. Vesicle formation is found only in those parts altered by syringomyelia; a pemphigus affecting the entire body has very rarely been observed. Herpes zoster also is extremely rare, but this may appear as the gangrenous herpes zoster described under syringomyelia.

The changes in this area produced by altered *sweat secretion* are very interesting. *Hyperhidrosis* as well as *anhidrosis* occurs. Schlesinger has also described a "paradoxical sweat secretion" in which the patient perspires under

the influence of cold, not under that of heat. Usually these alterations in the secretion of sweat are circumscribed and unilateral, stopping at the median line, and are most common in the face, next in the trunk, and sharply circumscribed in circles. Often, however, arms and hands unilaterally and bilaterally are affected; this is much rarer in one leg. These anomalies sometimes develop early, and occasionally are said to be the first symptoms of the disease. Their duration varies; frequently they persist for months or years.

Deep inflammation of the subcutaneous tissue or other soft parts, especially that of a purulent, phlegmonous character, occurs in the hands but also elsewhere, most frequently in the arm, rarely in the feet and toes. This is painless, and may persist for a long time. Here and there it is steadily progressive, and may finally cause death. It is usually due to infection after injuries which the patient with syringomyelia has sustained; in how far it may be caused by a greater predisposition and vulnerability of the tissue itself in consequence of nervous disturbance is difficult to decide. In addition to the ordinary bed-sore which is so common in severe and progressive diseases of the spinal cord, *gangrene and necrosis* may also occcur. *Mal perforant,* perforating ulcer of the foot, which occurs in tabes and peripheral neuritis, is also seen in this affection. But circumscribed acute necrosis may rapidly develop upon the skin of other parts of the body, either spontaneously or after an assignable cause. As in mal perforant, desquamation of the gangrenous tissue is sharply demarcated, leaving a round ulcer which usually shows little tendency to heal. Here we must also mention Raynaud's disease which is rarely observed in syringomyelia.

It will be observed that these symptoms are referable to the domain of dermatology, but the frequent *arthritic and osseous affections* which so often accompany syringomyelia and which may even *introduce* the disease, belong to the surgeon. As in tabes, *inflammatory and deforming diseases of the joints* are common. Certainly the analgesia of the deeper parts in combination with the effect of trauma is the cause of this. To what extent nervous influences directly effect this by an alteration of the vascular innervation and the chemism cannot be stated. Certainly their conjoint action must be assumed. It is different in tabes, particularly when the upper extremities are affected, hence, according to Schlesinger's statistics, 80 per cent. or more of arthritic affections occur in the arms. The shoulder-joints are most frequently affected, next the elbow-joints, and less often the wrist. Even the joints of the jaw and the sternoclavicular joints may be affected, although rarely; in a somewhat larger proportion of cases the knee- and hip-joints are involved. As a rule, these changes take place on only one side of the body and are most common in the male, which is probably because males are more exposed to trauma. According to statistics, these changes in the joint are noted in 10 to 25 per cent. of all cases of syringomyelia. They develop in various ways: Either slowly with moderate pain and with grating sounds on active or passive movements of the joints, or suddenly, with or without such prodromes, by the appearance of swelling in the joint due to a considerable effusion into its cavity. *There is usually no pain.* The swelling may persist for days or weeks and then disappear, but readily returns. The usual changes seen in the anatomical picture of a deforming joint inflammation now develop, the individual features being partially determined during the life of the patient by the aid

of the X-rays. As a rule, there are at the same time atrophic and hypertrophic processes in the membranes of the joint as well as in the cartilage and the neighboring bone. *Spontaneous luxations* may occur, particularly in the shoulder-joint, and these may become so habitual as to divert attention from the underlying syringomyelia which remains unsuspected. In other and rare cases there is ankylosis. The atrophic change, as well as the accumulation of fluid, is usually most marked in the affected shoulder. In the elbow-joints hypertrophy is more prominent, particularly of the bony parts. When the wrist is affected, we note dislocation of the hand to the volar side and swelling of the bones of the forearm, while the changes occurring in the hip- and knee-joints are chiefly hypertrophic. The periarticular soft parts are also sometimes implicated; the muscles and tendons of these areas may swell and sometimes show a partial ossification. Here and there *purulent inflammations* develop in the joint and its surroundings, most readily in the wrist-joint. In the hands and forearms, where, as has been stated, fissures, erosions and ulcerations are most common, infection readily takes places and may be transmitted from the skin to the deeper parts, to the bursa mucosa, the tendons, etc. Sometimes primary necrosis occurs in the bones and leads to arthritic suppuration; or the purulent infection may be traced to a fistula which has arisen in the inflamed joints from external rupture of the sac containing fluid. Such purulent arthritic inflammations like phlegmons may be rapidly progressive and lead to death; but they may also terminate favorably, particularly under suitable and early treatment. At other times fistulæ may persist for a long time.

The changes in the *bones* are quite peculiar; and *fractures of the bone* may occur under singular circumstances.

One of my patients, a baker, fractured his arm during ordinary kneading of dough. Although he felt no pain, yet crepitation and the thickening of the arm attracted his attention. A patient of Bernhardt's also sustained a fracture of the ulna while at work, but continued to break stones, and on the next day even carried water. A patient of Schlesinger's, while turning her bed-cover, heard a loud cracking sound, and found that she had fractured the bones of the left forearm. In my experience, the *bones of the forearm*, in particular, are prone to fracture upon the slightest movement without producing pain, and the fracture is a simple transverse one. Such fractures may occur also in other bones, less frequently in the legs than in the arms. Curiously the right side seldom appears to be implicated, in spite of the fact that most persons are right-handed. The fractures are apt to occur only in the late stages of syringomyelia; they may heal normally, quite rapidly, or may require a long time. The callus may be normal or may develop in superfluous quantities. Here and there pseudo-arthroses may form. What changes cause these bones to fracture so readily has not been sufficiently investigated. Naturally atrophic processes are thought of; but in one of my cases I was unable to determine any such change. A difference in the structure of the osseous trabeculæ or a decrease in calcium has been suggested. At all events, we cannot exclude physical or chemical changes of the bony substance due to nervous disturbance, although in some cases the assumption of an immoderate action of the muscles on the bones while at work appears to be sufficient. On account of the loss of muscle and pain sense, the pa-

tient may not be aware that he is subjecting these muscles to any excessive strain.

Besides fractures, atrophic and hypertrophic changes occur in the bones, especially in those of the hand, of the wrist, and of the forearm. Now and then exostoses, even multiple ones, have been found on the bones of the upper extremities and the upper portion of the trunk, changes attributed to existing syringomyelia. That enlargement of the bones of the hands and forearms with a corresponding growth of the soft parts may simulate acromegalia has already been stated; but these are cases merely of pachyacria (Arnold).

Spontaneous necrosis may occur in the small bones, in the long tubular bones, or in the scapula without preceding a periostitis which always impairs the nutrition of the bony substance. In the bones as well as in the skin necrotic processes may be the direct result of internal, but still unknown, changes, hence vascular spasm or a chemical action upon the nerves must be considered. In the hands and feet the desquamation of detached fragments of bone may be observed.

In how far the numerous diseases of the vertebræ may be referred to primary changes in the bone cannot be decided at present. At all events *scoliosis* and *kyphoscoliosis* are very common, occurring, according to Bernhardt and Schlesinger, in about 25 per cent. of the cases, and according to other authors much more frequently. In the last patients I have observed they were almost invariably present. Scoliosis usually develops first, and in the thoracic vertebral column; later compensatory curvatures of the deeper parts are formed and kyphosis is added. These changes usually arise in the later stages of syringomyelia, but sometimes they occur in the beginning. Their pathogenesis is by no means clear. Unilateral atrophy of the muscles of the back and shoulder with a unilateral limitation of muscular activity has certainly an influence. Nevertheless, primary bony changes are observed in cases in which there is no such atrophy, therefore these may be produced by nervous influences. Sometimes there are simultaneous congenital changes, particularly spina bifida.

The *thorax* shows a change in form corresponding with that of the vertebræ, a peculiar malformation to which Pierre Marie and Astié have given the name "thorax en bâteau." The anterior and upper portion of the thorax present a depression; the shoulders protrude anteriorly, but neither atrophy of the pectoral muscles nor kyphoscoliosis necessarily accompany it.

Other anomalies of the *nervous system* which may appear in syringomyelia must be enumerated. The *reflexes*, especially the *cutaneous*, show no peculiarities; they are absent when the conduction arcs or their central stations are destroyed, which, however, is rarely the case; this applies also to the reflexes of the abdominal walls. Often they are increased; the Babinski toe-reflex may be present either where the pyramidal tracts are directly altered or merely irritated from pressure; the *tendon reflexes* of the arm are usually early lost or they decline in strength, especially when atrophy is prominent, while in the legs, even in this stage of the disease, they are either unilaterally or bilaterally more or less increased. Of course, they may be absent or diminished when there is cavity formation in the spinal cord which extends to the lumbar portion or is chiefly seated there, or when the posterior columns and posterior roots are implicated, as may be the case in a complication with

chronic meningitis. Now and then when syringobulbia is combined with degeneration of the cervical pyramidal fibers, or with an increase in pressure of the fluid in the fourth ventricle, then the tendon reflexes in the arms and lower jaw may be increased.

Various disturbances in the function of the *bladder, rectum* and *genital organs* may appear. Since the seat of motor change is usually the central gray of the cervical portion, in the majority of cases these disturbances either do not occur or only appear late. In lumbar syringomyelia bladder disturbances early become prominent. If massive gliotic or gliomatous proliferations from within press upon the white columns of the spinal cord, the same disturbances may be produced as in compression of these parts.

The *bladder disorders* are usually of a motor nature; as a rule, there is at first difficulty in urination, then incontinence, sometimes even early the incontinence of retention, cystitis may be added. As nephrolithiasis has been somewhat frequently found, this condition also has been directly attributed to the spinal affection.

Sensory disturbances of the bladder are rare, but they may exceptionally implicate the temperature sense of the mucous membrane. Strangury may appear with or without other sensory disturbance of the mucous membrane of the bladder, in which case there may be a decided retention of urine yet the patient be unaware of the condition.

Gastrointestinal disturbances, from simple constipation to incontinence, are rare; but sensory disturbances may sometimes exist which render the patient unconscious of the evacuation of feces.

Little is known of anomalies of the *sexual functions.* As the lumbar cord is usually not involved, these may be unimpaired; however, not rarely there is a disappearance of sexual desire although erection and ejaculation may be normal. In the case of pollutions the sensation of libido may be absent.

Concerning disturbances of the cranial nerves, in so far as these have not yet been considered, especially in regard to disturbances of sight, the following may be mentioned:

The olfactory nerve is not implicated; the optic nerve may be affected by complications, or when marked gliosis and gliomata have developed in the bulbus medullæ and the pressure of the cerebrospinal fluid is consequently increased, *choked disc* may then appear and gradually pass into atrophy. The other rare anatomical changes in the optic nerve depend upon complications. Of course, in severe disturbances the faculty of sight is correspondingly impaired.

Derangements of the field of vision have been frequently observed and studied. It has been shown that there is usually no limitation of the visual field, that in some cases a narrowing due to hysteria arises, but that it is more often observed without hysteria. A defective appreciation of colors, particularly green, is said frequently to exist.

The *auditory nerve* is rarely affected, although in syringobulbia the spaces and glia folds may implicate the individual nuclei of the auditory; not those which are connected with the sense of hearing, but those which serve to maintain the equilibrium. Notwithstanding this, however, it cannot be denied that the trunks of the auditory itself, or the acoustic portions of the central apparatus of this nerve, may suffer from increased pressure due to marked glia

proliferation, from hemorrhage, etc. Certainly in some cases more or less defective hearing or perverted hearing can be assigned to no other cause.

The *sense of taste* is often impaired. Its disturbances, like other bulbar symptoms in syringomyelia, are usually bilateral. The anterior as well as the posterior portion of the tongue may be involved with or without simultaneous decrease of sensation. Now and then only certain perceptions of taste are impaired or destroyed; perversions may also appear. In how far these disturbances are due to alterations of the nucleus and tracts of the glossopharyngeal or the fifth nerve, has not yet been determined.

Disturbances on the part of the *vagus* have already been described; when gliosis produces marked pressure in the bulbus, *vomiting* may appear. Sometimes, as Friedreich and I observed, *diabetes insipidus* may develop, while glycosuria is rare. Persistent and transitory glycosuria have been observed, as well as an alimentary form.

Among the *general cerebral symptoms* which may accompany syringomyelia, *vertigo* must be mentioned. It occurs in paroxysms in the form of rotary vertigo, and is quite independent of muscular disturbance of the eye. Sometimes it is so intense that the patient falls; usually there is no loss of consciousness. In explanation of its development affections of the vestibular nerve have been considered—those which contain the fibers for the equilibrium. Hemorrhages, changes in arterial tension, anemia, thrombosis, edema, and sudden or rapid increase of the pressure of the cerebrospinal fluid may cause the vertigo.

Apoplectiform attacks not rarely occur, probably from the same causes, and may be conjoined with vomiting, dyspnea, and changes in the pulse, as well as with various succeeding paralyses in the bulbar area and the course of the spinal nerves. Paresis of certain cranial nerves as well as weakness of the arms and legs or the cerebellar gait may be more or less persistent. Now and then sensory disturbances follow, chiefly paresthesia.

Independent of other attacks there is often *severe headache* which is probably due to increased pressure of the cerebral fluid.

Among the complications are decided *psychical disturbances* which are not rarely caused by a predisposition to anatomical anomalies of the brain as well as to an abnormal composition of the spinal cord. Of course, the sometimes very rapid rise in cerebral pressure noted in syringobulbia may produce a transitory dulness and confusion of mind.

Generally, however, there is no implication of the psychical functions which produces true mental disturbance. But Schlesinger states that, in his experience, patients always show a peculiar behavior in regard to analgesia and thermo-anesthesia. Even if specially instructed that the application of heat will not lessen their feeling of cold, they persist in burning themselves in some way. I have never observed anything of this kind.

COURSE AND PROGNOSIS

In describing the course of syringomyelia and syringobulbia I may be brief. The disease is of long duration; it may be protracted through years or decades. The cases combined with gliomatosis are more speedily fatal, terminating in from one to six years, but of this we have no accurate statistics.

Syringobulbia is not likely to be so soon fatal, for instance in a few years; but it may last a dozen years or longer. In this affection death occurs earlier when the nuclei of the vagus are implicated than in the ordinary cervical and dorsal syringomyelia, while the rarer lumbar affections combined with cystitis and its consequences are even more rapidly fatal than the first mentioned. Variations in the course of the disease are not uncommon. I have already mentioned apoplectiform attacks with their sudden aggravations; these may occur in true syringomyelia without implication of the bulbus; usually the symptoms gradually increase in severity. The onset varies greatly. It may be marked by paresthesias with succeeding analgesia as well as by atrophy, various trophic disturbances, and sometimes bulbar or bladder symptoms. Intercurrent diseases, particularly enteric fever, may aggravate the condition. Sepsis after any kind of suppuration, and a complication with tabes, progressive paralysis or other disease of the central nervous system may hasten the end. Otherwise, and so far as duration of life comes into question, syringomyelia is a benign affection, but it leads to early invalidism and deformity.

OCCURRENCE AND ETIOLOGY

In regard to the *occurrence* of the disease, it must be emphasized that syringomyelia and syringobulbia, contrary to the views long maintained, are comparatively frequent manifestations of disease of the central nervous system. True, they are not so common as ordinary tabes, and probably not so frequently noted as multiple sclerosis, but still they are much less rare than motor (spastic) tabes in its various forms and the combined system diseases in general. Even acute myelitis is not so common; naturally I except compression, the poliomyelitis of children, and that due to syphilis. *Men* are more frequently attacked than women; according to Schlesinger the proportion is 2 to 1. Youth, especially the second and third decades, is the most vulnerable period; but the disease is also found in the aged. Now and then *heredity* has been observed.

The *cause* and *pathogenesis* of the disease were described under the pathologic anatomy. I shall only repeat here that these are not uniform, that an inhibition of development must be considered, and that hemorrhages, inflammation, thrombosis, and perhaps also vascular obliteration, may play a rôle. The view that *leprosy* leads to syringomyelia is absolutely untenable from anatomical demonstration; moreover, it cannot be maintained that peripheral neuritis leads to the disease by an advancing inflammatory process and the formation of cavities in the central nervous system; for, in the first place, syringomyelia has never been observed even after most advanced and extreme polyneuritis with disease of the posterior columns or the ganglion cells of the anterior gray substance, although there may, of course, be an accidental combination of these pathological conditions. Ascending neuritis, too, is very rare, even if the diagnosis be based only upon the development of the clinical symptoms; finally, additional *ad hoc* hypotheses are necessary to explain why, in cases quite analogous clinically, like those in which an ascending neuritis is followed by a secondary syringomyelia, the posterior and anterior nerve roots which enter have not been "neuritically" altered.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

The *diagnosis* and *differential diagnosis* are difficult problems. For the recognition of classical cases of syringomyelia and syringobulbia they are quite easy, but not otherwise. In the first place the existence of the disease may be overlooked, particularly in the early stages, and especially when merely vague trophic disturbances of the skin and joints or isolated bulbar symptoms are present. Even in the examination of apparently simple affections we should invariably be thorough; as, in furunculosis or in certain eczemas, diabetes mellitus is thought of and investigated, so also in the case of whitlow, disease of the joints, and unilateral disturbances in the secretion of sweat, etc., the possibility of syringomyelia must be considered. If there are no symptoms, the probability of cavity formation cannot be decided.

This disease may also be *confounded with others*: (1) With functional nervous disease, *hysteria*; (2) With *destructive diseases of the nervous system*; (3) With *certain diseases of other origin.*

Unless it be an accompanying affection, *hysteria* can only seriously come into question when it is the rare *sensory* form of hysteria with analgesia. In both diseases the previously described analgesia limited to members may be present, while a form limited to segments favors syringomyelia, and the hemianesthesias of hysteria manifest a different symptomatology. Another decisive fact is that hysterical analgesias may be psychically influenced, they often appear and disappear suddenly, which is not the case in syringomyelia.

The disease must be differentiated from many *other organic diseases of the nervous system.* When syringomyelia runs its course without sensory disturbance, which is rarely the case, it may be confounded with *amyotrophic lateral sclerosis* with or without amyotrophic bulbar paralysis, with *atrophy of the nuclei of the motor spinal* or the bulbar, ganglion cells, or perhaps with chronic poliomyelitis; but in fully developed motor tabes the entire course of the disease is more rapid, the atrophies and spasms are symmetrical, the paralysis is more extensive and more moderate. Paresthesia and pain are usually absent. Further, DeR and fibrillary contractions are generally more conspicuous and distributed. The bulbar symptoms are symmetrical, and not wholly or in part unilateral, as in syringobulbia.

Atrophy of the anterior spinal ganglion cells alone is extraordinarily rare. This can be diagnosticated only when the slowly developing muscular atrophy of the hand and arm has existed for years, and when we are thoroughly convinced that after this long time there are no indications of sensory disturbance. Otherwise the affection is most likely syringomyelia.

Chronic poliomyelitis is also quite rare, and differs by its much more rapid development, occurring within weeks or months, and by early and conspicuous DeR. Lead paralysis may produce the condition; this can be determined from the history.

Dystrophia muscularis may sometimes occasion perplexity, particularly if it be Erb's juvenile form with exclusive implication of the muscles of the arms and shoulder; for syringomyelia, as we have seen, may sometimes begin with atrophy of the muscles of the shoulder. But dystrophy attacks the muscles much more uniformly and symmetrically, and runs its course almost without fibrillary contractions and reactions of degeneration. Finally, hypertrophy

of the infraspinatus muscle, which is almost always present, contraindicates syringomyelia, while increase of the tendon reflexes of the legs or even spastic symptoms are in favor of the latter disease.

As *multiple sclerosis* sometimes runs its course with muscular atrophy of the hands or even partial sensory paralysis, and, on the other hand, intention tremor, even scanning speech and nystagmus may appear, in syringomyelia, confusion in diagnosis is possible. But in sclerosis the symptoms relate to the head, for instance, headache, changes in the temporal half of the papilla, and peculiar visual disturbances which begin as definite central color scotomata and frequently lead to transitory but decided amblyopia. Besides, the scoliosis of the vertebral column and all of the previously described trophic disturbances are absent.

Ordinary chronic myelitis affects chiefly the white substance, and produces *early* spastic and spastic-paretic symptoms, frequently referable to the bladder, but there is neither muscular atrophy nor dissociated sensory paralysis. If syringomyelia is considered to be a peculiar *central myelitis* running its course with cavity formation (otherwise it does not occur without spaces and cavities), we are dealing with syringomyelia itself. The differential diagnosis of *spastic paralysis* may sometimes occasion great or even insurmountable difficulty if there is dorsal syringomyelia with implication of the lateral columns, which has been now and then observed. When sensory or bladder disturbances appear, it is certain that the rare affection, Erb's spastic paralysis, is not present.

It is sometimes impossible to differentiate *chronic syphilitic meningo-myelitis* owing to cavity formation. Generally, however, the ordinary picture of spinal, and sometimes of simultaneous cerebrospinal, syphilis may be differentiated by the more rapid development of the individual symptoms, by the severity of the pain in the back and in the head, by the early implication of the brain and the nerves supplying the muscles of the eye, and, above all, by the improvement frequently seen after antisyphilitic treatment. In syphilitic meningo-myelitis we almost invariably observe variations in the severity of the symptoms and relapses with new symptoms. Of course, these are not lacking in syringomyelia, for, as we know, apoplectiform attacks may supervene; but in syringomyelia there are trophic disturbances and curvatures of the vertebræ, which is not the case in syphilis.

If *pachymeningitis* or chronic leptomeningitis is coexistent, there is early *rigidity* of the neck and back, and the *pain* upon *pressure* is much greater than in syringomyelia; but this affection may be combined with chronic pachymeningitis, hence cannot be diagnosticated. If extensive gliomata are present, there is stiffness of the vertebræ and greater pain, but usually only in the late stages of the disease.

Caries of the vertebral column may usually be excluded, since in this affection, at least in the cervical portion of the vertebral column, deformities gradually occur with other symptoms of tuberculosis. There are no dissociated sensory disturbances. *Extra-medullary tumors* cause a prolonged initial period of pain which does not occur in syringomyelia, although it may be present in gliomatosis and gliomata.

The differentiation from *tabes dorsalis* is sometimes difficult because sensory irritative phenomena, such as lancinating pain or even girdle sensation, may be prominent in syringomyelia; also because partial sensory paralyses

occur in both diseases, and the diagnosis causes the greatest perplexity if the syringomyelia be seated in the lumbar region, for this produces an early disturbance of the bladder and the loss of the patella tendon reflex. In differentiation we should bear in mind: 1. That in tabes there is often rigidity of the pupils with feeble reaction to light, symptoms which do not occur in syringomyelia; 2. That in the last named disease we never observe the peculiar, narrow, segmental, horizontal zones of *hyperesthesia* tactile irritations which in tabes often appear so early in the skin of the trunk. The concurrence of these diseases cannot be diagnosticated, but can at most only be presumed when both atypical clinical pictures are present.

Hematomyelia in its entire localization produces the same symptoms as syringomyelia, but its onset is sudden or, at least, it develops rapidly.

The differentiation of *syringomyelia* from *polyneuritis, including the form seen in leprosy,* was described under the differential diagnosis of the case whose history I detailed. In this much discussed differentiation from leprosy, it might be added that the entire facial nerve, particularly the orbicular portion, is usually soon implicated, but that the picture of unilateral bulbar paralysis of the lower nerves is not seen in this disease. On the other hand, in leprosy the lower extremities and the hands are usually affected much earlier than in syringomyelia and—except for trophic disturbances in the feet and legs—according to Laehr the reflexes of the sole of the foot are almost invariably absent, while in syringomyelia they are usually intact. In the differential diagnosis of obscure cases the entire clinical picture and the development of the disease, as well as the finding of the bacillus of leprosy, will be absolutely decisive. *Non-leprous polyneuritis* may occasion decided differentio-diagnostic perplexity, particularly when it runs its course with arteriosclerotic changes, because in this affection also there are dissociated sensory paralyses.

Some time ago I saw a patient, aged 68, who five and a half years previously had fallen from a ladder and sustained an injury of the cervical vertebral column and in whom six months later the following symptoms appeared: Sensations of cold with hyperesthesia in the hands and feet; two years later a difficulty in lifting the feet appeared, subsequently a weakness of the hands and inability to perform finer movements. Gradually the muscles of the hands atrophied, they showed cyanosis, and *vesicles formed* upon them as well as upon the feet.

Objectively the following was noted: Trophic disturbances in the skin of the hands and toes and changes in the nails; motor symptoms; weakness of the interossei, of the peronei and of the tibialis anticus; sensory symptoms; dissociated sensory paralysis in the arms and legs. There were no bulbar symptoms; there was no deviation of the vertebral column, no bladder disturbance, no pain upon pressure over the muscles and nerves, no demonstrable alcoholism nor arteriosclerosis. On account of the dissociated paralysis, the possibility and the probability of a rare combination of cervical and lumbar syringomyelia was considered, particularly as there had been trauma in the region of the medulla spinalis. The autopsy, however, showed none to exist; unfortunately the peripheral nerves could not be examined, but these must unquestionably have been changed since DeR were observed in the muscles of the hand and foot.

Syringomyelia is contraindicated here by the distribution of the distal changes, even of the partial sensory paralysis, to all of the extremities, by the absence of scoliosis, by the normal tonicity of the bladder, and by motor and sensory disease of the leg and foot.

Now and then paralysis of the *brachial plexus,* particularly if bilateral,

may obscure the differential diagnosis, for this condition also produces muscular atrophy with sensory disturbances. But these disturbances usually set in rapidly, are not progressive but either stationary or regressive, they probably never assume the form of distinct dissociated paralysis of segmental type, and they usually permit the recognition of their cause.

Among the *cerebral diseases* which come into question is hemiparesis combined with hemiatrophy, which form a rare combination; in this, however, there are no signs of a spinal semi-lesion nor of partial sensory paralysis. Besides, they usually develop suddenly and are regressive, except for the characteristic implication of consciousness, of speech, and of certain bulbar nerves, above all the centrally paralyzed hypoglossal and facial nerves which are usually simultaneously affected, while atrophy of the tongue does not appear.

If, among *other affections*, scleroderma appears, particularly in the form of *sclerodactylia* and Raynaud's disease, the symptoms of syringomyelia and syringobulbia must be carefully weighed and considered. The same is true of *arthritis deformans* in the upper extremities. In this disease we are preserved from error by the circumstance that more or less severe pain usually appears upon pressure on the diseased joints as well as also in moving them. I have already spoken of *acromegalia*. This disease, with its unmistakable features, particularly the changes in the skull and chin, the enlargement of the ears, the tonsils and the hypophysis, bears no resemblance to syringomyelia. The latter affection leads, if such a term be permissible, only to pseudo-acromegalic conditions, to simple pachyacria.

Finally, as Schlesinger quite properly emphasizes, *arterio-sclerotic changes* in the arteries of the extremities may give rise to symptoms which resemble the sequelæ of syringomyelia—paresthesia, trophic disturbances of the skin, necrosis of varying degree, even muscular atrophy—and the majority of these are probably localized in the lower extremities but may occasionally implicate the arms and hands. There are no dissociated sensory disturbances in the extremities, on the trunk, or on the face; disturbance of the circulation in the atheromatous vessels is readily recognized; when the lower extremities are implicated, a peculiar intermittent limping sometimes develops.

When the diagnosis of syringomyelia with or without syringobulbia has thus been made, either with certainty or a certain degree of probability, we may go a step farther and try to ascertain whether it is the ordinary anatomical form of the disease with predominant processes of destruction and comparatively slight glia proliferation or actual tumor formation, *extensive gliomatosis*. The latter runs a more rapid course than the former, having a duration of about two years; in consequence of increased pressure, symptoms of transverse lesions, in the form of rapid paresis and paralysis of the legs appear early and rapidly, not rarely in the form of semi-lesions, also sudden and extensive muscular atrophy if the motor cells are compressed by swelling. There is severe pain of a radiating as well as local character, and stiffness, sometimes even distinct swelling, of the cervical vertebral region. The bulbar symptoms develop suddenly and run a rapid course. Finally, there may be marked variations in the symptoms as in ordinary syringomyelia.

In contrast with other extensive and multiple extra-medullary tumors, distinctly limited dissociated sensory paralyses are easily differentiated.

TREATMENT

The more minutely and the earlier a positive diagnosis is made the better for the patient: For *diagnosis is the mother of treatment.*

Although we cannot absolutely cure syringomyelia and syringobulbia with our present remedies, nevertheless, if the disease is recognized early the patient can be greatly benefited. By careful treatment at the onset, good nursing, and the choice of another occupation for the patient we can at least prevent early invalidism. And by explaining to him the nature of some of his disturbances, we may prevent trauma such as blows, burns, etc.

Such lesions as occur will be treated on the basis of the underlying affection, by operation or whatever other means seem necessary for the disease of the joints, the bones, or the skin. When there is *paralysis and muscular atrophy,* we must proceed as in other spinal diseases; in addition to much rest, we may try moderate exercise and the cautious use of warmth (not hot applications) and electricity. Warmth and passive movements are beneficial for the *tension,* contractures, and stiffness in the joints. When there is edema and swelling of the joints, massage may be resorted to. Surgical and dermatological measures, above all physical therapy, are chiefly indicated. *Internal remedies* are in order when the patients suffer from pain; primarily the well-known antineuralgics, including the salicyl preparations, especially aspirin. If syphilis is suspected, antisyphilitic treatment must be instituted.

As they arise, other individual symptoms are treated with the remedies usually employed in spinal and central nervous affections; to these I cannot here refer.

A more detailed discussion of this subject will be found in the second edition of Hermann Schlesinger's monograph "Syringomyelia." If further information is desired, the reader may consult 1,175 articles bearing upon this disease which have appeared within the last twenty years, and which form the basis of Schlesinger's work.

HEREDITARY ATAXIA (FRIEDREICH'S DISEASE)

By H. LÜTHJE, Erlangen

There are quite a number of diseases of the central nervous system in which we must accept a congenital hypoplasia of definite portions of the brain and spinal cord as the main cause, or in a part of the cases an inferiority of some special fiber systems. According to our present experience we must, at this time, here include the group of hereditary or family ataxias.

Friedreich was the first to recognize hereditary ataxia as a special disease in a number of typical cases—2 years after the typical description of tabes dorsalis by Duchenne—and of them he gave a classic description in 1861. Following Brousse the affection is accordingly designated as Friedreich's disease.

The fundamental symptoms of this disease are progressive ataxia, which proceeds from the legs to the trunk thence to the arms, but the loss of the patella tendon reflex, sensory and sphincter disturbances are lacking. According to Friedreich's investigation, but especially from Fr. Schultze's researches, a constant gray degeneration of the posterior columns was found as the anatomical lesion. The casuistics of the following years furnished further clinical and anatomical researches (especially those of Friedreich and Schultze), so that the symptomatology and the pathological anatomy of the disease received further development.

However, reports of cases appeared which closely resembled Friedreich's disease, from their family appearance as well as from the prominence of the progressive ataxia, but differing in other essential details, so that they were quite properly regarded as special varieties of the affection. Such were especially the cases reported by Nonne as hereditary ataxia, in which the anatomical lesion proved to be a congenital hypoplasia of the cerebellum. These cases as well as others that were examined by P. Marie led the latter author to include them as an especial pathologic condition, as *hérédo-ataxie cérébelleuse.* The chief symptoms of this disease were progressive ataxia and its family appearance. While in Friedreich's disease the patella tendon reflex was abolished in *hérédo-ataxie* it was retained, often even actively increased. In addition to this there were also signs of absence of function on the part of the optic and oculo-motor nerves. The pathologic lesion proved to be changes of the cerebellum, but above all, congenital hypoplasia.

Subsequently other cases were reported which were regarded as transitional ones; but there are also observations of maladies occurring in several members of the same family in which in some the character of the affection resembled throughout that of Friedreich's ataxia, while in others Marie's type was present. It was also noted that in one and the same patient the symptoms

varied in the course of the disease, for in some the patella tendon reflex was at first retained and later was abolished (Senator). Anatomically, transitional cases were also noted, and in some besides hypoplasia of the spinal cord the same condition was also present in the cerebellum.

This brief historic retrospect appears to be necessary to justify the succeeding description. Our present clinical and anatomical experience permits the view that the different varieties of hereditary ataxia are nosologically connected, that Friedreich's disease and Marie's *hérédo-ataxia cérébelleuse* and the transitional forms, clinically and anatomically, present one form of disease which is characterized by the family appearance, progressive ataxia, and in which other symptoms may present the greatest variety, according to the distribution of the congenital hypoplasia and the intensity and extent of the columnar sclerosis. The greatest number of authors incline to this opinion of the nosologic unity of these forms of hereditary ataxia (Schultze, Edinger, Londe, Seiffer, Bing and others) for only in this manner does the manifold and varying picture of the casuistic and anatomical observations become comprehensible.

CLINICAL COURSE

The disease shows a family appearance, i. e., several members of the same family are attacked; it may also be observed in succeeding generations, or skipping one generation it may appear in the preceding and the succeeding one. Now and then sporadic cases have been reported. In the ascendence there is always a neuropathic predisposition or degeneracy of some sort. Both sexes are equally affected.

The onset of the disease occurs during youth (between the 10th and 20th years). However the limits are by no means so fixed as was formerly assumed. Sometimes the symptoms appear in early childhood (between the 3d–4th years), while in other cases the symptoms arise after the 30th year.

The first symptom is commonly great lassitude and a languid sensation in the legs. Walking becomes difficult without it being possible to discover the exact cause. Soon the signs of a disturbance of coördination develop in the muscles of the lower extremities. The character of this ataxia is static, i. e., the limbs cannot be fixed in the desired position. Associated with this, in most cases, there are signs of a locomotor ataxia with its associated peculiarities: improper choice of the muscles to produce a desired effect, immoderate use of power and persistence of the innervation impulse after the maximal contraction has been obtained. Slowly and gradually the incoördination attacks the higher portions of the musculature, namely, the muscles of the trunk and the upper extremities, perhaps also those of the head and of the organs of speech. Now only does the cerebellar character of the ataxia become distinct. There is a swaying, waddling gait, resembling that of a person under the influence of alcohol. In many, not in all, cases the disturbance in coördination is increased by the absence of control by the eyes. Walking finally becomes so helpless and dangerous that the patient cannot get along unassisted; even this ceases and the patient is either compelled to seek the rolling chair or is confined to bed. While at first it is still possible for him to sit up, this power also is gradually lost on account of the ataxia of the muscles of the

trunk. Thus these patients, in the final stage of the disease, present the picture of complete helplessness: they are not even able to take food without assistance; they must be fed like children. It takes years until this pitiable stage is reached, but the condition is all the more unbearable on account of the complete absence of mental disturbance for disturbance of the intellect is exceedingly rare.

The degree of ataxia, the sequence and the rapidity with which the different members are implicated, are in the main as stated. But variations are noted corresponding to the lack of uniformity in the propagation of the anatomical process.

In comparison with the disturbance in coördination all objective and even subjective symptoms are decidedly secondary. The *patella tendon reflexes* are abolished or they disappear in the course of the disease. Only in some few cases are the reflexes retained or even increased and these are especially the cases which were formerly designated as *Marie's disease.* This variation in the behavior of the reflexes is explained from the progressive extension and from the topographical arrangement of the anatomical lesion; the Achilles tendon reflexes, in the majority of cases, are lost. In some patients *choreiform movements* have been observed but this is by no means frequent. Often the peculiar form of ataxia may have led to the belief that choreiform movements were present.

Very often there are *alterations of speech*; it becomes lalling, slow (bradylalia), uncertain and now and then shows an explosive character. There can hardly be a doubt that this disturbance of speech is due to incoördination of the muscles of articulation and accordingly this symptom is a part of the general ataxia. The same is true of *nystagmus* which is a common symptom. It is not present in every case and usually presents a horizontal character, but at times a circular nystagmus has been reported. The same importance must not be attached to this symptom as formerly, for movements resembling nystagmus are also noted in normal persons with a rapid fixation end-position of the bulbi (Schultze).

As a rule Babinski's *sole reflex* is present, and according to the opinion of some authors (Cestan) the *permanent dorsal flexion* of the great toe, so frequently present, is due to this. Another anomaly of position, which is very common, is the gradual formation of pes equinus; this condition, as well as *kyphoscoliosis of the vertebral column* which is often noted, are not quite clear as to pathogenesis.

True *pareses* are rare; they do occur and as a rule are complicated by antagonistic contractures. *Absence of sensory phenomena* is the rule. There are, however, exceptions especially in the late stages of the disease. Then there are phenomena relating to deep as well as to superficial sensation.

The absence of *sphincter implication, trophic* and *vasomotor symptoms* is the rule; but there are occasional exceptions.

On the part of the *cranial nerves* the *optic* and *oculomotor* are the ones that are principally involved embracing optic atrophy, limitation of the field of vision, especially of the color field, sometimes rigidity to light, paralyses of the ocular muscles. The symptoms relating to the optic and oculomotor nerves particularly belong to the affection named by Marie *hérédo-ataxie cérébelleuse.* It has been shown in this particular also how limits are oblit-

erated, for cases of Friedreich's disease are reported with abolished patella reflex and symptoms on the part of the optic and oculomotor nerves and on the other hand cases of Marie's disease with increased patella reflex in which symptoms on the part of the nerves of special senses are lacking.

The *mental faculties* are intact but occasionally anomalies are observed particularly in the later stages of the disease. Often a conspicuous euphoria has been noted.

This then, in the main, represents the essential features and the course of hereditary ataxia. In a clinical article it is impossible to describe all the varieties and complications minutely; besides this would serve no useful purpose. The casuistics of the last decades show an increasing number of symptoms which complicate and obliterate the actual picture of the disease. This great number of secondary symptoms is by no means so remarkable as we shall see later on. As an anatomical starting-point we have a congenital deficiency of some areas of the central nervous system in which secondarily sclerotic processes develop. These congenital defects are not always confined to the same fiber systems and, therefore, in the special case it must depend upon the duration of the malady, upon the greater or lesser tendency to secondary sclerosis, and upon its topographical arrangement as to what clinical pictures the symptoms will present. We have reason to assume that in some cases there is complete hypoplasia or at least a functional lack of development of the central nervous system, and that in such instances the clinical picture broadens and would increase in complexity, depending upon the duration of the disease; provided intercurrent diseases would not terminate the affection by causing an early death. Only in this manner is it possible to comprehend the continuous series of reports of cases of the so-called pure types and those complicated by numerous and manifold symptoms. This is also true of the necropsy reports in which there have been found variations from an almost exclusive degeneration of the posterior columns to cases with extensive degeneration of the entire transverse section of the cord, lesions in the cerebellum, in the mid-brain and even in the cerebrum.

Nevertheless we must adhere to the clinical picture first described by Friedreich [1] and consider all varieties and differences from the standpoint of this sharply circumscribed symptom-complex.

PATHOLOGIC ANATOMY AND HISTOLOGY

It is advisable first to review the autopsy findings. The most common and constant lesion is a gray degeneration of the posterior columns. This fact was sharply emphasized by Friedreich and is prominent in the explicit report of the year 1862 "Degenerative atrophy of the posterior spinal columns." But Friedreich also recognized an occasional involvement of the lateral columns, beside atrophy of the posterior roots and a more or less well developed chronic leptomeningitis; he regarded the latter as the starting-point of the "chronic inflammatory" process.

[1] It appears quite proper to retain the name "Friedreich's disease" for hereditary ataxia. The forms later described by Marie also belong to this affection; they present only a variety of Friedreich's disease. If desirable we may refer to Marie's type and thus give Marie the credit for the development of our knowledge of hereditary ataxia.

It was, however, Fr. Schultze who broadened and developed our knowledge of the anatomic changes. He showed that Clarke's columns, the cerebellar lateral column tracts, the pyramidal lateral column and anterior column tracts may be degenerated. He pointed out that possibly a hypoplasia of the spinal cord and of the medulla oblongata, which might be considered as an arrest of development, were the origin and final cause of Friedreich's disease. The following years furnished a number of necropsy reports which we need not consider in detail. The discussion between Schultze and Senator may be mentioned as a historic factor, for Senator assumed that a congenital hypoplasia of the cerebellum was the anatomical lesion of Friedreich's ataxia. The dispute may be regarded as closed, for later autopsy reports proved that both authors were correct.

The following lesions have been found in the post mortem examinations:

In the *spinal cord* there is a constant gray degeneration of the posterior columns, principally in Goll's, less constant in Burdach's columns, being more or less extensive both in the transverse section and in the longitudinal axes, even up to the nuclei of the posterior columns. At one time the lumbar cord, at another, the dorsal or cervical areas are implicated to a greater extent.

The *pyramidal lateral column tracts*, to a greater or less extent, are frequently found to be sclerosed. Sometimes only the areas adjacent to the posterior horns, at other times the border portions at the junction of the anterior and posterior horns, in still other instances large areas of the lateral columns are involved. Occasionally there are lesions throughout the entire length of the cord, sometimes even up to the pons; at times only in isolated portions in the cervical, dorsal, or lumbar regions. Much less constant are the lesions of the non-decussated pyramidal anterior columns from the decussation downward. Here the extent of the degenerated portion varies greatly in the longitudinal axis of the cord. In the majority of cases the cerebellar lateral column tracts and Gowers's bundle are more or less involved, the latter from the lumbar cord up to the olives. Lesions of Clarke's columns, of the posterior roots and occasionally also of the posterior and anterior horns are almost as constant as in the posterior columns. Here, also, there is the greatest variation in the degree and intensity as well as extent of the anatomic process.

If we add to this the occasional degeneration of the peripheral nerves and of the spinal ganglia, the changes in the pia mater (chronic leptomeningitis) and of the central canal (occlusion, division, etc.) we may understand how exceedingly manifold is the anatomic picture and how it is hardly possible to speak of a disease of only special fiber systems.

One important finding has not been sufficiently dwelt upon: the hypoplasia of the cord or of the cerebellum or of both together, found in so many cases. There are several reports of a hypoplasia of the entire central nervous system. In these, minute reductions in size are not considered but marked ones which would be obvious at first glance. Hypoplasia of the cord had already been pointed out by Schultze, but Nonne was the first to call attention to a hypoplasia of the cerebellum as the anatomical cause. While it was first believed that hypoplasia of the cerebellum produced Marie's *hérédo-ataxie cérébelleuse* and hypoplasia of the cord and medulla gave rise to Friedreich's type, subsequent investigation revealed that the relations were not so simple: clin-

ically the symptoms referable to *hérédo-ataxie* were noted without atrophy of the cerebellum being present post mortem, and inversely cerebellar atrophy was observed without the clinical picture corresponding to the Marie type.

In addition to this apparent contradiction is the fact that in some cases the autopsy revealed no decrease, neither in the cerebellum nor in the cord, so that the entire pathogenetic importance of congenital hypoplasia, to which Friedreich and Schultze attached great importance and which is probably recognized by all authorities at this time, became very questionable. But quite unjustly so! Although in a few isolated cases hypoplasia has been missed macroscopically, we must not forget that there are degrees of hypoplasia which we are unable to recognize in the usual gross manner in which our examinations are made. At all events, the few, exceptional cases, in which hypoplasia has been missed, are calculated to overthrow our views regarding the pathogenetic importance only when they have been proved by positive means, to be such exceptional instances.

We note here, as in the clinical picture, a great variation in the post mortem findings; but also as in the clinical picture a definite symptom-complex is found to dominate. So again in the autopsy reports we note a definite group of changes recurring with great constancy. These almost constant lesions are: *hypoplasia, degeneration of the posterior columns, a portion of the pyramidal tract, the cerebellar lateral column tract,* and *Clarke's columns.*

The regularity of these lesions led a number of authors to the view that in hereditary ataxia we were dealing with a "*combined system disease,*" i. e., according to the definition of Kahler and Pick, the simultaneous affection of several fiber systems due to a common cause. This view was held for some time. Only recently, from the analysis of a large number of autopsy reports, have our opinions undergone a change. In part, this is the consequence of the knowledge that we cannot regard the posterior columns, the pyramidal lateral column tracts, etc., as functional substantive fiber systems, as was done formerly. The following considerations will explain this more in detail.[1] According to our present views, the posterior columns serve to transmit the sensation of position and of motion (sensibility of the deeper areas). Most authors combine the property of coördination with these "sensation fibers." It has, however, been determined by numerous investigations, that a large part of Goll's and Burdach's columns pass through the posterior column nuclei directly to the cerebellum, but not to the cerebrum. Therefore these fibers, in the true sense of the word, cannot produce "sensations" (for this presupposes an activity of the cerebrum). Another portion, however, passes to the cerebrum and arouses the corresponding conceptions. Thus we may explain why in some cases of "degeneration of the posterior columns" we have a high graded disturbance of the sense of position and of motion without ataxia (in certain forms of tabes) while in the pure forms of Friedreich's ataxia there are severe alterations of coördination with intact deep sensation. We must, therefore, differentiate at least two fiber systems in the posterior columns: a cerebellipetal fiber system, in which destruction produces severe ataxia, and another system which passes to the cerebrum, a lesion of which gives rise to anesthesia of the deeper parts. Quite similar is the course of

[1] I follow Stcherbek's descriptions.

the centripetal tracts of the skin (lateral column tracts and Gowers's bundle) in part to the cerebrum (sensory perceptions), in part to the cerebellum (coördinating fibers).

The "cerebellar" tract of the posterior columns functionally corresponds to the cerebellar lateral column tract, which is interrupted by Clarke's column as the latter is interrupted by the posterior column nuclei. All these cerebellar tracts (cerebellar bundle of the posterior columns, the cerebellar lateral columns, Clarke's columns and parts of Gowers's bundle) degenerate in Friedreich's ataxia. Degeneration also appears in the centripetal tracts of the brain-stem and occasionally in the centrifugal cerebellar tracts (anterior column lateral bundle and the intermediate bundle of the pyramidal lateral column tract, which resembles degeneration of the pyramidal tract itself) as well as in the cerebellum. Therefore in Friedreich's disease we are not concerned with a combined system disease but with a "pure system disease" of the centripetal and centrifugal cerebellar tracts.

These views of Stcherbek are very enticing; but we must not forget that degeneration often proceeds far beyond this system and in consonance gives rise to other clinical symptoms (sensory and trophic lesions, pareses, etc.). Nevertheless we must admit that Stcherbak's explanation of the pathologic condition may be correct for the uncomplicated cases of Friedreich's disease. But such uncomplicated cases are very rare and the longer the disease lasts the more complicated the condition. Correspondingly it would be far better to drop these diagrammatic limits: in Friedreich's disease we are confronted neither with a combined system disease nor with a pure system affection but we are dealing with a progressive malady which is due to an uncontrollable degeneration in the cord and in the brain. The anatomical process conforms to a certain regularity of the sequence in which the systems are attacked, so that clinically a very characteristic symptom-complex results which we designate as hereditary ataxia. But this timely and topographic regularity is by no means constant.

We will hardly have an opportunity of observing autopsies, in which, corresponding to the view of the uninterrupted, progressive nature of the anatomical process larger areas of degeneration will be found than have been noted up to the present time. For the limit of the anatomical lesions is the limit of the capability of life for the possessor of these lesions.

This almost standard, timely sequence, in which degeneration appears and its limitation by the lethal consequences of the somatic condition, have probably led to the opinion that the condition was one of combined system disease. But this view cannot be justified after we have learned to recognize that the degeneration, neither in the transverse section of the spinal cord nor in its longitudinal axis and even beyond the cord, is halted by any fiber system.

We must now add a few remarks in regard to the histologic processes. These details we primarily owe to Schultze. There is always a secondary glia proliferation in areas in which there has been primary atrophy or complete absence of the nerve fibers. In addition there is also occasionally perivascular proliferation of the connective tissue with thickening of the walls of the vessels. This view of the secondary glia proliferation has not been undisputed and especially Déjérine and Letulle, from their own researches and their histologic findings, have expressed the opinion that the sclerosis of Friedreich's

ataxia was of a special nature. According to these authors there is a primary proliferation of the neuroglia tissue, a true gliosis, with secondary degeneration of the specific nerve elements. They arrived at the opinion that there was a special variety of sclerosis, from the peculiar course of the proliferating glia fibers in the transverse section of the cord: most all of them did not run vertically but in the transverse plane of the spinal cord and at their terminations revealed peculiar rolls (*tourbillons*). Weigert soon after rejected this opinion, for he showed that the nature of the sclerosis in Friedreich's disease differed in no way from that of ordinary tabes, of ascending and descending secondary degeneration, of multiple sclerosis, etc. Bing rendered it probable that the radiating arrangement of the glia proliferation develops only during a period in which the glia bundle still has a horizontal arrangement; the glia proliferation therefore "retains the topographical characteristics of the youthful mother tissue." The "*tourbillons*" observed by Déjérine are to be regarded as secondary contracting processes.

The ganglion cells of Clarke's columns are either atrophied or have disappeared in great part. In the degenerated areas the walls of the vessels are thickened. The lesions found in the anterior and posterior horns consist mainly of atrophy or degeneration of the ganglion cells. In the peripheral, sensory fibers a predominance of the non-medullated (embryonal?) fibers has been found.

In the cases in which the autopsy showed a hypoplasia of the cerebellum important histologic changes were not constant. Nonne in his classical cases missed histologic changes in the cerebellum. Fraser and Menzel observed a decrease of Purkinje's cells in the gray substance of the cerebellum; no sclerotic processes were ever present.

We now approach the interesting question in how far the clinical symptoms may be explained by the anatomical lesions? Only a partial answer can be given, for no disease of the central nervous system appears to be in greater contrast, regarding our views of the functions of the fiber systems in the cord, than Friedreich's ataxia: there is degeneration of the pyramidal tracts —and yet no paresis! there is degeneration of the posterior columns as in tabes—and yet no sensory or sphincter disturbance. If we consider further, that in "combined column sclerosis" with very similar areas of degeneration anatomically, an entirely different clinical picture may arise, these contradictions appear to indicate that within these coarse fiber systems there must be finer differences of a functional and systemic nature, in other words, that we cannot conceive as substantive systems the pyramidal tracts, or Goll's and Burdach's columns. I mention this only so that an answer can be given to a question that must have arisen: if the pyramidal tracts are the motor tracts why do pareses not appear when they are degenerated as in Friedreich's disease, or why are sphincter and sensory phenomena absent in spite of the fact that the posterior columns are so constantly implicated to a considerable extent? The most plausible explanation is perhaps the one given by Stcherbek: degeneration of the pyramidal tracts is only simulated by degeneration of the cerebellifugal tracts which run within the pyramids (intermediary bundle). And in the posterior columns only those fiber contingents are degenerated which pass to the cerebellum after interruption by Goll's and Burdach's nuclei; these serve the cerebellum for purposes of coördination, while the tracts, which

enter the cortex of the cerebrum and give rise to sensory impulses remain intact. Only when in the later stages true pareses appear, with sensory and sphincter disturbance, may we assume that the corresponding motor tracts, as well as the posterior and lateral columns, are affected.

We may, therefore, explain the incoördination of Friedreich's disease by the assumption of an elective, partial degeneration of the posterior columns, of the pyramidal tracts, the cerebellar lateral column tract and Clarke's columns, as well as by an affection of the cerebellum itself. Spastic conditions which are sometimes present (there always is dorsal flexion of the toe!) as well as vasomotor disturbances which are noted now and then, find their anatomic explanation in a degeneration of the reflex inhibitive and vasomotor fibers which are assumed to lie within the pyramid contingent. That the patella reflex is abolished in the majority of cases is easily understood by the fact that the degeneration of the posterior columns is always most intense in the lumbar cord, in which its reflex is situated. Whether the speech alteration is to be explained, as Stcherbek believes, by a degeneration of the cerebellar tracts in the crus cerebri, the choreiform movements by disease of the centrifugal tracts of the cerebellum, cannot be decided at the present time. The centers that are here involved have not as yet been sufficiently studied. The same theories serve to explain the nystagmus. Intention tremor, which is often mentioned, requires no special anatomical explanation; for we are not dealing with an oscillating tremor clonus but with a simple ataxic disturbance (Bing). Lancinating pain, which is observed now and then is due to a root irritation from chronic leptomeningitis which is often present.

PATHOGENESIS

As a well founded view we may assume that hereditary ataxia is an agenesis of the spinal cord, of the medulla oblongata and in many cases of the cerebellum, perhaps also of other portions of the central nervous system. Schultze and Friedreich had already pointed out the fundamental importance of hypoplasia of the cord and medulla. From this fact hereditary ataxia approaches a large group of hereditary, or family diseases which are due to similar congenital defects, the clinical picture being dependent upon the seat and extent of these anomalies. The intimate relation of these diseases was only recently emphasized by Jendrássik.

As hereditary ataxia almost always develops relatively late in youth we may conclude further that the anatomical changes, especially the degeneration, develop after birth in the congenitally hypoplastic sections of the central nervous system. That these hypoplasias are not the result of secondary or inactivity atrophy is proved by the marked degree of the hypoplasia—for example even where there has been an absence of function in certain tracts after amputations performed decades previously there is not an atrophy which at all approaches this hypoplasia. Some cases reported by Rennie are, however, decisive: here 18 months after the appearance of the first clinical symptoms an autopsy was held. This revealed a high graded hypoplasia of the spinal cord while the areas of degeneration were not extensive; this could not have been a case of secondary atrophy. Records of this kind are of the greatest importance in regard to the question under discussion. In connection with

this is the frequent simultaneous occurrence of other defects of development (obliteration or a double central canal, occurrence of non-medullated embryonal nerve fibers). But now the question arises why does degeneration take place in these organs that have a tendency to hypoplastic conditions?

I believe, that we must agree with Edinger and his pupil Bing, that the appearance of these degenerations may be accurately explained by Edinger's compensation theory. I may be permitted to digress here and consider somewhat more in detail Edinger's theory, all the more so, as according to Bing hereditary ataxia is a typical example for the elucidation of the compensation theory.

The theory depends upon the law of Weigert and Roux in regard to the condition of equilibrium of the normal tissue which also has such a large part in the law of productive inflammation. When cells are destroyed anywhere in the organism and the equilibrium of the tissue is disturbed thereby other cells soon take the place of those that have disappeared so as to bring about the normal tension of the tissue. This equilibrium, in which the cells stand to one another, will, however, be disturbed when some cells become feebler in comparison with their neighbors. The adjacent cells, in proportion to this debility, gain the preponderance and this exists until compensation again takes place.

Every function causes a decrease in the power of the cell, as the function of the cells is associated with the use of certain cell products. Under normal conditions this is soon compensated by the presence of new nutritive material and the equilibrium is restored at once. But if for any reason this substitution does not occur, the cell which has been weakened in function is destroyed by the preponderance of power of the neighboring cell. Thus it is explained that a function which uses up more material than can be compensated for, always leads to the destruction of the nerve fiber. In this manner the development of many nervous diseases may be explained "in that insufficient substitution occurs within the ganglion cell. The function produces the symptom picture; the disturbance in compensation renders this possible" (Edinger).

An insufficient compensation of this kind may arise in two possible ways: Either the possibility of substitution, for any reason (which usually remains unknown) is less than under normal conditions. Then a damage in the power of function must appear which does not exceed the average of life. Or compensation is normal, as in healthy individuals, but the function itself is permanently increased beyond the average. Such a condition, a relatively too slight power of compensation, is, perhaps, the cause of occupation neuritis, while an absolute, too feeble substitution caused by the hypoplasia of the organs in question, leads to degenerative conditions in hereditary ataxia even without excessive employment of the function of the nerve tracts in question.

If this theory is correct, the anatomical changes and correspondingly the clinical symptoms in hereditary ataxia must develop to the degree and in the sequence which would correspond to their employment during life.

Let us consider whether such a condition can be recognized in Friedreich's disease.

In this affection we have a spinal cord or cerebellum—or both together—which are characterized by an abnormal, congenital lack of development.

"The claims made upon such a central organ by the exigencies of normal life are surely too great, relatively"; sufficient substitution certainly cannot be brought about. The tracts which are most employed, namely, the centripetal tracts which regulate motion and tonus, are first affected. Clinically there is ataxia and anatomically degeneration of the corresponding peripheral sensory neurons (spinal ganglia, posterior roots, posterior columns). On account of the lack of regulation by the posterior columns, compensation must be brought about by the cerebellar tracts and by those fiber systems which pass to the senso-motor regions of the cortex in the lateral columns (according to Stcherbek, not to the cerebrum but to the cerebellum); there is here in addition to an insufficient capability of compensation an additional vicarious function, so that besides degeneration of the posterior columns the same lesions appear in a portion of the pyramidal columns, the cerebello-lateral column tract, and in Gowers's bundle.

We observe, therefore, rapid consumption of the parts that serve to maintain equilibrium, which are utilized almost continuously by the child learning to walk. On the other hand we observe those tracts which are only exceptionally in function, such as the pain tracts, only rarely attacked, and then late in the course of the disease.[1]

"The compensation theory, therefore, explains satisfactorily"—I am here quoting Edinger literally—"all of the symptoms which depend upon the anatomical lesions with which we are familiar. It is not, however, able to give a satisfactory explanation for nystagmus, spasm, the intention tremor, and the speech disturbance. But this is no reason to reject the theory as unsatisfactory; for up to this time we are not acquainted with the anatomical lesions which produce these disturbances; we do not know where and how they are localized. It is not impossible that a view which explains so much more than the previous ones may subsequently also present reasons for what has thus far remained unknown in regard to its development."

I believe that we really cannot escape the impression that Edinger's compensation theory is calculated to explain in a concrete manner the pathogenesis not only of hereditary ataxia but of quite a number of diseases of the nervous system. We cannot conceal that some things are left unexplained—and Edinger is the first to admit this. Thus we may ask why in hereditary ataxia the tracts of the will, which are first and most frequently employed, they also having a predisposition to hypoplasia, are not attacked or at least only late in the disease and then undergo degeneration. Why do we note in tabes (in this affection the compensation theory has also been invoked to explain the condition) that the pain tracts are involved so early although they are so rarely employed spontaneously? But apart from these difficulties he may still be correct: "In favor of the theory in tabes (as well as in Friedreich's disease) is the fact that it serves to explain the symptoms better than any other theory."

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[1] A case reported by Bezold is very remarkable; in this patient the ataxia developed first in the arms. The man was a merchant and he was required to write very much, so that the nerve tracts employed for this purpose were involved to a greater extent and much earlier than otherwise—quite contrary to the usual condition in which ataxia appears first in the legs (quoted from Bing).

ETIOLOGY

The hereditary character of the affection has already been emphasized; but it must be stated that the term "hereditary" is employed here in the widest sense. Not only direct heredity, from one generation to another, is implied, but also those cases are included which occur in several members of the same generation; we have even noted that Friedreich's disease may occur sporadically. It is more proper, therefore, to speak of "family ataxia," as has been proposed by various authors. But as we have seen that in most cases, perhaps in all, there is a congenital agenesis or hypoplasia of the cord or cerebellum, the etiologic factors must be present in the ascendents; in this sense we speak of hereditary ataxia.

In regard to the etiologic factors in the ascendency various opinions have been expressed. The most undoubted rôle is played by the neuropathic predisposition in the widest sense. Signs of degeneration of the most varied kind are mentioned; Nonne, in the cases reported by him, found that conspicuous asymmetry of the face was noted in several members of the family.

Consanguinity of the parents appears to play a part; at least this has been very frequently mentioned in the cases. In how far alcoholism or syphilis, in the ascendents, play a rôle cannot be determined with certainty; by some authors, alcoholism among the parents, is considered very important.

A remarkable factor, which has as yet not been explained, is furnished by a number of acute infectious diseases. Quite often the first symptoms of the disease have been observed after some acute infection, such as enteric fever, variola, meningitis, measles, scarlatina, diphtheria, acute articular rheumatism, dysentery, pneumonia and rötheln; Allen Starr is even of the opinion that in hereditary ataxia there is less of a congenital lack of development than an affection of the entire nervous system appearing in connection with an infectious disease. It is all the more difficult to estimate the importance of the acute infectious diseases, for, in connection with them very similar clinical pictures are observed which have been described under the name of *acute ataxia.* In the latter affection, however, the patella tendon reflex is exaggerated and it almost always terminates in recovery.

We may assume that the tracts which serve coördination are especially exposed to the toxic action of the acute infections. In favor of this view is the fact, emphasized by Friedreich, that if in the course of hereditary ataxia an infectious disease (enteric fever) develops, the course and the severity of the clinical picture are influenced in an exceedingly unfavorable manner. We must, therefore, consider the etiologic importance of the acute infectious diseases in the manner that their toxic action is readily transmitted to the fiber systems which are hypoplastically predisposed, which are, therefore, the *locus minoris resistentiæ.* Immediately after an acute infection the first symptoms of ataxia may appear.

DIFFERENTIAL DIAGNOSIS

The symptoms of an uncomplicated case are decisive in making a diagnosis: the family appearance, onset in relatively early life, the predominant cerebellar character of a gradually developing ataxia which, as a rule, begins

in the legs, then affects the trunk and upper extremities and the typical speech disturbance. Sensory and sphincter symptoms, as well as pareses are absent in typical cases. If the patella reflex is absent but nystagmus, scoliosis of the vertebral column, and dorsal flexion of the great toe are present (equino varus), we may speak of the spinal type; if on the other hand the patella reflex is present or even increased, as well as symptoms on the part of the optic, or oculo-motor nerves, we designate this the cerebellar type of hereditary ataxia (*hérédo-ataxie cérébelleuse* of Marie). But we have seen that a sharp differentiation is impossible, that transitional cases occur and besides depending upon the stage of the disease, how quite a number of supplementary symptoms may appear. But as soon as the fundamental symptoms, which have been briefly sketched, are present, a diagnosis of hereditary ataxia may always be made.

A special discussion of the differential diagnosis from tabes dorsalis hardly seems necessary. The reflex pupillary rigidity, the almost constant pains of paroxysmal character, the absence of speech disturbance, the typical spinal character of the ataxia, the involvement of the sphincters and of the sensory tracts, etc., readily permit of a differentiation.

In well developed cases disseminated sclerosis will rarely give rise to difficulties in diagnosis. The behavior of the patella and abdominal reflexes, the spastic symptoms, the special nature of the speech disturbance (scanning), the history of apoplectiform attacks, must be investigated in doubtful cases.

Huntingdon's chorea has sometimes been the cause of mistakes and in fact, in well advanced cases of Friedreich's disease that are complicated with marked choreiform movements, it may be quite difficult to make a differential diagnosis from hereditary chorea. In such cases the analysis of the motor disturbance may be exceedingly difficult; in addition to this is the fact that Huntingdon's chorea is preeminently hereditary. Usually with accurate observation it is possible to analyze the movements. In favor of hereditary chorea, in doubtful cases, is the late onset (after the 35th–40th year) and the presence of severe mental symptoms.

In tumors of the cerebellum, the symptoms of pressure on the brain such as headache, vertigo, vomiting, and convulsions, which are rarely absent, will aid in the correct diagnosis.

It is remarkable that in spite of the profound anatomical agreement in some cases that have been described by Strümpell as hereditary or family spastic spinal paralysis and Friedreich's disease they should differ so much clinically, as a rule, and that their differential diagnosis is so easily made. Anatomically, in spastic spinal paralysis, there is degeneration of the pyramidal columns but also of the cerebello-lateral column tracts and of Goll's columns. The clinical symptoms are of an exclusive spastic-paretic character; ataxic symptoms are not present. As this disease is, for the most part, limited to the lower extremities it can rarely be confounded with Friedreich's disease.

"Subacute ataxic paralysis" which has recently been described by several American and English authors may, in its early stages, be mistaken for Friedreich's disease. The disease begins with mild spastic pareses of the lower extremities and ataxic symptoms. The spasms in this stage serve to differentiate the disease from Friedreich's ataxia. In the later stages of this ataxic

paralysis a mistake is hardly possible: a sensory and motor flaccid paralysis develops, with degenerative-atrophic conditions and lost reflexes (Seiffer).

In my experience the differential diagnosis of acute ataxia after infectious diseases and hereditary ataxia may occasionally give rise to great difficulties. I reported 3 children in the same family who during an attack of enteric fever, in a certain stage, developed severe ataxic phenomena in all the muscles of the body, a marked tottering and swaying gait, nystagmus, lalling speech, and choreiform movements. Sensory disturbances were absent, as well as symptoms relating to the sphincters; the patellar reflexes were increased. The affection, therefore, revealed the characters of a family disease with the symptoms of hereditary ataxia. As Friedreich's disease may show its first symptoms in connection with an acute infection it is quite impossible to decide the question at any given stage of the malady. The further course alone is decisive: while acute ataxia almost invariably terminates in recovery, hereditary, Friedreich's ataxia, is a progressive disease, slowly but surely terminating in death.

PROGNOSIS, COURSE AND TREATMENT

The prognosis is exceedingly unfavorable, no recoveries having been reported as yet. This is due to the nature of the anatomical process and to its congenital substratum. Now and then remissions are observed and even transitory improvement. But this is exceedingly rare, so rare that the absence of these remissions may be used as an important differentio-diagnostic factor in contrast to certain forms of multiple sclerosis.

The course, as a rule, is very slow but nevertheless progressive. Cases have been reported in which the affection lasted 30–40 years. Death is due to intercurrent diseases. With extraordinary frequency an acute infectious disease is the terminal stage in the life of these unfortunates.

In regard to **treatment** we are helpless; now and then slight improvement has been reported from the employment of electricity (Eulenburg), from massage and electricity (Zabludowski). Actual, permanent and decided improvement has never been reported.

Frenkel's exercise therapy, which has been employed, is without effect. This is easily understood if we consider Edinger's compensation theory; every function, therefore, especially systematic exercise, must have a deleterious action. Friedreich's disease is rather a disease in which skillful nursing should be employed than one in which medical treatment is to be considered.

I have attempted to give a picture of our present knowledge in regard to Friedreich's disease. I have purposely avoided monographic details as these are not suitable to the character of a clinical article. Those who are more deeply interested in the subject will find an opportunity in the study of the comprehensive literature.

Literature

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SPASTIC SPINAL PARALYSIS, AND HEREDITARY SPASTIC SPINAL PARALYSIS

By E. REDLICH, Vienna

In 1875 Erb described as *spastic spinal paralysis*, and soon afterwards Charcot as *tabes dorsal spasmodique*, a peculiar spinal disease which in their opinion was chiefly characterized by spastic paresis of the lower extremities, its anatomical lesion being considered to be an isolated degeneration of the pyramidal lateral column tracts. Naturally, the views of such prominent authors in the realm of nervous diseases were accepted with absolute confidence; and since that time text-books upon neuropathology have invariably devoted a chapter to spastic spinal paralysis. But objections from various distinguished clinicians were not lacking. The clinical picture described by Erb and Charcot was, in its clear-cut simplicity, easily recognized, but the subsequent course of many cases apparently belonging to this category, particularly the autopsy findings of these, was absolutely different from the suppositions of Erb and Charcot, i. e., not a simple primary degeneration of the pyramidal lateral column tracts was found, but quite unlike lesions, for example, multiple sclerosis, compression myelitis, and syphilitic processes, even some of a cerebral nature. This demonstrated that the clinical picture of spastic spinal paralysis is primarily merely the consequence of an interruption of the motor tracts, due either to cerebral or spinal processes.

A few years ago, Schüle, a pupil of Erb's, expressed himself as convinced of the existence of spastic spinal paralysis, and Erb himself has lately come forth, and, upon the basis of additional necropsy results, has positively demonstrated a form of pure spastic spinal paralysis in the sense postulated by Charcot and himself.

But we must first analyze the facts. Except for the cases occurring in children, in whom, as a rule, the symptom-complex of spastic spinal paralysis is accompanied either by cerebral processes or the so-called form of hereditary spastic spinal paralysis, the clinical picture develops in either sex between the ages of twenty and forty. Little is known of the etiology; sometimes syphilis is said to be the cause, in which case, of course, the so-called syphilitic spastic spinal paralysis is not considered. Oppenheim mentions the puerperal state, acute infectious diseases, trauma and lead poisoning. *Lathyrus poisoning*—after the ingestion of bread prepared from various kinds of lathyrus—has produced symptoms corresponding to those of spastic spinal paralysis. In these cases no anatomical lesions have yet been observed, and in other cases of spastic spinal paralysis we are ignorant of the etiology.

SYMPTOMS

In regard to the clinical condition we must mention first that the course is extremely chronic, the duration being from 10 to 20 years, and even longer; this is one reason why these cases are so difficult to judge, for such a patient rarely remains permanently under the observation of one physician. The symptoms begin gradually in the lower extremities, usually bilaterally, rarely in one leg. After a prolonged walk the patients are conscious of fatigue and stiffness in the legs. Gradually the symptoms become more pronounced, and the patients have a permanent sensation of stiffness in the legs; upon examination there is increased rigidity, passive movement of the legs meets with resistance, but the motor power, certainly in this stage, is good or shows but little deterioration. The gait now becomes spastic, the steps of the patient are shorter and more clumsy. After a long time, perhaps after years, we find a further increase in the symptoms. The patient's power of locomotion becomes decidedly limited. The legs are in a state of extension and slightly adducted at the hips; the contractures can hardly be overcome, the gait being conspicuously spastic, the feet seeming to cling to the floor, and in walking the tip of the foot merely glides over the surface. Now the motor power also is distinctly diminished, but the power of walking, although under difficulties, is usually retained until near the end. The tendon reflexes which were increased from the onset become markedly so, there is patellar and foot clonus, and, according to recent observations, Babinski's sign is present. In his most recent work Erb included this among the classic symptoms of spastic spinal paralysis. On the other hand, we find no marked emaciation of the legs, no fibrillary muscular tremor, and no disturbance of the electric contractility. In the late stages there may be slight spastic conditions with a certain decrease of power in the upper extremities and increased tendon reflexes. In the stage of onset there are occasional reports of pain in the legs, but, otherwise, sensory disturbances are absent, as well as disorders of the bladder and rectum, vasomotor or trophic symptoms.

If a case is to be considered as spastic spinal paralysis it follows that, after a course lasting for years, no other symptoms will have appeared than those just described. The symptoms are limited to spasm, to weakness of the lower extremities, perhaps also of the upper, an increase of the tendon reflexes with clonic phenomena and Babinski's sign. As a matter of fact this is actually true in a considerable number of cases; but in a much larger number the subsequent appearance of new symptoms of another kind reveals an entirely different clinical picture.

PATHOLOGY

What does the autopsy reveal in these clinically pure cases? In his last report Erb described 11 necropsies, which he regarded as proving the existence of spastic spinal paralysis, in which there was isolated primary degeneration of the pyramidal lateral column tracts or in which this formed at least the most predominant change; for example, besides degeneration of the pyramidal lateral column tracts there was slight degeneration of the cerebellar lateral column tracts, or of the posterior columns, or of both. This disease of the

lateral columns extends throughout the entire spinal cord, and with decreasing intensity up to the medulla, beyond which point it does not exist. It is not always strictly limited to the area of the pyramidal lateral column tracts; Erb has recently referred to a degeneration of the *posterior half of the lateral columns*. Among the cases he cited as positively proving the existence of the disease are Bíschoff's cases of two brothers suffering from hereditary spastic spinal paralysis; these will be described later. In a prior case, reported by Strümpell, cerebral symptoms were noted, and the slight atrophy in the ganglion cells of the anterior horns and the nucleus of the hypoglossal nerve which is here mentioned cannot be wholly ignored. Friedmann's case merits consideration because this revealed an endarteritis obliterans of the entire basilary artery, and the implication of the cerebellar lateral column tracts and of Gowers's bundle is not unimportant. But we do not desire to become wearisome by a too minute criticism; the case of Donaggio is pure, the last case described by Strümpell is almost pure. We agree with Erb that the pathological anatomy of diseases of the spinal cord should not be too minutely analyzed, hence cases of *spastic spinal paralysis* such as we have just described may from a *clinical standpoint* be acknowledged as *forms of this disease.* But we must be certain that they are rare cases, the pathology of which is still obscure. Above all, we must remember that the picture of spastic spinal paralysis is still only a syndrome which may be produced by many causes, and that a clinical diagnosis of spastic spinal paralysis is only possible when this picture develops very gradually and presents nothing new after some years' duration.

We will briefly review the affections which may transitorily resemble this disease, and discuss those features which enable us to make a *differential diagnosis.*

DIAGNOSIS

Caries of the vertebral column with *compression of the spinal cord* may for a long time be manifested only by spastic phenomena in the lower extremities, while there is at first little if any pain in the vertebral column. In the course of time, however, symptoms referable to the vertebræ appear, a protuberance forms, sharp radiating pains are felt in the vertebral column, spinal symptoms such as paresthesia, hyperesthesia, and anesthesia are added, finally disturbances of the bladder, even complete incontinence, the paresis in the legs becoming paralysis, etc. In syphilitic spinal paralysis, unlike simple spastic paralysis, at the onset or soon after the appearance of the symptoms that have been described, slight sensory and bladder disturbances are noted. We know that *multiple sclerosis* may at first be manifested only by spastic phenomena in the lower extremities, but in the course of time the picture becomes more distinct, for symptoms attributable to the optic nerve develop, above all, charteristic pallor of the papilla, nystagmus, or paralyses of the muscles of the eye, and subsequently intention tremor, marked paralytic phenomena, or ataxia. Some forms of combined system disease may run their course like spastic paresis, even continuously, while in others we note in addition symptoms referable to the posterior columns—ataxia, or absence of tendon reflexes.

Amyotrophic lateral sclerosis may sometimes begin with spastic paresis

of one leg; more rarely spastic paresis then attacks the other leg before atrophy is observable in the member first affected. Hence, in a few cases, beginning amyotrophic lateral sclerosis may be mistaken for beginning spastic spinal paralysis, while, as a rule, if atrophy appears and progresses it soon makes the differential diagnosis clear. *Chronic myelitis,* which is more readily recognizable clinically than anatomically, may also for some time simulate the picture of spastic spinal paralysis; but its course is, as a rule, much more rapid, and symptoms appear which are unlike those of spastic spinal paralysis, namely, sensory disturbances, affections of the bladder, trophic phenomena, etc. Of other spinal processes which may sometimes cause perplexity, I must mention syringomyelia and tumors of the vertebral column and of the spinal cord, without minutely discussing the differential diagnosis. Finally, we must consider cerebral processes, particularly the bilateral. As these are most likely to occur in children, and the distinguishing factors are also applicable in the differential diagnosis of so-called **hereditary spastic spinal paralysis,** I shall defer the discussion of the differential diagnosis of this form until we describe the latter affection.

We owe the first reported case of this affection to Strümpell, who subsequently described others. Then cases were published by Bernhardt, Tooth, Erb, Hochhaus, Newmark, Raymond, Kühn and others, and recently by Bischoff. Lorrain has minutely described the affection. Of course, these published cases present no uniform and conclusive picture, which to a certain extent is also true of many other hereditary diseases of the nervous system. While the cases occurring in one family may almost completely correspond with one another, the cases published by different clinicians often disclose certain differences from, and transitions into, other related forms of hereditary disease.

As the name indicates, this is a *hereditary* affection. As is evident from the reports of Bernhardt or Kühn, the disease occurs in successive generations; more often it attacks several children in one family, in which the hereditary predisposition shows itself in the ascendency in the presence of other nervous diseases. A case described by Kühn reveals a typical clinical picture, although it occurred alone. Males predominate among the patients. In a series of cases reported by Newmark, Bischoff and Kühn, the disease appeared in early youth, between the tenth and eighteenth year, while in others, for example, those reported by Strümpell and Bernhardt, the symptoms appeared only in adult life. We have no accurate knowledge of the etiology.

The main feature of the clinical picture is invariably the syndrome of spastic spinal paralysis, such as we have minutely described, and which needs no repetition; therefore we observe a gradual stiffening of the legs which increases in the course of years and, with a slight but at first scarcely noticeable decrease in motor power, leads to spastic paresis of the lower extremities and the typical spastic gait, also contractures, an increase of the tendon reflexes with clonic phenomena and Babinski's toe reflex; occasionally pes varus or pes calcaneus has been observed. The functions of the bladder and rectum as well as sensation remain intact; there is neither emaciation of the muscles nor other trophic disturbance. The upper extremities and the cranial nerves are not implicated—we are here speaking of typical cases. The course is extremely chronic, the disease lasting more than twenty years; there is a very

gradual, and scarcely noticeable, aggravation of the symptoms, which may be arrested for years or even improvement occur, the patient's power of locomotion being retained to the last.

And now as regards the deviations from this picture. It is of little importance that Strümpell observed in one case a slight decrease of the temperature sense and Kühn noted the same diminution of the pain sense. In some cases an affection of the cranial nerves has been observed, as in the cases of Bernhardt, in which besides intention tremor there was considerable difficulty in speech and deglutition as well as slight disturbance of the innervation of the muscles of the eye; these cases perhaps account for the opinion, sometimes expressed by French authors, that this affection undergoes a certain transition into multiple sclerosis. One of Strümpell's cases had a slight impairment of speech; and Bischoff has reported slightly defective movements of the muscles of the lips and tongue. The latter cases so far deviated that, in the more advanced stages, there was a certain spastic paretic disturbance in the upper extremities, in the muscles of the trunk, and in the nape of the neck, and in the terminal stages there was even a decrease of tonus with emaciation, as well as psychical alterations quite unlike anything in previous observations—a gradually increasing dementia having appeared.

Necropsy reports were published by only two investigators. In Strümpell's case a degeneration of the pyramidal lateral column tracts was found in the spinal cord, increasing in intensity downward, but also implicating the cerebellar lateral column tract; in Goll's column also there was slight degeneration. Bischoff's case, too, showed anatomical peculiarities. Here there was a conspicuous degeneration of the pyramidal lateral column tract, increasing from the lumbar cord upward, but less marked in the cervical cord, and not perceptible in the medulla, where, however, the degeneration extended somewhat beyond the actual area of the pyramidal lateral column tracts. In the posterior column there was slight degeneration, limited in the lumbar cord to Burdach's column, and in the proximal areas to Goll's column. In the last stages a decrease in the volume of the muscles corresponded with the decided atrophy of the anterior horn cells. Finally, an extreme internal hydrocephalus must be mentioned, the cortex of the brain showing histologically no change.

What has been stated regarding spastic spinal paralysis is mainly true of the **differential diagnosis** of hereditary spastic spinal paralysis, the proof by the history of its hereditary or family appearance being the main condition. One point only needs a somewhat more minute description, and that is the differentiation from so-called **Little's disease**, or *infantile bilateral cerebral paralysis,* the chief symptom of which is spastic paresis of the lower extremities. This is all the more necessary because hereditary spastic spinal paralysis, as we have seen, appears in youth, while, on the other hand, Little's paralysis may be noted in several children of the same family. A prominent feature of Little's disease, in comparison with hereditary spastic spinal paralysis, is the following: The early appearance of the symptoms, these being occasionally recognizable immediately after birth, while in hereditary spastic spinal paralysis the affection is prone to appear about the tenth year or even later, and the demonstration of the well known etiologic factor of infantile cerebral paralysis (premature birth, trauma during

birth, infectious diseases, etc.), while in hereditary spastic spinal paralysis, aside from heredity, no other etiologic factors are known with certainty. In contrast with the majority of cases of hereditary spastic spinal paralysis, in cerebral infantile paralysis we frequently observe epileptic attacks and strabismus, sometimes paralysis of the cranial nerves and even pseudobulbar symptoms, and intellection is often impaired to the point of actual idiocy. That these factors are not absolute proof is shown by the cases of Bischoff (here the concomitant hydrocephalus may also have played a rôle) and Bernhardt. Finally, in hereditary spastic spinal paralysis hereditary syphilis and the spinal diseases attributed to this must be excluded, and this, as a rule, is not difficult.

TREATMENT

Little can be said of the treatment. Therapy is ineffectual in this gradually progressive disease; in the main, it is less a question of treatment than of careful nursing. Mild massage or electricity should be tried, and, of course, all highly irritating methods are to be avoided. Mild hydrotherapy may be beneficial. In the late stages, tenotomy or transplantation of tendons may be considered.

PROGRESSIVE MUSCULAR ATROPHY (DYSTROPHY), PROGRESSIVE SPINAL MUSCULAR ATROPHY AND BULBAR PARALYSIS

By FR. SCHULTZE, Bonn

For a long time the progressive muscular atrophies have been the step-children of clinicians and physicians. Neither surgeons nor internists devoted special attention to them. Thus I remember from 1869–1871, my student years in Berlin and Bonn, that Traube, Frerichs, or Rühle, and among the surgeons, v. Langenbeck and Busch never demonstrated this conspicuous and curious disease. I saw the first case in Friedreich's Clinic in Heidelberg. This has probably been the experience of many other physicians. And yet Duchenne about 1850 had devoted considerable attention to this affection; he gave a thorough description and endeavored to determine its anatomical foundation. Later many other investigators took up this work, in France, Cruveilhier, Charcot and Déjérine, in England, Lockhart Clarke, and in Germany, Friedreich, v. Leyden, Kussmaul, Erb, J. Hoffmann and Strümpell.

The reason of this prolonged negligence of progressive muscular atrophy was probably due to the fact that the pathogenesis and the anatomical nature were obscure, and particularly that treatment was ineffective; for the malady can hardly be overlooked. Only the muscular atrophy, especially in children and women, may be unrecognized on account of the profuse development of fatty tissue. The affection may also be confounded with secondary atrophy after disease of the joints. But we are not now considering any partial muscular atrophy, but purely that variety which progresses from *muscle to muscle, and from one muscle region to another.* That a circumscribed muscle atrophy, which remains isolated, does not arise suddenly but more or less gradually, or in a progressive manner, is obvious; but we are not considering this condition.

1. PROGRESSIVE DYSTROPHY (MYOPATHIE PRIMITIVE, OF THE FRENCH)

After these preliminary remarks I shall discuss the various chief forms of the disease, and first those which have been known for the longest time and in which our present methods of anatomical investigation usually reveal only *changes of the muscle substance itself*, the varieties called by Erb *progressive muscular dystrophy.*

Infantile pseudomuscular hypertrophy was first described. This affection is comparatively common and has been investigated very often, both clin-

ically and anatomically. It is commonly noted in several children of the same parents and may be transmitted through several generations. When completely developed the clinical picture is very characteristic.

The following is the history of a boy, aged 13, who had healthy parents, brothers and sisters; there is no similar disease among his relatives. He never learned to walk or stand properly; but, with the exception of pains in the back, never complained. An increase in the volumen of the calves was early noted without the affected muscles showing a corresponding power. Upon examination an enlargement of the calves is noted at once, which is all the more prominent on account of the thinness of the thighs. The cutaneous fatty tissue is richly developed, so that in the upper extremity and in the shoulder girdle there appears to be a normal configuration. Nevertheless upon careful palpation the trapezei, the deltoidei, the pectorales, the latissimi dorsi, and the serrati antici majores are markedly atrophic and very weak, as is proven by weakened resistance movements. Active raising of the arms, as well as abduction and adduction and rotation in the shoulder joints are not vigorous, while movements of the elbow and wrist joints are much stronger; the muscles of the hands, both in regard to size and power, are normal. Yet prominence of the supinatores longi upon strong flexion of the elbows cannot be noted.

FIG. 184.—INFANTILE MUSCULAR DYSTROPHY. (After Moritz.) Pseudohypertrophy of the Muscles.

It is further conspicuous that the infraspinati are over-sized and like the muscles of the calf are upon palpation coarser than normal. If we attempt to raise the patient by the shoulders, they seem to slip upward ("loose shoulders"), as they cannot be held down by the muscles of the arm which have this function. Active flexion and extension of the thigh is as little possible as abduction, adduction, and rotation, although the corresponding muscles contract somewhat and even become prominent. Nor is extension of the leg possible, while the peronei and the muscles of the calf act quite well. On the other hand the abdominal muscles have no power; only with the aid of the arms and hands is it possible for the patient to rise from the recumbent posture after supporting himself upon the elbows; lateral movements of the trunk are incomplete.

Standing is possible, the sacrolumbales becoming very prominent posteriorly but at the same time the lumbar vertebral column shows marked lordosis; when the patient assumes the sitting posture, kyphosis of this portion of the spinal column is noted. Walking is possible only with the aid of a cane; it is very slow and tedious; the gait is waddling, like the walk of a duck. The slightest push is sufficient to cause the patient to fall. It is impossible for him to sit down slowly; the patient lets himself fall. If he falls too far or upon the floor, he cannot rise unaided.

The muscles supplied by the facial, hypoglossal, fifth, and pneumogastric nerves, as well as the sternocleidomastoids and the ocular muscles, are normal. Nowhere in the naked body, even upon prolonged standing, are there fibrillary contractions. The skin presents a peculiar bluish marbling, especially in the legs and in the region of the buttocks; this is less marked in the arms.

Electric examination shows a decrease or an absence of response to faradism and

galvanism; nowhere is there DeR; mechanical contractility is also either decreased or absent. Sensation for all qualities is everywhere normal; there is no pain. Deglutition, speech and respiration are intact. The vegetative functions and the intelligence are normal. The patella and Achilles tendon reflexes as well as those of the arms are absent; this is also true of the abdominal reflexes. The pupils are normal in reaction.

It is hardly possible to mistake this clinical picture or to fail in recognizing it. There are no symptoms such as appear in cerebral affections or in diseases of the sensory or coördinating portions of the nervous system; all refers to the motor-trophic sphere of the muscles and all symptoms may at once be explained by a disease of the muscle substance; for those sequels are lacking which appear in a primary chronic disease of the peripheral motor neuron extending from the motor spinal ganglion cell to the motor nerve termination in the muscle, namely DeR and fibrillary contractions. In fact upon anatomical examination we find in nearly all cases—I purposely say nearly—that the muscles only are diseased. The large motor ganglion cells of the spinal cord are of normal size and even upon examination, with the most improved methods—as we were able to note in two cases of our own that were investigated by Nissl's method—there is no demonstrable change in structure. Of course we cannot maintain that the most minute chemical and physical properties of the ganglion cell are normal in every respect. But it is astonishing enough that no gross changes, above all no atrophy, is present, such as appears secondarily after amputation of members, or as takes place in young animals when motor peripheral nerves are early removed. It appears, therefore, that the motor innervation, which makes its way from the brain unhindered, as well as the reflex ones which act from the skin and the deeper areas also in an unobstructed manner, are usually sufficient to prevent atrophy and a demonstrable change in structure of the ganglion cells.

The peripheral nerve trunks and the nerve terminations, which could be investigated in a very incomplete manner only, have generally been found unaltered, while the muscle fibers and their intermediary substance reveal severe lesions. Briefly, besides hypertrophy of some fibers with nuclear increase and with very frequent vacuole formation, there are atrophic changes of varying extent and intensity. Whether a hypertrophic stage always precedes the atrophy cannot be definitely answered. Marked increase of the interstitial tissue is also very conspicuous, either in the form of an increase of fatty cells or of connective tissue cells. This produces the increase in size of the different muscles which has been described.

An increase of nuclei may also be found around the vessels, as I was able to convince myself from the autopsy of a boy aged 3 who suffered from this malady. This is probably a secondary process, such as commonly takes place after primary destruction of parenchymatous elements.

Therefore these findings justify us from the anatomical standpoint in assuming that the clinical foundation of the pathologic picture is a *disease of the muscles*, which in some cases is congenital, but in most cases develops early in youth in a family or hereditary manner. For this reason an abnormal predisposition of the muscles has been mentioned. The development of muscles is independent, to the widest extent, of that of the nerve apparatus in embryonal life, so that without spinal cord and without motor ganglion cells muscle fibers may develop. *This abnormal predisposition of*

the muscles may be so marked that a proper development of muscle fibers does not take place, it may be so constituted—similar to the diabetic predisposition—that at first there is a normal muscular system which, sooner or later, under different influences, alters in a progressive pathologic manner and finally almost disappears.

Symptoms.—Returning to the description of the clinical picture of pseudohypertrophy, there are other manifestations which must be described. The first symptoms do not permit a positive diagnosis. The waddling duck-like gait, which occurs comparatively late, may lead to the view that a *congenital bilateral dislocation of the hip* is present.

I remember a case in which not even the combined endeavors of my surgical colleague Schede and myself were sufficient to assure a physician that in a certain case of pseudohypertrophy, which had only involved some of the muscles of the back and pelvis and a few of the leg, that the correct diagnosis was pseudohypertrophy and not a dislocation. At the present time, X-ray examination is an aid which determines dislocation of the hip in a decisive manner. Difficulties may appear in elderly persons in the differentiation from *osteomalacia,* which is so often overlooked. But in this affection pains develop early, both in the bones of the pelvis as well as in the lower ribs and within the vertebral column; these pains are caused by external pressure, walking, and motion. Pain of this kind is foreign to the picture of muscular dystrophy. If it is present, in addition to nervous symptoms of a hysterical nature, it is not limited to the bones and depends upon psychical influences. The late stages of osteomalacia produce certain changes in the bones which do not occur in muscular dystrophy.

Pain of another kind which is localized in the muscle appears in the disease, but it must be regarded as partly due to exertion; this is more liable to occur in the diseased muscles upon slight action than in the normal ones. Another early symptom which was not present in the case whose history I related is that children fall much more frequently in jumping and running, and when executing complicated movements of the trunk and legs. This naturally may be due also to other causes. Moreover there are other nervous diseases such as Friedreich's disease with its disturbance in coördination; Little's disease, the spinal amyotrophies and neurotic atrophies which will be described later on. Then arthritic affections and lesions of the bones of a rachitic and tuberculous nature may be present; all of these must be carefully investigated, for in rare cases they may be associated with muscular dystrophy.

In general, however, certain disturbances in movement are characteristic, these are the following:

At the onset of the disease, perhaps in the brothers or sisters of a child affected with pseudohypertrophy, a sign may be observed which has hardly as yet been described. Such a child, while the symptoms that are to be described are still lacking, finds it impossible to rise from a stooping position unaided. In one of my cases of this kind only the muscles of the calf were somewhat thickened and there was a slight lordosis of the vertebral column, which in the sitting posture changed to a slight kyphosis. Further, the 9 year old child could not rise from the horizontal posture without the aid of the arms, which otherwise, on account of the early weakness of the

abdominal muscles is impossible in the early stages of the affection. In sitting down the child permitted itself to fall upon the seat.

If the atrophy and weakness of the muscles of the trunk and legs has made greater advances we observe those peculiar movements, which they require to assume the erect posture from the horizontal one. The patient first turns upon the side, then assumes the abdominal position, braces himself with the hands, brings the feet into the kneeling position, raises the knees from the floor while extending the legs so that only the hands of the extended arms and the feet of the extended legs are in contact with the floor. Finally, first one hand is braced upon the knee of the same side, then the other one and in this manner the patient climbs up upon himself, the hands reaching higher levels upon the thighs. Naturally these movements are not always performed in precisely the same manner, but they may be modified, one or the other point being omitted or changed according to the degree and extent of the weakness and atrophy of the existing condition.

It must be remembered that a similar condition, in regard to the patient rising from the ground, may be present when there is disease of the vertebræ or some other affection of the muscles, as in a rheumatic inflammation. The disturbance that has been described depends upon the fact that an extension of the trunk upon the pelvis is only possible to an abnormally slight degree, and the bracing of the hands upon the thighs takes the place of the sacrolumbales muscles, that, further, the extension of the pelvis upon the thigh and that of the thigh upon the leg succeeds only with the greatest exertion (especially with weakness of the glutei and of the quadricipites femoris). But there may be difficulty in motion in all of these muscles from inflammatory affections, or disease of the vertebræ may give rise to a similar difficulty in movement of the muscles which are commonly involved in dystrophy.

The differential diagnosis, apart from the more rapid development of the symptoms, will be possible from the fact that painful pressure points over the vertebræ or muscles are present, quite apart from marked changes in the bones; a condition that may be readily recognized by an alteration in shape of the vertebræ or by an X-ray examination.

The ascent of stairs in dystrophy is early rendered difficult from weakness of the glutei maximi; for in this movement power must be exerted by these muscles. The feebleness of the glutei medii is chiefly responsible for the waddling gait.

Often at the beginning of the disease and even in the advanced stage the alterations in the muscles of the arms and shoulders are overlooked, first because no examination is made and second because there appears to be no change. If, however, the power of the individual muscles is tested those of the hand are usually found normal; of those of the forearm the supinator longus upon both sides is atrophic simultaneously with the biceps and brachialis internus. In the upper arm the deltoid and triceps are hypertrophic bilaterally, but weakened, therefore, pseudohypertrophic; bilaterally at the shoulder girdle the serratus anticus, latissimus dorsi, the major and minor pectorales with the exception of their clavicular attachments are affected. All of these muscles are weak and atrophic. The trapezius with the exception of the upper acromial or clavicular portion and the supraspinati reveal the same conditions. On the other hand the infraspinati are commonly volumi-

nous and even in the late stages of the affection are prominent in the midst of the general atrophy although they do not always possess their normal power. These muscle atrophies produce many abnormal positions and displacements of the shoulder which cannot be separately discussed and which are familiar from our knowledge of the pathology of peripheral paralyses.

The conspicuous *symmetry* of these changes is remarkable although slight asymmetry may be present in some of these pseudohypertrophies. The sternocleidomastoid and the muscles of the head are usually not involved; but we shall see that in certain cases they are attacked and may even be the first to reveal the condition.

In the early stages of the disease, therefore, disturbances are present in the upper part of the trunk and in the arms as well as in the lower parts and in the legs. It is also noteworthy that primarily the proximal muscles are implicated and further that in the upper half of the body as well as in the lower half certain muscles are especially involved; in the upper portion the deltoids, the tricipites and particularly the infraspinati; in the lower the sacrolumbales, not infrequently the glutei, nearly always the gastrocnemii and solei and often the tensores fasciæ latæ as well as the sartorii. In other instances other muscles show the condition; now and then many simultaneously.

As the affection advances, as is the ordinary course, the force and size of the muscles first attacked decreases, even those that are hypertrophic; in the muscles of the legs, especially those of the calves, retractions and contractures appear, which are probably due to the fact that the length of the muscle fibers decreases. Walking, and finally standing become impossible; the use of the arms and hands becomes limited for atrophy may also attack the muscles of the forearm and hand in which exceptionally there may also be an early hypertrophy and atrophy.

Finally the *respiratory muscles* may be involved and particularly the *diaphragm* so that dyspnea appears. In some cases the heart appears to be involved; at least in some autopsy reports atrophy of its muscle fibers, and increase of the interstitial substance has been reported. In a recent case reported by Meerwein clinically by the aid of percussion as well as with X-ray examination a decided hypertrophy of the heart could be demonstrated. The many reports of acceleration, slight irritability and irregularity of cardiac action may be explained in many ways, and cannot be definitely referred to atrophy or pseudohypertrophy of the heart muscle.

Quite often the tongue is attacked in the pathologic process; unilateral involvement (hypertrophy) has occasionally been reported. The masseters and the facial muscles may be involved. Usually such patients succumb early although Oppenheim saw a patient aged 58, who suffered since his earliest childhood from pseudohypertrophy. They are often attacked by tuberculosis probably on account of enforced living in closed, infected rooms. Sometimes pneumonia in connection with bronchitis, is the lethal cause; the reason being the insufficiency in respiration due to the muscle involvement that has been described. At all events the disease is a pitiable one which is more easily borne by children than by adults. One of my patients committed suicide by hanging.

Some of the *complications* are noteworthy. First those rare cases associated with *general atrophy of the bones*, such as described by Le Gendre, Fried-

reich, and myself; an atrophy which may be regarded as a coördinate affection. Then the more frequent *anomalies in the formation of the skull and of the bony structure* of the body, such as *hydrocephalus,* asymmetry, clinocephalia; protrusion of the upper jaw as noted by Lorenz; *changes in the thorax* (wasp-waist by Marie), *funnel-shaped chest,* spontaneous fractures and the like. Now and then cerebral symptoms are noted, such as *weakness of mind* and *imbecility*; this condition is, however, very readily simulated to a certain extent, as children with pseudohypertrophy are kept from school.

This briefly is the picture of *pseudohypertrophy* of the muscles of which there may be several *varieties*; these were formerly described as special affections but Erb quite properly combined them under the general term "*progressive muscular dystrophy,*" as the other term "progressive muscular atrophy" is of too general a nature and includes the spinal and neurotic atrophies and does not include the conspicuous symptoms of hypertrophy and pseudohypertrophy.

Thus v. Leyden described a special form of "*hereditary muscular atrophy,*" which is characterized by: first, heredity or family predisposition, second, by weakness appearing primarily in the muscles of the small of the back and legs late in childhood or during puberty, and, third, by the lack of pseudohypertrophy of the calves and only after years are the muscles of the shoulder and of the upper extremity implicated. As, however, some muscles, even in this form of the disease, may show pseudohypertrophy, as further, the ordinary form of pseudohypertrophy may appear in children of the same family, and, finally, heredity and the late infantile occurrence is observed often enough in ordinary pseudohypertrophy, it is hardly correct to maintain a special form of disease; for all other symptoms such as electric contractility of the muscles, fibrillary contractions, the presence of motor-trophic symptoms and the progressive character are quite the same.

In other cases the disease may begin in the muscles of the face as described by Duchenne; later this type was again made prominent by Déjérine and Landouzy, who added a description of the anatomical lesions. In this type the affection also begins in early childhood, as is so common with ordinary pseudohypertrophy of the muscles, so that the name "*infantile form of progressive muscular atrophy*" which was applied to Déjérine's type possesses no differential factor and besides is incorrect, for the malady may arise during advanced life.

The disease in this form invades the sphincter muscles of the eyes and mouth, then the muscles of the forehead and the other muscles supplied by the facial nerve. From atrophy and parallel debility the same picture appears as from bilateral facial paralysis with inability to close the eyes completely so that a space of several millimeters remains. The forehead cannot be wrinkled and remains smooth. The mouth is sometimes held slightly opened and the lower lip droops ("tapir lip"). The movement of the muscles of the mouth is decreased; whistling and showing the teeth is impossible. The muscles employed in laughing cannot be used in the normal manner, the buccinators do not press the cheek strongly enough against the teeth, the face thereby assuming a "mask-like" appearance. The other muscles of the head are intact; those of the shoulder and trunk are involved later quite

in the same manner as in pseudohypertrophy; the infraspinati are not affected; there is the same lordosis of the vertebral column, etc. Pseudohypertrophy of isolated muscles is also observed. The electric contractility, the absence of cerebral, mental, and sensory symptoms, the lack of fibrillary contractions and the condition of the reflexes corresponds to the type of ordinary pseudohypertrophy.

Occasionally, as especially described by J. Hoffmann, at the onset of the disease, beside myopathy of the regions supplied by the facial nerve, some of the bulbar muscles are involved, so that a complete *bulbar type* may appear. Hoffmann saw 2 children, twins, who had, besides atrophy of the muscles supplied by the 7th nerve, *atrophy of the tongue,* degeneration of the soft palate, and in one of these patients also *paralysis of the muscles of deglutition.* With this there were the ordinary changes in the muscles of the trunk and extremities, such as have been described; while the infraspinati and levatores angulæ scapulæ were spared and the muscles of the calf of good size.

More frequent than these varieties or types is a form of progressive dystrophy described by Erb, namely the *juvenile* or *scapulo-humeral form,* while the Duchenne-Déjérine type is called the *facio-scapulo-humeral type.*

In this condition there is less of a hereditary or family predisposition than in the previously mentioned ones and only after childhood, as a rule, from puberty up to the twentieth year does the disease develop. The *muscles of the shoulder girdle* are first affected; most markedly, as in pseudohypertrophy, both pectorales, the trapezii with the exception of the upper portion previously described, the serrati, latissimi dorsi, rhomboidei, while the levatores scapulæ, sternocleidomastoidei and infraspinati are not implicated; the deltoids are often normal or like the infraspinati and the triceps increase in volume. The bicipites become atrophic, while the muscles of the forearm, with the exception of the supinatores longi, are normal. In the lower extremities and in the pelvis the glutei and the quadricipites femoris are attacked while the muscles of the calf are spared for a long time just as the tensores fasciæ latæ and sartorii increase in size. The distal muscles of the extremities are not affected, nor, as a rule, the muscles of the face; later, however, as is sometimes the case with the sternocleidomastoidei, they may be involved.

The posture of the body, the movements, the difficulties in rising and standing are the same as in pseudohypertrophy, depending upon the distribution of the affection. All of the other symptoms are similar, the positive as well as the negative ones. The course is slow, usually progressive, sometimes remaining stationary for a long time or lasting decades.

Finally, it must be mentioned, that, while in all the types described the *distal muscles of the extremities* are not involved until late in the course of the disease or at most simultaneously with the others, J. Hoffmann several years ago reported a few cases, among which, in a man aged 23, with a most probable hereditary predisposition, the malady began in the muscles of the leg and 6 years later the same changes appeared in the muscles of the forearm, while the small muscles of the hands and feet, as well as, during the period of observation, the muscles of the shoulders, upper arm, trunk, and pelvis were not attacked. In the other case, that of a child, in which there was no trace of a hereditary predisposition, the disease developed first

in the leg, so that at first a right-sided, and 4 years later a left-sided, equinovarus developed and finally a right-sided club foot was added. Besides there was weakness of the lower muscles of the back and pelvis with the peculiar manner of rising from the floor which has been described and a slight implication of the muscles of the shoulder and arm. All phenomena, referable to an involvement of the nervous system, were lacking.

In rare cases, therefore, the malady may begin in more distal regions than is ordinarily the case, and occasionally as in a case of Friedreich's which was also seen by Erb and myself, simultaneously with the first symptoms of the disease the muscles of the hands may be attacked; nevertheless up to now positive reports are lacking that the affection had a distal and peripheral onset and distributed itself in ascending, which, *a priori*, is certainly possible. *The disease may, however, begin in any transverse stripped muscle,* except the heart and, as it appears, in the diaphragm, therefore, in the most vital organs. Perhaps according to J. Hoffmann the *external muscles of the eye* may also be attacked (a point not mentioned up to now), so that the picture of *ophthalmoplegia externa* appears.

In such cases just as in very early involvement of the muscles supplied by the facial nerve or in defects of the large pectoral muscles which are often noted, the question may arise whether we are dealing with a defective development of isolated muscles, which is by no means rare. Occasionally these defects are not congenital but appear during infancy and are progressive for some time; they run their course with the corresponding atrophy of their ganglion cell nuclei or may even be produced by this cause. Upon prolonged observation we will generally be able to reach a decision from the fact that the early muscle defects remain stationary and are not associated with decided muscle atrophy. Duchenne, in a case of congenital dystrophy of the muscles of the face, observed no progression even after 30 years.

We note, therefore, that in all of these forms of disease, we are dealing only with varieties of the same affection, a fact that has been determined by careful investigations. The differences in the varieties or types relate to the varying localization of the muscle atrophy and also to its development at different ages. Their unity, apart from the similarity in symptoms, is proven by the fact that the individual types may be distributed in various ways in one and the same family, also that new combinations may arise in the family, and, finally that in one and the same patient another variety may develop upon the one already existing.

The essential point is this, that in the great majority of cases there is a permanent absence of symptoms relating to the nervous apparatus and that this lack of clinical nervous symptoms as far as our present knowledge reaches corresponds to an absence of lesions in the ganglion cells, in the anterior roots, and peripheral nerves. In a small number of cases pain may be present, which, as has been mentioned, may be referred to exertion but which may also be due to irritative conditions (still unknown) within the sensory nervous system.

Now and then *DeR* may be noted in some muscles, in which it cannot be determined, in spite of very careful researches, whether there is a primary nervous disturbance or whether the muscle is entirely or partly " without nerve

control." As the changes in the muscle substance which produce a slowing of contraction to galvanism are still unknown, we cannot reject the view that among these unknown conditions there are some which are independent of nervous influences.

Finally motor *irritative symptoms* are exceptionally noted, in the form of *fibrillary contractions*, such as occur to a much greater extent in primary atrophy of the motor spinal and bulbar ganglion cells. It is not known whether these may not arise without the action of nervous irritative conditions even of a peripheral nature, in the muscle fibers.

A conspicuous tendency of some muscles to *hypertrophy* and *pseudohypertrophy* as well as the localization of the *dystrophy* which usually shows a preference for the proximal muscles and those of the trunk is characteristic. I shall refer, later on, to anatomical lesions in the motor ganglion cells which may appear to be contradictory to this condition.

I have already indicated the differential diagnosis of the affection, especially in the early stages, from *disease of the bones* and congenital dislocation of the hip. As, however, after gross *chronic disease of the joint* marked atrophy appears secondarily, a polyarthritis of this kind may occasionally come into question in differential diagnosis. Upon careful examination the signs of an arthritic affection of this nature can hardly be overlooked; besides there is no indication of pseudohypertrophy or even of true hypertrophy of the muscles. Joint pain, due to pressure or motion, is absent in dystrophy and so a diagnosis can be made in those arthritic cases in which a more or less general atrophy of the muscles of the trunk or extremity is present.

Now and then the question may arise, whether or not a hypertrophy of the muscles has been produced from a primary *venous change* with chronic stasis of the venous blood. This condition has occasionally been observed. However, this circulatory anomaly, with its consecutive cyanosis and venous dilatation, can readily be determined and it is only necessary to know that especially after chronic venous thrombosis muscular hypertrophies may develop.

Of other diseases of the muscles only *polymyositis*, with or without simultaneous dermatitis, may give rise to diagnostic difficulties, for, as Oppenheim-Cassirer and myself have determined, this condition may be succeeded by general atrophy. In this very rare affection there is an acute process accompanied with pain, which is regressive and not progressive as in dystrophy, leaving at most stationary residues not accompanied by hypertrophy. If *dystrophia muscularis* is regarded as a progressive polymyositis, as was done by Friedreich at one time, anatomically we are then concerned with the same condition; the only difference would be in development and the lack of progression. *Neuromyositis*, a very rare affection, begins much more acutely and is accompanied by pressure pain of the peripheral nerves and other sensory irritative symptoms as well as those due to absence of function. The differential discussion from the neurotic and spinal forms of progressive muscular atrophy had better be deferred until these varieties have been described.

Etiology and Pathogenesis.—In regard to the etiology and pathogenesis it must be stated that our knowledge is still very deficient, although a clear insight would be exceedingly important from the standpoint of treatment. The most interesting factor in the etiology is *heredity*, which is observed so

commonly in all varieties of dystrophy. How this affection is transmitted from parents to children, or how the affection appears in children without the same malady being present in the parents is unknown. At all events there are no relations of dystrophy to the infectious diseases affecting the parents, or of the mother during the period of pregnancy. Syphilis has no recognizable influence. Whether intoxications of any kind are active cannot be answered. The influence of alcoholism has been most considered. It can by no means be always detected, either in the ascendants or in the patients. It is remarkable that in some regions the occurrence of hereditary *dystrophy* and of hereditary nervous diseases is much more common than elsewhere. This observation can hardly be due to the personality of the investigator; for example, in the Palatinate and in Heidelberg many of these affections have been reported, although it must be admitted that not everywhere is the same attention devoted to these maladies as in this district. It is also noticeable that the disease occurs frequently in the members of the working classes, especially, as it appears in the population of the country districts. Accurate statistics are, however, not at hand.

The actual cause of the disease is unknown. We must assume that often a *predisposition* is present, which many and I also believe to be resident in the muscular and nervous systems. This predisposition is, therefore, the essential part. The affection is endogenous; but it may be caused by external causes, above all by *exertion* and *trauma*. In how far other factors are operative, such as improper nutrition, infectious diseases, intoxication, and the influence of cold, is unknown.

Friedreich attached great importance to *overexertion*. He assumes that a condition which damages the muscular tissue in a nutritive and formative manner "by which a lessened resistance and a greater tendency to nutritive disturbances is brought about" is the cause. Even the "normal function," therefore, in children learning to walk is exertion in one predisposed and "acts as a pathologic factor"; just as Edinger has expressed this later for quite a number of diseases. This relative overexertion also explains why the affection in children appears first in the legs and back, while in later life the arms are first involved. However, it remains questionable whether, in early life, the legs are most subject to "exertion"; besides we note sometimes that walking and standing are impossible from the start, while the hands, which are certainly much used by children (even in the recumbent posture) are not affected.

This factor is, therefore, not a satisfactory explanation; but it would be improper to deprive it of all value. It is remarkable that members of the working classes are especially affected by the "juvenile" form of dystrophy, which has its localization in the muscles of the shoulder and upper arm. It certainly cannot be doubted that a true hyperexertion affecting muscles that are already diseased is deleterious.

We must not place too much reliance in the report of an accident case, that a trauma was responsible for a dystrophy which is already widely distributed. In a case of this kind we determined positively that dystrophy, which was denied by the patient, had existed prior to the injury. Then also the disease may escape the observation of the patient for some time. It will hardly be believed how much may be accomplished with a defective muscle

substance, which is even capable of undergoing hypertrophy. It is a well known fact that decided muscular defects may be found in the pectoral muscles of healthy workmen. Therefore if we desire to refer a dystrophy, scientifically to trauma, a normal condition of all muscles prior to the accident must have been present.

Although this requirement cannot be fulfilled in any given case, there are, nevertheless, quite a number of instances—Erb recently collected several from literature—in which it is at least likely that serious trauma was responsible for the appearance of the affection. On account of the great number of accidents it is quite unlikely that a person suffering from dystrophy will be spared, especially as a dystrophic debility of the lumbar and pelvic muscles may be the cause of a fall or accident; nevertheless it would still be improper to deny, *a priori,* that a severe trauma might aggravate or give rise to the malady, especially if it affect the muscular or nervous systems. In regard to the action of trauma of this kind we cannot form a clear conception, as the pathogenesis of the disease is unknown.

It is now necessary to enter upon a brief consideration of the *pathogenesis,* on account of the importance of the question. Friedreich assumed that the primary element of dystrophy was a peculiar *inflammation of the muscles,* which is not rarely propagated to the peripheral nervous system and even to the spinal cord. Were this view correct, we could understand that trauma, which implicates a large mass of muscle in a predisposed person, might give rise to atrophy and then in some unknown manner, perhaps by propagation through the nervous system, distributes itself more widely. There is lacking in the typical cases of dystrophy the element which we recognize as true inflammation, an early implication of the vessels and of the interstitial tissue. Besides there would still remain the question, why an orderly and *symmetric* distribution of hypertrophy, pseudohypertrophy and atrophy should arise in consequence of progressive inflammation, which, under other circumstances, is so much more irregular. Finally the direct proof of the distribution of inflammation is entirely lacking.

After what has been stated it cannot be doubted that on account of the persistent absence of anatomical lesions in the nervous system, *disease of the muscles is the essential element.* These exceptional cases in which, as I observed myself, the ganglion cells of the cord and also the peripheral nerves revealed *marked atrophy* cannot disprove it. In the first place these changes may be of a *secondary nature,* i. e., in consequence of the muscle atrophy and, secondly, there may be *coördinative* disturbances, in that the underlying unknown cause of the disease at the same time also attacks the motor nervous elements, so that these lesions may be due to *complications.* It is difficult to assume that these nerve lesions are of a primary nature because by atrophy of the same ganglion cells other symptoms are produced, as we shall see later on.

Erb, for reasons that I cannot discuss here, has thought that functional changes of a *dynamic nature* might be at fault in the motor spinal ganglion cells, which produce a change in nutrition of the muscle fibers, so that finally a central disturbance becomes the cause of dystrophy. If these dynamic changes are very severe, a direct atrophy of the ganglion cells might result. If this view could be substantiated it could readily be understood that a

trauma, capable of producing shock, would greatly alter the function of the spinal, or in rare cases also of the bulbar, cells; this would give rise in predisposed persons to an extensive dystrophy. The chemical or physical composition of the ganglion cells would be so damaged that the regulation of the nutrition of the muscle fibers would suffer seriously. Hence a condition resembling diabetes mellitus would be present in which lesions, not yet demonstrated, must be present in the composition of certain cells of the body; lesions which under the influence of trauma produce functional insufficiency and thence diabetes, and such a condition is not at all unlikely. Against Erb's view, in my opinion, it may be said that it is unnecessary to the explanation of dystrophy, for the assumption that there is an abnormal predisposition of the muscle fibers and that they degenerate is sufficient to explain the development of dystrophy.

Further it is difficult to understand why the functional weakness of the ganglion cells upon prolonged duration of dystrophy does not ordinarily lead to atrophy, but only exceptionally, and why it is not usual for a secondary degeneration of the motor nerves to appear. Jamin recently, in very ingenious experiments on dogs "has not been able to demonstrate a special trophic action of the nerve centers upon the muscles except when they are stimulated to produce a contraction (motor function)." Nevertheless we are not in a position to state anything definite in regard to these views and thus to reject definitely one or the other of them.

If we accept the view of a primary *myogenic* development of dystrophy it becomes difficult to regard a *local* traumatic effect upon individual muscles, usually the case in trauma, as the causative factor of *general* symmetric dystrophy. If we believe that trophic changes in the spinal ganglia are at fault, the development of dystrophy from shock of the spinal cord substance with a predisposition already present could be understood, but this could hardly occur from local trauma of the extremities. For the present it is by no means certain that trauma causes dystrophy. That it may aggravate one that is already present, is easily conceived according to general pathological laws.

Treatment.—In regard to the *therapy* we are in the same unfortunate position as in other congenital pathologic diatheses—such as hemophilia and the hereditary nervous diseases—for we possess no specific remedies. In a prophylactic respect we must advise parents who have a dystrophic child that it is advisable not to rear any more children. Dystrophic males and females should be cautioned against marriage.

It is of great advantage to recognize the affection as early as possible so as to prevent its progress and to avoid improper treatment. Cautious exercise and passive motion of the diseased muscles is most important. Although the patient must avoid prolonged walking if there is weakness of the legs, and manual labor when there is difficulty in the muscles of the shoulder, nevertheless methodic exercise of the individual muscles, if possible as proposed by Hoffa, upon command is desirable. These movements must be practiced in all joints, several times daily and at short intervals, so that exhaustion does not appear. In this connection *passive gymnastic exercises* may be employed, particularly to prevent the development of pes equinus,

retraction of the muscles, flexor contractures in the knees and stiffness of the joints. To increase circulation in the atrophic muscles, *warm baths and massage* are suitable. The use of dry heat, or of Bier's stasis, is still problematical. It is likely that the nutrition of the atrophic muscles may be increased by these means. The employment of electricity is of no value although it has been in this affection applied in all possible forms. In very rare cases in which the prolonged use of electricity is said to have brought about a cure there was most likely a diagnostic error.

Orthopedic measures to improve the abnormal position of the foot consist of tenotomy, transplantation of muscles and tendons and in some cases the application of light corsets when the scapulæ are far apart. According to Hoffa corsets are badly borne as a rule. I must strenuously advise against the use of extensive apparatus, especially à la Hessing, the same caution being also expressed by Hoffa. This heavy apparatus presses too much upon muscles which are already sufficiently damaged; besides they prevent the patient from employing his individual exercises. When there is much adipose tissue and great lipomatosis, suitable diet is necessary so that the body does not become too heavy, and in the late stages of the disease when the patient can no longer stand and walk that respiratory and circulatory disturbances do not appear.

2. PROGRESSIVE NEUROTIC MUSCULAR ATROPHY (J. HOFFMANN)

We must now discuss *another variety of progressive muscular atrophy* which is much rarer than dystrophy, and in typical cases differs vastly from it; the forms will be described later on. I may be permitted to mention, in a historic review, so as to rectify some incorrect reports that I recognized this affection (which had been previously described, but associated with other maladies) in 1884 and I regarded it as a disease of the *peripheral nerve trunks.* I described it as a peculiar progressive atrophic paralysis of an infantile and hereditary nature. Later Charcot and Marie agreed in the main. J. Hoffmann gave the affection a positive, short name, described and studied it thoroughly in all directions (1888 and later).

Symptoms.—The clinical picture of the disease is quite simple; above all it has a *hereditary* and *family* character. It usually develops in childhood, frequently in youth, but it may develop during maturity even up to the 40th year. The symptoms develop in the *distal portions* of the members, usually first in the feet, more rarely in the hands or even simultaneously in all extremities. According to Charcot it is likely that the small muscles of the feet are first affected, thereby producing certain, not very obvious, difficulties in walking. Later a distinct paralysis and atrophy of the *muscles supplied by the peroneal nerve,* with all the consequences develop: foot drop, steppage gait in consequence of pes varus or pes equinus. Naturally such patients are more liable to fall than normal persons. Club foot may attain such grades that the patient walks upon the dorsum of the foot. In addition to the peroneal paralysis there is a gradual development of *atrophic paralysis of the muscles supplied by the tibial nerve,* so that the leg is characterized by extreme leanness in comparison to the thigh (Fig. 181). After a year or several

years there is an acute exacerbation in typical cases: *the hands are then attacked.*

Atrophy and *paresis* of the small muscles of the hand supplied by the ulnar and median nerves with all of the consequences of this condition develop; the muscles of the forearm gradually atrophy and are palsied.

After a prolonged period the muscles of the upper arm and thigh are involved; but the muscles of the shoulder, trunk and *face* are not entirely exempt. Dubreuilh describes a mask-like change in the features, due to implication of the facial muscles. But the most essential is the *early appearance of distal atrophies and paralyses.*

Electric examination of the muscles and nerves in certain stages of the affection reveals *DeR* in contrast to the exceptional and late occurrence in dystrophy. In the late stages of the disease there is naturally no electric response. An interesting condition observed by Ormerod, Hoffmann, and others, is this, that in the muscles which are not demonstrably diseased there is a decrease of contractility to both currents and, according to Hoffmann, in the entire motor apparatus, nerves, as well as muscles.

Fig. 185.—Neurotic Muscular Atrophy. (Case of Schultze-Hoffmann.) The atrophy of the muscles of the hands is not clear enough.

Fibrillary, lightning-like contractions of the usual kind are absent; according to Hoffmann, and Charcot-Marie, there is a widely distributed contraction, or a permanent unrest, more of a choreic nature, which need not necessarily cease during sleep. Déjérine has observed ataxic phenomena; *tremor* has been noted in the muscles of the face; painful spasms in the calves is occasionally reported.

The *tendon reflexes* are never increased, but decrease gradually and finally disappear in the tendons belonging to the muscles showing atrophy. The *cutaneous reflexes,* and particularly the plantar reflex, are retained; but they may be abolished. There is no report in regard to the abdominal reflexes. The pupillary reflex to light was absent in a remarkable case of Siemerling; in a patient recently seen by me it was sluggish.

Sensation is by no means permanently normal. Not rarely pains, even of a *lancinating* character, are present in the extremities, now and then even in the back. Sensitiveness to pressure over the nerve trunks is rare; hyperesthesia has only once been reported. *Tactile hypesthesia,* as well as *hypalgesia* (especially to farado-cutaneous stimulation) is particularly marked in the distal portion of the members. Hoffmann has also noted a slight decrease of farado-cutaneous sensation in the trunk and head. In a case reported by Charcot the *muscle sense* was disturbed; Hoffmann once noted Romberg's symptom.

Vasomotor symptoms are present in the form of *cyanosis* and marbling in the distal portion of the limbs. Trophic disturbances are absent, except the arthritic changes due to paralysis and atrophy. In the case previously mentioned, I observed *marked atrophy in the bones of the extremity,* especially the distal areas, therefore, an analogous condition to the osseous atrophy present in dystrophy of the muscles. The functions of the bladder, rectum, as well as of the brain, with the exception of slight psychical disturbance, of the heart, and organs of respiration, are normal.

The *course* is very slow, with periods of arrest lasting years and decades. It is impossible for the patient to earn his livelihood on account of incapability of using his hands. Death is due to some intercurrent malady.

This description reveals the differences from dystrophy, which does not exclude the fact that in some atypical cases there may be great similarity in both diseases. Thus Oppenheim and Cassirer report a case in which the same localization of the atrophy was present as in the typical cases of neurotic atrophy with DeR, but with the involvement of the muscles of the face. According to the autopsy, however, it was a case of dystrophy with an unusual localization, for the nerves and the medulla spinalis were not involved.

There is the greatest resemblance between the symptoms of *polyneuritis* and those of neurotic muscular atrophy; only the course of the former is more rapid and progressive. But the distal localization, the slight implication of sensation, the pains which are sometimes present, DeR, the lack of bladder and rectal symptoms are common to both affections. Ataxia, and slight rigidity of the pupil to light, also occur in severe polyneuritis; these symptoms therefore give an appearance of ataxic disturbance. The slight alteration of the muscle sense, seen now and then, Romberg's symptom and the "stamping gait" observed by Hoffmann may be regarded in the same sense. Besides some forms of polyneuritis have been described as *pseudotabes.* Thus the pathology of the disease was regarded by myself and others, especially Déjérine, as a hereditary polyneuritis, which, on account of the involvement of the distal parts of the limbs, might be called an *acroneuritis.*

The few anatomical investigations have shown, however, that besides an *atrophy* of the muscle fibers of varying degree with nuclear and late connective tissue increase, but without marked hypertrophy, and in addition to the *peripheral nerve disease,* there were *peculiar lesions in the spinal cord.* Particularly a *degeneration of the posterior columns,* such as we meet with in severe alcoholic neuritis; besides *atrophic changes in the anterior horn cells,* such as occur in some cases of lead paralysis which commonly also gives rise to peripheral nerve changes. Finally atrophic changes in Clarke's columns; in one case also lesions of the spinal ganglia and even in the *lateral columns.* The peripheral degeneration in the nerve trunk was always most severe in the periphery and in a case of Déjérine's it was associated with interstitial hypertrophy. As there are no reports of autopsies of the early stage of the malady it is impossible to determine which lesions are the first to arise, whether in the central cells, or peripheral nerves, or in both simultaneously. The circumstance that there is DeR so early and regularly favors according to our present experience, a primary peripheral and not a primary central affection.

But no matter how interesting we cannot enter upon a discussion of the

questions which these lesions force upon us. It appears to me to be the proper thing to adhere to the name which Hoffmann has given the malady, for this prejudices nothing, although other terms have been proposed, such as the *spinal-neurotic form of progressive muscular atrophy*, by Bernhardt.

It is clear that quite different lesions have been revealed than in dystrophy and for this reason (as is required by clinical observation) the separation from dystrophy is justified.

Etiology.—Here even more frequently a hereditary predisposition has been found as the essential element; a debility which, in contrast to dystrophy, is to be sought in the nervous system. All other deleterious factors, such as exertion, infectious diseases, trauma, are only contributing causes, and in part may play a localizing rôle. In regard to the conditions in the parents of children with neurotic muscular atrophy, Hoffmann's disease, absolutely nothing is known.

The **prognosis** is obvious from the description of the symptomatology; cures are unknown.

Treatment.—The therapy is the same as that of dystrophy. But orthopedic measures are more likely to be successful; especially as regards walking. But with the progressive tendency of the malady this does not last long, as I had an opportunity of convincing myself in one of my cases.

3. PROGRESSIVE SPINAL MUSCULAR ATROPHY WITH AND WITHOUT BULBAR PARALYSIS

While in the affection just described and also in dystrophy, the hereditary-family factors were early recognized, the occurrence of hereditary transmission in the affection now to be discussed was discovered quite late. Werdnig was the first, in 1891, to point out this condition and it was again J. Hoffmann who placed this new hereditary form of disease upon a broader clinical and anatomical foundation and distinguished it from the other varieties. The disease represents an *infantile, hereditary form of spinal muscular atrophy* and may be designated *Werdnig-Hoffmann's disease.* Its description very naturally follows that of neurotic atrophy.

(a) INFANTILE HEREDITARY FORM OF PROGRESSIVE MUSCULAR ATROPHY (WERDNIG-HOFFMANN)

Symptoms.—As in neurotic atrophy we are here concerned with a very rare affection, which usually appears in *early childhood,* during the first years of life. It is preeminently a family disease so that J. Hoffmann was able to note 19 cases in 3 families. The parents of these children were healthy; the birth of the children was normal nor could anything abnormal be discerned in them. In the course of weeks or months, without an assignable cause, the *movements in the hip-joints* become feebler and feebler. The child can neither flex nor extend the legs in the hip-joint, cannot abduct nor adduct them. Soon, also in an insidious manner, a feebleness of the *muscles of the back and abdomen* follows, so that the child, that may have already learned to walk, which faculty it had lost, can neither sit up nor turn in the horizontal position. After months or years the *muscles of the nape, neck and shoulders* are attacked

and at the same time the atrophic paralysis of the legs descends. In consequence of paralysis of the muscles of the neck, the head of the child falls back when it is raised. Later the *paralysis* and *atrophy* implicate the muscles of the forearm so that the child is quite helpless, all the muscles of the trunk and extremities being paralyzed. The facial, hypoglossal, and fifth, nerves are not affected. Only once Werdnig saw incomplete closure of the eyelid during sleep in a child.

The paralysis is flaccid. The muscle atrophy may be concealed by layers of fat; pseudohypertrophy is not present. If there be no fat there is a wretched condition of emaciation. Hypotonia gives rise to immoderate hyperextension in the joints upon passive movement, and the paralysis of the muscles of the back produces kyphoscoliosis.

FIG. 186.—INFANTILE HEREDITARY SPINAL MUSCULAR ATROPHY. (Case of Thomson and Bruce. Child aged five years and ten months.)

Electric examination reveals decrease or absence of contractility, even *DeR.* Fibrillary contractions are mostly absent but they may be very prominent; the tendon reflexes are abolished.

Sensation, in the main, is normal; but sometimes pains are present. The nerve trunks are neither thickened nor sensitive to pressure. The functions of the bladder and rectum are normal as well as those of the brain, the special senses, the pupils, etc. Bulbar symptoms are commonly absent; only Werdnig in his cases observed difficulty in deglutition.

The **course** is more rapid than in the previously described varieties of atrophy; death is the result of disease of the bronchi and lungs, due to an involvement of the muscles of respiration.

The majority of patients succumb in from 1–4 years after the onset of the affection. A patient of Bruns attained the age of 15.

The **necropsy**, differing from the ordinary dystrophies, reveals very decided *atrophy of the spinal motor ganglion cells* and of their motor nerve roots and peripheral nerve fibers, therefore, of the *peripheral motor neuron.* This coincides with the clinical condition. In addition, *atrophy of the muscles,* much as in the experiments of Jamin, and analogous to those of Stier, characterized by *simple atrophy* of the muscle fibers up to complete disappearance. Even nuclear increase is lacking; hypervoluminous fibers are scant. Fatty

degeneration may be present, interstitial fatty cells are deposited. It is remarkable that the so-called "*muscle spindles,*" peculiar structures which contain a number of muscle fibers, nerve fibers and vessels, structures whose nerve constituents are reckoned among the *sensory elements,* have also been found degenerated by Hoffmann. There are no other lesions in the sensory areas of the nervous system.

The **diagnosis, differential diagnosis** and **prognosis** is evident from what has been stated. The *cause* can be assumed to be only an early damage of the peripheral motor neuron, especially of the motor ganglion cells, a so-called abnormal predisposition, which for some unknown reason proceeds in this manner. Why the ganglion cells of the muscles for the legs and arms are not first affected is not clear; an especially marked and relative hyperexertion of the muscles of the hip and trunk cannot be proven. Infections and intoxications have not been demonstrated.

The **treatment** is ineffectual.

In connection with this affection which is well characterized both clinically and pathologically there are some cases which have not as yet been thoroughly studied, for the anatomical analysis is lacking; they must, however, be mentioned on account of their interest and for the sake of completeness.

Facio and Londe have reported some cases in which a *hereditary, bulbar,* atrophic paralysis appeared in early childhood. Instead of the *spinal,* motor, peripheral neuron being affected, as in Werdnig-Hoffmann's disease, the *bulbar* tracts were attacked. There was atrophic paralysis of the facial nerve, conspicuous atrophy of the tongue, difficulty in deglutition, paralysis of the vocal cords with *complete and partial DeR,* so that a primary muscular dystrophy must be excluded.

Bernhardt and Strümpell have reported cases which resembled the ordinary form of progressive spinal atrophy; such as we shall describe subsequently. In Strümpell's case there was anatomically, marked degeneration of the motor ganglion cells; in that of Bernhardt's there was no autopsy. In both cases there was *heredity;* the disease appeared in adult life.

We now come to those forms of spinal amyotrophy or spinal, progressive, muscular atrophy which were regarded for a long time as the most common and by its discoverers was given the name *Aran-Duchenne's disease.* Later it was necessary to differentiate syringomyelia which was most likely the underlying affection in the majority of so-called Aran-Duchenne's cases. Leyden isolated certain polyneuritic affections after Charcot had already defined *amyotrophic lateral sclerosis,* an affection which we shall describe later on.

Therefore we can hardly longer refer to Aran-Duchenne's disease, but it has become usual to consider it when the maladies just mentioned, besides dystrophy, neurotic muscular atrophy, and Werdnig-Hoffmann's disease can be excluded, and, further, when progressive muscular atrophy has a localization and a course that are now to be described. How rare is the malady in its nosologic limitation may be gathered from the fact that in the year 1886 I made a careful search through literature and could find but 9 cases; more cases were reported later and from a recent compilation by Lorenz, in 1904, there were only 9 additional ones.

(b) PROGRESSIVE SPINAL AMYOTROPHY, ARAN-DUCHENNE TYPE

Symptoms.—The affection develops in *adults,* beginning in the *small muscles of the hand,* therefore differing from dystrophy and Werdnig-Hoffmann's disease, and, finally also from most cases of neurotic muscular atrophy. Either the muscles of the ball of the thumb or the interossei first become atrophic and accordingly weaker, particularly upon the right side. Gradually atrophy and weakness, which are parallel, implicate all of the muscles of the hand and produce a condition of the hand as in ulnar paralysis, the claw-hand, or the ape's hand, as in median paralysis, or any combination of these. Then the muscles of the shoulder are commonly involved; but those of the forearm may be sooner affected. In some instances paralysis of the radial nerve may be prominent from the onset.

The *muscles of the shoulders* that are affected besides the deltoid are those that pull the arm down: the pectorales majores and latissimi, the trapezius and anterior serrati, supra- and infraspinati, the adductors and abductors of the shoulder blades. Gradually a high-graded emaciation appears in these regions with great groove-like depressions. The muscles of the nape of the neck may be affected; only late or in very rare cases are the muscles of the lower extremity implicated. More often the bulbar muscles are affected, especially the lower facial branches and those supplied by the hypoglossal and pneumogastric nerves. *Hypertrophy* and *pseudohypertrophy* are lacking, particularly of the infraspinati and deltoids which are so often thus affected in dystrophy.

The affected muscles which are simply soft but not coarse to palpation are not diseased *in toto* but only in areas in which, however, finally, complete atrophy may appear. In consequence of this state of affairs there may be function of these muscles for a considerable time. In the affected muscles there are *marked* and *widely distributed fibrillary contractions* and very often in those that are beginning to be diseased.

In the later stages these may disappear; now and then, in spite of careful observation, they have been found absent. By these fibrillary contractions we understand lightning-like contractions of a small number of muscle fibers; if larger areas are attacked *fascicular contractions* are spoken of. They may occur in health in consequence of the effect of cold, or under mental emotion; but then they are noted in many muscles simultaneously. When considering their pathologic importance both of the previously mentioned factors must be excluded.

Spastic conditions, therefore stiffness of the muscles in walking or in the recumbent posture are almost never noted; the *tendon reflexes* of the affected muscles *disappear gradually.*

Electric examination of the muscles, which have almost disappeared, reveals complete absence of contractility; in those less involved all varieties of decrease up to complete DeR may be noted. *Sensation remains normal*; also the functions of the bladder and rectum. Symptoms referable to other organs are not present. The disease exclusively involves the spino-bulbar *peripheral motor neuron.*

The malady runs a very protracted course lasting for decades. If the bulbar neurons are involved or the nerves of the muscles of respiration then death occurs in consequence of respiratory or circulatory disturbances.

The **prognosis** is, therefore, unfavorable. An arrest of the symptoms does not take place; marked permanent improvement or even a cure has not been noted.

In regard to the *cause*, only a *hereditary agenesis* of the corresponding nerve divisions is known. Whether this is always present cannot be answered but it is very likely. *Exertion* has a localizing and aggravating influence. In regard to the effects of trauma nothing positive is known. Infections and intoxications have not been demonstrated.

Treatment has a very limited sphere of action. Exertion of all kind must be avoided as this always aggravates the condition. By means of the cautious use of electricity, for a prolonged period, even though the treatment be interrupted, the muscles may be exercised. Massage may be employed in the same manner. Passive gymnastic exercises are necessary in those joints endangered by muscle atrophy. *Local heat* serves to improve the circulation, to decrease the coldness of the hands and exerts a favorable influence upon the disturbed nutrition of the muscles. Dry heat, as well as baths may be used; local as well as general. An attempt at relief may be made with the administration of *strychnin* as advised by Gowers.

The **differentiation** of this form of muscular atrophy from the typical cases of dystrophy or from neurotic atrophy (Hoffmann) and from Werdnig-Hoffmann's disease is easy. It may be difficult and for a time even impossible to separate it from syringomyelia when, as is sometimes the case, the sensory manifestations are absent. In this connection I must refer to the differential diagnosis of syringomyelia. *Amyotrophic lateral sclerosis*, with and without bulbar symptoms, may be mistaken for the affection but this will be described later on.

Hypertrophic pachymeningitis of the cervical cord, a very rare affection, begins with radiating pain, which is often severe; *tumors of the spinal membranes* present the same symptoms. If pain is absent, which is sometimes the case in tumor, paresthesia and spastic paralysis of the legs are present, which are also noted in pachymeningitis; besides the progress of paralysis of the arms is much more rapid. *Disease of the vertebræ*, in the region of the neck, in addition to local and radiating pain in the neck, produces compression paralysis of the legs, quite apart from the deformities which soon develop.

It has happened now and then that *arthrogenous muscular atrophies* have been mistaken for this affection, especially when disease of the shoulder-joints has been overlooked. It is true both affections, spinal muscular atrophy of the humero-scapular type and disease of the shoulder-joint, may occur conjointly and the development of the latter may be favored by the primary atrophy; in arthrogenous atrophy the fibrillary contractions are absent, while in Aran-Duchenne's disease there are no pains in the shoulder-joints, not even upon passive movement of the latter, even backward and downward.

Oppenheim pointed out that *peripheral neuritic processes*, such as arise after local overexertion from continuous or professional movements, may be confounded with the much more unfavorable prognostic condition of spinal muscular atrophy. This was the case in one of my own patients. As a rule a neuritis of this kind is unilateral, associated with paresthesia and hypes-

thesia; but it is necessary in some instances at first to make a provisional diagnosis.

It is impossible up to the present time to determine by clinical methods of differentiation whether, in cases of progressive muscular dystrophy, there is simultaneously a more or less decided atrophy of the ganglion cells; this condition has been frequently noted by Kahler, Erb, Strümpell, Heubner and myself.

This brings us back to the discussion of the relations between atrophy of the ganglion cells and dystrophy, which was touched upon in the discussion of the pathogenesis of this disease. As it is difficult to assume that a progressive atrophy of the ganglion cells at one time produces a dystrophy and at another time spinal progressive atrophy of the Aran-Duchenne type, it is also difficult to appreciate how a supposed dynamic change of these motor ganglion cells gives rise to the severest muscular dystrophies without a demonstrable alteration in their structure, while in other quite similar cases the structure suffers greatly. Hence the following appears to me to be the simplest solution:

Under the influence of unknown deleterious agents, acting upon hereditary predisposition, the muscles at first appear to suffer alone and thus the picture of muscular dystrophy in its various forms arises. But a degeneration of the motor neuron may appear in addition, which may also be the result of a hereditary predisposition. Such a combined system disease of the muscles on the one hand and of the motor nerve tracts and nervous apparatus on the other hand might be placed upon the same plane as the combined system diseases within the nervous system itself, such as Friedreich's ataxia or Hoffmann's neurotic muscular atrophy, or other combined affections of this kind, perhaps also the combination of dystrophy and of the rare form of atrophy of the bones. Most likely these affections do not result simultaneously but are separated by an interval of time.

If muscular dystrophy has advanced to any extent, atrophy of the motor ganglion cells can change the clinical picture but little, as for instance in Heubner's case. If this is not the case degeneration of the cells which sets in earlier may give rise to a combination of symptoms, those of dystrophy and of spinal atrophy; then DeR, fibrillary contractions and a different localization of the atrophy may result. This conception appears to me to be the most reasonable one but it has by no means been scientifically proven.

A few words must be devoted to another affection which bears a very close resemblance to the Aran-Duchenne type of progressive muscular atrophy, so that they have even been regarded as identical, namely *chronic anterior poliomyelitis*. As we do not know whether this affection has the same etiology as Aran-Duchenne's disease and as there are certain clinical differences it is better to describe these affections separately.

(c) CHRONIC ANTERIOR POLIOMYELITIS

Symptoms.—The few typical cases of this affection run their course *much more rapidly* than the Aran-Duchenne type of progressive muscular atrophy

and are not always progressive in the same sense. Just as in acute poliomyelitis an extensive paralysis develops in a few days and later becomes regressive so that finally there are but the remains of pathologic conditions, so also may chronic poliomyelitis, after advancing for months, become stationary.

In adults, in the course of weeks, an increasing debility may occur in one or the other extremity, without atrophy being noticeable from the onset as in Aran-Duchenne's disease. The terminal parts of the extremities are by no means alone implicated. Any muscle group may be involved. The paralysis is *flaccid* and permanently of a *motor nature.* Later on other extremities are involved so that finally all may be paralyzed. The sequence of the paralysis varies. The muscles attacked finally become atrophic and usually show fibrillary contractions. Electric examination reveals various forms of DeR; finally galvanic contractility disappears completely. The tendon reflexes in the involved muscle areas disappear; sensation as well as the functions of the bladder and rectum are normal.

The **course** is usually this, that after the atrophic paralysis has spread for a few weeks or months, a period of arrest appears. In other instances the malady becomes progressive. The muscles of the trunk and of the neck are involved; finally, in consequence of the paralysis of the respiratory nerves, difficulty in breathing appears, to which the patient succumbs after a previous period of immobility. The duration of these paralyses, according to Oppenheim, is from 1–3 years. These cases, therefore, differ only from the Aran-Duchenne type by the localization and the rapidity of the course.

Anatomically, there is mainly, as in the Aran-Duchenne type, an *atrophy of the motor ganglion cells*; whether the vessels, which may be thickened, are always secondarily diseased or whether this is only coördinated, or in some cases a primary involvement, cannot be answered. In a case reported by R. Ewald, there was, simultaneously, a decided thickening of the pia with cellular increase and thickening of the vascular walls, so that here there was an analogous condition to the lesions in acute poliomyelitis rather than to those of simple atrophy of the ganglion cells.

In the **differential diagnosis**, *polyneuritis* must be primarily considered. In this affection there are commonly *sensory irritative* symptoms such as pain and paresthesia, also sensitiveness to pressure over the nerve trunks and the muscles. Later there are sensory phenomena of absence of function which are completely absent in chronic poliomyelitis; besides some etiologic factor may usually be discerned, such as alcoholism or the effects of arsenic. *Lead paralysis* presents the characteristic localization, the lead line on the gums, and lead colic.

The differential factors, distinguishing this disease from dystrophy of the muscles, are obvious from the previous description. In regard to syringomyelia I must refer to the description of this malady; amyotrophic lateral sclerosis will be next discussed. It can always be differentiated by the spastic symptoms and by the increased reflexes from simple atrophy of the ganglion cells of the Aran-Duchenne type and from chronic poliomyelitis.

In regard to **etiology** very little is known; hereditary influences have not been noted. Now and then in persons who have had acute poliomyelitis in childhood, chronic pathologic processes of the kind just described develop.

Among the infectious diseases, in exceedingly rare cases only syphilis need be considered; of the intoxications only lead can come into question. The affection has been noted in the course of pregnancy. Nothing is known regarding the effects of exertion and of cold. But shock, therefore severe trauma, according to the observations of Erb, may give rise to a clinical picture which closely resembles progressive chronic poliomyelitis, but this has not yet been proven anatomically. As shock, however, may produce minute chemical and physical alterations in the white substance, it is very likely that the ganglion cells—especially with an existing predisposition—may be so influenced that degenerative processes may gradually develop in them.

In **treatment** we must insist, especially in the course of the affection, upon absolute rest. This is much more important than any chemical or physical action. Later when the symptoms are no longer progressive, when arrest has taken place, just as in the Aran-Duchenne type, the application of heat and all the other measures, such as the cautious use of electricity, massage, and of active and passive gymnastic exercises, may be employed.

4. AMYOTROPHIC LATERAL SCLEROSIS AND AMYOTROPHIC PROGRESSIVE BULBAR PARALYSIS (CHARCOT'S DISEASE)

I have already referred to amyotrophic lateral sclerosis, the disease with the long name, especially if we include with it amyotrophic progressive bulbar paralysis. For this reason the designation, Charcot's disease, has been chosen because this investigator was the first to define the affection clinically and anatomically. I may mention that I proposed the term *motor tabes*, as it is in direct contrast in the motor field to tabes dorsalis which implicates the sensory areas. Pierret proposes "*tabes moteur*" for the malady. However the term amyotrophic lateral sclerosis has found general acceptance, and it has the advantage of describing the lesion which is present in the vast majority of cases.

The **symptom-picture** of the disease is quite distinct and very much resembles that of spinal progressive muscular atrophy. But besides atrophic paralysis of the upper extremity, which develops much more rapidly than in the Aran-Duchenne type, there is spastic paralysis of the lower extremities and, commonly *atrophic paralysis of some of the bulbar nerves.* The malady develops during mature age, but has been observed among brothers and sisters in childhood. It begins with gradually increasing feebleness of the upper extremities, which is often unilateral and may involve only one hand; then it proceeds to the forearm and upper arm and only later to the legs. In the upper extremity, in addition to the weakness, there is *wasting of the muscles* to which sooner or later paresis is added. The muscle wasting is associated with frequent contractions of the muscle bundles of a fibrillary as well as fascicular character; this sign appears, as a rule, first in the small muscles of the hand. Here and there these symptoms may be almost unilateral. Of *sensory phenomena* only paresthesia is present; tearing, excessive pains are very rare. Hypesthesia or even anesthesia is always absent; if present they are due to complications, such as hysteria.

In the *legs* there is first retardation in walking and stiffness; the patients are only able to take small steps; later the picture of spastic paresis and

spastic paralysis develops. If the atrophic paralysis is advanced in the hands and arms the claw position develops; the hands are pronated and flexed, the forearm is flexed. The arms are pressed toward the trunk. These abnormal positions are the consequence of rigidity and permanent contraction of the corresponding muscles. In the later course of the malady the muscles of the trunk and neck are affected; finally the head can no longer be raised, lowered, or rotated.

Electric examination of the paralyzed muscles shows first a decrease of electric contractility as soon as the wasting appears, later there is often partial DeR, in some muscles even complete DeR.

The *muscles of the legs* are implicated late and to a comparatively slight extent in the atrophy; Oppenheim saw a few instances in which there was early and great atrophy of the muscles of the lower extremities. *The tendon reflexes* are greatly exaggerated. There is bilateral foot-clonus, often also patellar and hand clonus, and even chin clonus. In all tendons, fasciæ, muscle fibers, and muscles of the legs, there is decided contraction not only in the individual muscles but generally and also in the crossed groups. When isolated muscles are wasted entirely, the associated reflex also disappears. Babinski's reflex is nearly always present, i. e., isolated extension of the great toe upon irritation of different parts of the skin of the sole of the foot.

The development of *bulbar symptoms* is of great importance. Sometimes at once, occasionally only after several years, there develops a disturbance in speech which is especially noticeable to the patient. Speech becomes slow, difficult, at first only transitorily, but later permanently. Sudden exacerbations are also noted. The faculty of modulation is lost; speech becomes monotonous, lalling, nasal and, finally, unintelligible and unarticulated. Anyone who has once heard this peculiar "bulbar" change in speech easily recognizes it.

Upon close investigation the *tongue* is found altered. In the first stage of the malady the rapidity of its movements has suffered; but its size is still normal. Marked fibrillary contractures are observed in its muscles and then gradually *atrophy* appears, especially at the tip of the tongue. Finally the organ is greatly reduced in size, there are great groove-like depressions, and permanent glistening; it has lost the power of motion.

The *lower branches* of the facial nerve are affected, and from their paralysis add to the disturbance in speech; this may be studied in all of its details, especially early, by having the patient recite the alphabet. We may thus readily determine in how far the lingual, labial, and other letters are involved. From debility of the muscles of the lips there is difficulty, and, finally, impossibility in puckering the mouth and closing it; therefore it remains open and the lower lip droops. As the orbicularis oris wastes the lips become abnormally thin.

The muscles which raise the mouth and the alæ of the nose become feeble and atrophic; this causes the naso-labial folds to disappear; the upper lip also droops. The patient can no longer show his teeth. Blowing and whistling are no longer possible for, finally, the muscles of expiration are also involved.

In contrast to this affection of the lower branches of the facial nerve, in the greatest majority of cases the behavior of the upper branches is normal; however, in some cases these may also be affected.

In the area of distribution of the motor *fifth nerve* as well as in that of the hypoglossal and facial nerves the muscles of mastication are affected although not to the extent of the other muscles. The jaws can no longer be firmly closed. In addition to altered speech there is sooner or later difficulty in deglutition. As the soft palate is paralyzed, in consequence of disease of the pneumogastric and glossopharyngeal nerves, in deglutition, especially of fluids, the nasopharyngeal space and the cavity of the nose are inundated. The power of deglutition is finally lost. If, at the same time, the constrictors of the larynx are involved, food and fluids introduced into the mouth may find their way into the larynx and into the bronchi and lungs.

Often there is also paralysis of the vocal cords, so that the voice is lost; the cough is then also toneless, and constantly becomes weaker, which is also accounted for by the debility of the expiratory muscles.

In addition there are symptoms on the part of the *heart*, due to paresis of the vagus, such as periodic tachycardia, which may be associated with all forms of irregular cardiac action.

A conspicuous symptom is the flow of large quantities of saliva from the open mouth; this may even occur in the early stage of the disease and if the patient is still able to use his hands he is kept busy with his handkerchief in removing the secretion. Whether the amount of saliva is abnormally increased or not, has not been determined. To increase the terror of the affection, the mental powers are not involved. Intelligence is retained and the functions of the special senses are normal. Attacks of constrained laughing and weeping are to be regarded as automatic processes, as in multiple sclerosis. The bladder and rectum are normal.

The duration of the disease is shorter than that of dystrophy, neurotic atrophy and of the Aran-Duchenne type of myelopathic muscular atrophy; it is estimated by Charcot at about from 2–3 years; but it may often be much longer up to 10 years, even when bulbar symptoms are the first to appear. The insidious onset is exceedingly difficult to determine with accuracy. Raymond and Cestan recently calculated the average duration of their 18 cases at 28 months. The affection is alway fatal, death being due to deglutition pneumonia and a gradual increasing inanition.

Typical lesions are found, in the majority of cases, in the *central nervous system*. Primarily there is *atrophy of the large motor ganglion cells* as well as of their *nerve roots* and the *motor portion of the peripheral nerves*; this lesion in the cord is most prominent in the cervical portion. In consonance with the clinical symptoms, the ganglion cells of the hypoglossal nerve, the motor portions of the vagus, spinal accessory, and trigeminal nuclei, as well as some parts of the facial nerve are involved. The *pyramidal tract*, from the central convolution to the lumbar cord, is often degenerated; the deeper parts are always implicated, beginning at the crus cerebri. The remaining part of the anterior lateral columns in the cord is degenerated, while the posterior columns usually escape. Nevertheless Goll's columns have been found affected. Finally, Charcot and Marie have found degeneration of the large ganglion cells of the central convolutions from which the pyramidal tracts originate.

The *muscles* present about the same lesions as in the infantile form of spinal muscular atrophy.

It has been found, in general, that when there was a clinical predominance of the spastic symptoms, degeneration of the pyramidal column tract was especially severe; when the muscle atrophy was profound there was marked degeneration of the ganglion cells. However, this parallelism does not always exist. There may be spastic symptoms with a lesion of the spinal portions of the pyramidal tracts and inversely there may be lesions of the pyramidal tracts without contractures. The rapidity in the appearance of the affection and its localization which at one time appears early in the pyramidal tracts at another time in the ganglion cells is responsible for this lack of parallelism. However, this does not explain everything; but I shall not discuss this very interesting theoretic point which at present is not of such great importance.

The **etiology** is still obscure. The statement that females are especially affected has not been proven. The age at which the disease develops most often is given by some at from 30–50, by others at from 35–50. A family incidence has been noted in children although the correctness of the diagnosis in these cases has been questioned. As, however, simple atrophy of the motor ganglion cells is hereditary and shows a family appearance, and as lateral sclerosis, running its course with simple spastic paresis, occurs as a hereditary affection as is proven by Strümpell, it is very likely that the summation of these 2 symptom complexes, namely amyotrophic lateral sclerosis is due to a hereditary predisposition of the peripheral as well as of the central motor neuron. But the agenesis of these neurons has not been proven. On the other hand it does not appear that infections or intoxications precede this affection. Overexertion may have a deleterious action and perhaps determine the localization of the symptoms. Nevertheless the peculiar limitation of the malady to the lower branches of the facial nerve as well as to the lower motor nuclei of the bulbus is incomprehensible without the aid of auxiliary hypotheses.

Gowers and lately others (Oppenheim and Giese) have ascribed the affection to the action of trauma particularly if mental shock and exertion were also present. As the actual cause of the disease is X it cannot be denied, *a priori*, that trauma, especially that form which affects the blood-vessels, such as great fear, may have an action upon this X. It is not unlikely, especially according to the rich material which has been accumulated, that it may have a causative influence upon the disease and certainly aggravate an already existing malady.

Differential Diagnosis.—Charcot's disease may be differentiated from simple trophy of the anterior horn cells by the presence of spastic symptoms and the exaggerated reflexes; from Aran-Duchenne's type by its more rapid development.

Syringomyelia is characterized by the peculiar sensory symptoms; only very rarely is there a cavity formation of the anterior gray substance with degeneration of the pyramids alone, and correspondingly the symptoms of amyotrophic lateral sclerosis. But even in these cases the atrophy of the muscles is not so symmetric as in Charcot's disease and the malady is much more prolonged. If the medulla oblongata is involved, the affection may be unilateral.

Chronic myelitis of the cervical portion may give rise to very similar symptoms but also presents sensory as well as bladder and rectal symptoms. *Pachymeningitis cervicalis,* besides atrophy of the upper and spastic paralysis of the lower extremities, produces local pain which radiates into the arms and later there are sensory signs and absence of function, with bladder and rectal phenomena. The stiffness of the cervical vertebræ, in addition to the other symptoms, may be present in tuberculous and other maladies of the cervical vertebral column. Confusion with the other forms of muscular atrophy, dystrophy, and neurotic atrophy, is impossible even though occasionally, in amyotrophic lateral sclerosis, there is a lipomatosis of some few muscles.

The symptom-complex of *amyotrophic progressive bulbar paralysis* may appear without spastic symptoms and just as it may be associated with simple nuclear atrophy of the motor spinal ganglia, it may precede it and may even be present alone. The symptom-picture itself is not altered thereby; all these diseases, fully developed amyotrophic lateral sclerosis, and the isolated atrophies of the motor nuclei in the spinal cord and in the bulbus belong together and as far as we may judge have the same etiologic factors in common, so that what has been stated previously in regard to bulbar paralysis requires no additional comment.

In regard to the differentiation of amyotrophic bulbar paralysis, which often appears first or which alone may be present permanently from other maladies of the bulbus, it may sometimes be difficult to differentiate *syringobulbia.* This is rarely an isolated affection, presents sensory symptoms in the distribution of the fifth nerve and finally either unilateral or at least asymmetric disturbances in the motor tracts of the hypoglossal, pneumogastric, and spinal accessory nerves.

Compression of the medulla from within or externally by tumors gives rise not only to motor and myotrophic symmetric symptoms but is often associated with pain and stiffness of the neck. Finally there are also the signs of cerebral pressure which are foreign to amyotrophic bulbar paralysis.

Asthenic bulbar paralysis or *myasthenia* (Jolly), or Erb's disease, does not produce such typical atrophies as amyotrophic sclerosis nor is there DeR. On the contrary the *myasthenic reaction* is present and finally the rapid fatigue of the voluntary muscles is characteristic. The differentiation from some of the dystrophies of the bulbar muscles, particularly in children, may be very difficult, but dystrophy also involves the muscles supplied by the upper facial branches and spares the smooth muscles of the pharynx.

Finally diphtheritic or toxic paralyses of a different nature which affect the muscles of the soft palate and the pharynx must be excluded; this is easily accomplished for these paralyses appear suddenly and do not show amyotrophy. We must also by direct examination exclude purely mechanical difficulties in speech, deglutition, etc.

Oppenheim calls attention to the possible confusion of rare localized forms with acute polioencephalitis of children; these may sometimes involve the bulbar nuclei. However the development of such an affection is neither insidious nor progressive, but rapid, sudden, and regressive. Besides the conspicuous symmetry of the disorder, as in amyotrophic bulbar paralysis, is absent.

Treatment.—This is very difficult in these serious maladies. There is no specific and an attempt must be made to delay the disease and to prevent some of the sequels. The greatest care is necessary from the onset, and extreme moderation in indulgence of alcohol. The electric current is to be employed for the paralysis, especially in the case of the tongue and difficulty in deglutition. It is serviceable in producing deglutition movements with the aid of the galvanic current, by opening and closing the current. The internal use of tincture of nux vomica or strychnin hypodermically should be tried; arsenic and other nervines are of value. The spasm may be overcome by luke warm baths, mild massage, and cautious active and passive gymnastics. Roberant nourishment must be given as long as deglutition is still possible; later, when there is difficulty in this direction, or paralysis of deglutition, the feeding tube must be employed.

I hope that I have been successful in giving a brief and somewhat lucid description of our present knowledge of progressive muscular atrophy. An interest in these maladies is of practical importance to-day as these questions are very liable to come up in the examination for life insurance and in accident claims.

In regard to diagnosis the most important factor is a frequent examination of the patient. We must never content ourselves with the demonstration that a progressive muscular atrophy is present for this is no more than determining that a febrile condition or dropsy is present in a given case; it is therefore not a diagnosis.

We must first examine the muscles of the entire body from head to foot. The presence of hypertrophy or of relative increase in size must be determined and particularly the infraspinati and gastrocnemii should be examined. We must look for fibrillary contractions and the electric reactions must be taken. The mechanical contractility should be investigated, for in cases with sluggish contraction there is also a slow contraction upon direct galvanic stimulation; but one method is not a substitute for the other.

Following this, the sensory functions are to be examined so as to determine whether beside motor disturbance, therefore, in addition to dystrophy and atrophy of the muscles, there are also other anomalies of the nervous system, for instance, in the peripheral nerves, as in neurotic progressive atrophy, or in the spinal cord, as in syringomyelia. Then the tendon reflexes are to be examined, and this is of particular importance in the investigation of a central disturbance, particularly of the pyramidal tracts.

After testing the other spinal, cerebral, and sensory functions and determining the condition of the body organs it is generally easy, in most cases, to make a diagnosis of the different forms of the malady by following the description which has been detailed.

In a small number of cases it is impossible to make a diagnosis at once, largely on account of the many transitional varieties, or better, mixed forms. The anatomical diagnosis must then remain open. But these difficulties are also encountered in other realms of diagnosis and they must not prevent but, on the contrary, they should stimulate us, to increase and widen our knowledge in all possible directions.

PARALYSES OF THE PERIPHERAL NERVES

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1. PARALYSIS OF THE FACIAL NERVE (MIMETIC PARALYSIS, PROSOPLEGIA, BELL'S PALSY)

In the last few decades the study of diseases of the nervous system has received such an impetus as to make it impossible, even for a specialist in this branch of medical science, to follow carefully and comprehensively all of its rapid advances.

Although the *study of diseases of the peripheral nerves*, and especially their *paralyses*, forms only one branch of this science, yet because of the great variety of symptoms which it presents we have such a mass of material that we must presuppose a certain amount of endurance on the part of the lecturer, and inexhaustible patience and interest on the part of the listener, if this great and interesting branch is to be studied merely in the abstract. We may readily comprehend how the different peripheral nerves constantly innervate different groups of muscles, how the various nerve trunks or branches may be diseased, and by what very unlike symptoms these individual conditions are manifested. Without other prelude I shall begin at once with the subject under discussion, and shall present the most important points in the etiology of individual paralyses, their symptomatology, diagnosis, prognosis and treatment, and, with this end in view, I shall also describe several cases of *facial paralysis* (prosoplegia, mimetic paralysis of the face).

Three patients of *different ages*, a girl aged 6, a young man aged 22, and an old lady aged 63, suffered from peripheral facial paralysis. In fact, facial paralysis (as also radial paralysis which is probably the most common form of peripheral paralysis) occurs at all ages, but it is most common in both sexes between the 20th and 50th years. Prosoplegia may also be congenital, as we shall see later.

If these three patients are closely observed while the face is in repose, characteristic symptoms are noted; these, however, are much more distinct when imitative movements are attempted. On the paralyzed side the forehead is smooth and unwrinkled, the palpebral fissure in the eye of the affected side is larger and wider than upon the normal side; the eye cannot be closed. We may distinctly perceive that the eyeball is drawn first upward and inward, and then upward and outward, a movement long familiar to us, and first described by the celebrated English physician Bell, which, however, has recently again become especially interesting to investigators. Without entering into the numerous discussions stimulated by this phenomenon, to which the name of Bell's sign has been given, it may be stated that, at least in man, it is probably due to an impulse of the will by which the eye (in the tract of the facial nerve) is closed, and by which also one or several impulses of innervation are conveyed to the muscles of the eyeball from the oculomotor nerve (the inferior oblique, perhaps also the superior rectus), while it has an inhibitive effect upon the tract of another branch traversing the same region (the levator palpebræ superioris).

Defective vision in the eye which remains open (lagophthalmos), of which these patients complain, is chiefly to be attributed to a flooding of the eye with tears. We note that the area near the nose on the paralyzed side is smooth, that the naso-labial fold is obliterated; wrinkling the nose is impossible. The nasal opening is narrower upon the paralyzed than upon the normal side, and the tip of the nose may deviate to the normal side. The angle of the mouth upon the affected side is lower than that upon the normal; on opening the mouth only the healthy side of the upper lip rises, and by the unilateral action of the muscles of the lower lip (only upon the healthy side) the mouth appears to be crooked, much more so than when the face is at rest. Saliva flows from the angle of the mouth upon the paralyzed side; upon inflating the cheeks the air escapes from the paralyzed side; in consequence of paralysis of the muscles of the lips the patient cannot whistle, cannot blow out a candle, or can do these only from the paralyzed side. On account of deficient formation of the labial sounds, the speech becomes indistinct; mastication is difficult, or, more correctly speaking, the proper revolution of the bolus and the power to force it from the mouth is limited on account of diminished or lost power in the muscles of the lips and cheeks; therefore it lodges in the paralyzed side of the mouth.

When these patients attempt to put out the *tongue*, it appears to be closer to the paralyzed angle of the mouth than to the normal, and this is due to the crooked position of the mouth. At other times the tongue does not deviate to the paralyzed but to the normal side, as was first noted by Hitzig, and this is attributed to an unconscious, voluntary innervation in regard to the correct position of the tongue to the angle of the mouth under ordinary circumstances. The observation of Fr. Schultze that in facial paralysis the tongue is lower upon the paralyzed side than upon the normal could not be confirmed in my cases.

Whether, under these conditions, as unquestionably sometimes occurs, there is a paresis of the stylohyoid and of the digastric muscles I cannot state. The probable implication in the paralysis of the muscles of the lobe of the ear, as can be readily demonstrated in man, is of only subordinate importance, while the implication of the platysma myoides, as recent observations have shown, is almost invariable.

The *changes in the facial expression* of all patients with unilateral facial paralysis need no special mention. Psychical irritations, such as crying and laughing, lead to grimaces which, no matter how indifferent the individual may be, in the majority of persons suffering from them result in a feeling of profound depression and psychical alteration.

While the majority of authors formerly assumed that the facial nerve influences the *innervation of the soft palate*, and that when this nerve is paralyzed changes in position and movement occur here as well as in the uvula, careful anatomical and experimental investigations have led to the opinion that the facial nerve bears no relation to the innervation of these structures. This nullifies a symptom previously regarded as significant in localization, as will appear later. The deviations in the function of the soft palate actually seen in a few cases of facial paralysis are probably due to the implication of the pneumogastric nerve, which nerve, as we know to-day, innervates the soft palate. The position of the uvula, which varies even in normal persons, is even less calculated to be used in the diagnosis of the localization than that of the soft palate.

Since affections of the middle ear frequently cause disease of the nerves of the face, *anomalies and disturbances in hearing* are often found in facial paralysis. In severe affections with suppuration, caries, and necrosis of the middle ear, this complication and its origin may be readily understood. Since, however, as will be explained later, many cases of facial paralysis follow the milder affections of the ear, the coincidence of slight deafness, of perverted hearing, etc., at the onset of facial paralysis is not surprising.

Besides the anomalies of hearing which have just been named, in some cases of facial paralysis as, for instance, in the case of one of my patients, the faculty of perception of musical sounds and noises shows a peculiar change which was first described by Roux, Wolff and Landouzy, and which is known as *abnormal acuteness of hearing*, hypercusis Willisiana, oxyekoia. The disturbance is probably due to a lesion of the nerve above the point where the stapedius nerve branches off, and therefore in paralysis

of the stapedius muscle, which diminishes pressure in the labyrinth; the compensatory action of the tensor tympani muscle no longer taking place, the tension in the tympanic membrane rises, and also simultaneously the pressure in the labyrinth (Lucæ).

Another important symptom of localization is a *disturbance of taste* in the anterior two-thirds of the paralyzed half of the tongue. This anomaly affects the sense of taste as well as the reaction to galvanism; on the other hand, subjective disturbances of taste are occasionally perceptible by the patient as the first symptom of a subsequent facial paralysis, and he so reports them. There is no doubt that the chorda tympani supplies the fibers of taste in the anterior two-thirds of the tongue, while the posterior third and the base of the tongue, etc., are innervated by the glosso-pharyngeal nerve.

These taste fibers in the chorda tympani come from the trigeminal nerve through the spheno-palatine ganglion and the geniculate ganglion (of the facial nerve), and are conveyed by the vidian nerve to the facial nerve which then transmits these fibers for some distance in its trunk, and introduces them into the lingual nerve through the chorda tympani. These conditions will later be discussed in the description of paralysis of the fifth nerve.

Less important and more rare are *anomalies in the secretion of saliva*, sometimes observed as the consequence of paralysis of the facial nerve. In some cases there is a decrease, in others (perhaps as an irritative symptom) there is a decided increase of the saliva furnished by the sub-lingual and sub-maxillary glands; of anomalies in the secretion of saliva from the parotid nothing positive is known. Some patients also report that their sense of smell is impaired upon the paralyzed side of the nose. This defect may possibly be due to an insufficient dilatation of the nasal opening, although in some cases we may certainly assume with v. Frankl that the same damage which has affected the facial nerve has simultaneously implicated the olfactory nerve.

I have stated that patients who suffer from facial paralysis complain of dimness of vision; this is caused by the failure of the lacrymal tubes to drain off the tears, owing to paralysis of the muscles which close the eye. Goldzieher, Jendrássik and Köster have lately called attention to the fact that in a few cases of facial paralysis the *secretion of tears is very slight or wholly lacking* in the eye of the paralyzed side; therefore, in crying tears flow only upon the healthy side. Although, up to this time, experiments in animals have failed to demonstrate the innervation of the tear ducts through the facial nerve, clinical observation in man seems to show, what Köster attempted to prove, that a lesion of the facial nerve in the geniculate ganglion produces impairment of hearing as well as a diminished secretion of tears. We shall again revert to this.

Besides the secretions of saliva and of tears a possible *change in the secretion of sweat* in the paralyzed half of the face has been described. We know that the secretion of sweat may be decreased as well as increased because, as Köster has shown, irritative and paralytic phenomena may appear conjointly.

Disturbances in sensation may be objectively demonstrated in the paralyzed half of the face, as v. Frankl pointed out, but they are unimportant in the symptomatology of facial paralysis. I once called attention to a slight diminution of sensation in that part of the tongue which is supplied with taste fibers by the chorda tympani, but the symptoms produced by this are of little practical interest.

The same is true of the slight *vasomotor anomalies* which, according to v. Frankl, manifest themselves in a certain puffiness and porcelain-like luster of the paretic half of the face.

Trophic changes, especially in paralyses of considerable duration, may lead to wasting of the muscles of the face, and in rarer cases, as Schultze and Salomonson have reported, to slight changes in its bony structure.

The *appearance of herpes* is among the most interesting but by no means common symptoms observed in connection with facial paralysis. This may assume the form of herpes occipito-collaris and, as we have reason to assume, be caused by the same infection, as yet unknown, which produces the facial paralysis or it is limited to the region of the trigeminal nerve and combined with facial paralysis. In these somewhat uncommon cases, the vesicles appear on the palate, on the uvula, and on that half of the tongue on the paralyzed side.

Owing to the frequency with which facial paralysis occurs, and the zeal with which this interesting affection has been studied for many years, it will cause no surprise when I state that the foregoing embraces but a portion of the symptomatology of facial paralysis.

The question may be asked, How does peripheral facial paralysis make its appearance?

In many cases the onset is apparently sudden, in others progressive, and in the course of a few hours or a day the paralysis gradually becomes complete. In my experience contractions are rarely observed prior to the paralysis which later affects the muscles. Frequently the patients complain of tinnitus aurium, of slight difficulty in hearing, of vertigo, of pain in the head and face, not rarely there is a mild febrile condition or general malaise, perhaps the signs of a general infection of the organism.

That symptoms on the part of the ear should precede the paralysis is not to be wondered at if we bear in mind the prominent part which diseases of the ear play in the etiology of prosoplegia.

I must call attention to a symptom which may either precede or accompany facial paralysis, and which in my judgment has been somewhat overlooked in practice. This is pain in the head, in the ear, in the neck, over the forehead, over the temporals and zygomatic arches, etc., which most likely depends upon the implication of numerous sensory nerves (trigeminal, vagus, glosso-pharyngeal, and upper cervical). The significance of these pains, particularly in facial paralysis, will be described when discussing the prognosis. Another important point is their effect upon the patient, as well as their treatment.

It sometimes happens, as I know from experience, that the patients do not mention their paralysis nor ask to have it treated, but they seek relief from the pain, frequently excruciating, which robs them of sleep, and may be felt for more than a week before the paralysis appears.

The symptomatology of facial paralysis would be incomplete without a somewhat minute description of *the state of electric irritability* which has been especially studied in this form of paralysis.

The following statements in regard to the electric irritability in peripheral facial paralysis will apply equally well to all paralyses of peripheral nerves, no matter whether of the cerebral nerves, the nerves of the extremity, or of the trunk; thus unnecessary repetition will be avoided. I shall purposely refrain from discussing the more minute points in electro-diagnosis which, although exceedingly interesting and well worthy our close study, are of less practical importance to the physician in comparison with other conditions. Of course, those interested may study these rarer electrical changes in contractility from the literature of the subject.

Until very recently the opinion was prevalent that in a so-called, mild, peripheral paralysis there were no changes in contractility; these paralyses in fact are called mild. We know that they may completely disappear in a few weeks. Opposed to these mild forms are the severe ones with which we have long been familiar, in which the electric contractility for the faradic as well as for the galvanic current gradually decreases after the second day, and in the course of from eight to twelve days response to either current is wholly lost. Since the pioneer investigations of Erb, these electrical changes have been designated by the term reactions of degeneration.

The study of the *reactions of degeneration* is so extensive that their description and presentation would necessitate a special chapter on electrodiagnosis. With reference to this subject, I will state that one of my patients, an elderly lady, had a left-sided facial paralysis which had existed for four weeks. In distinct contrast to the healthy right side, there was no reaction from the most powerful faradic current which could be borne upon the face, and upon indirect irritation the same was true of the galvanic current. On direct irritation with the constant current upon the healthy side the muscles showed a prompt and lightning-like contraction with two or three milliampères. This occurred upon the paralyzed side which did not respond to the faradic current, and followed a very much weaker current with a strength of only 0.5 to 1 milliampère and—a point to be especially noted—the contractions were slow and sluggish. It was also evident that the preponderating effect of the cathode which is noted in normal muscles—cathodal closure contraction as is well known occurs before anodal closure contraction—was here less marked than that of the anode, and cathodal opening contraction, which in normal muscles and nerves appears late and only with very strong currents, in this case soon appeared upon the diseased side and with a very feeble current. We know that such conditions last for weeks; what the action may be after recovery or after the paralysis becomes incurable, how it may be modified or changed, we shall not here detail. Nor shall we just now discuss the importance of these reactions in making the diagnosis and prognosis, although we must mention a modification of electric contractility which has recently attracted the attention of investigators.

Besides the mild and severe forms of facial paralysis which have just been described, there is, as in all other peripheral paralyses, a third which has been designated the medium because it occupies a place between the mild and severe forms.

In this form indirect and direct faradic contractility are somewhat lessened on comparison with the normal side, as well as also indirect galvanic contractility; but it is not wholly lost nor even greatly diminished as in the severe forms. Besides a retained or only diminished direct faradic contractility, for about two weeks, sometimes even longer, we note upon direct muscle stimulation an increased galvanic contractility with sluggish contractions and an alteration of the normal electrical formula. I describe this form because it warns us not to be too sanguine in the diagnosis, and especially in the prognosis. A paralysis which appears mild for two or three weeks may in the third week assume a medium type, in which the prognosis is always decidedly better than in the severe form, but which does not permit the promise of speedy recovery.

On the other hand, if we find eight or ten days after the onset of paralysis that the faradic contractility has almost entirely disappeared (I wish to state here emphatically that a test by the galvanic current is not absolutely necessary) we are dealing with a severe form of paralysis, recovery from which can only ensue after some months. When discussing the prognosis we will appreciate the importance of this for the family physician, and how it will protect him from making an erroneous prognosis, either too favorable or too unfavorable.

I cannot deny that competent authorities have recently expressed doubt

as to whether there are any mild peripheral paralyses in the old sense, in which changes in electrical contractility do not arise. I cannot here discuss this very interesting question, but merely allude to it.

Before considering the course, the duration, and the outcome of peripheral facial paralysis, I shall briefly recapitulate what is known of the *etiology* of this common affection. It is apparent that wounds of the face, of the ear, in the region of the parotid gland, in the lower jaw, or of the skull, particularly fractures at the base, also inflammatory processes in the region of the parotid gland, may lead to facial paralysis.

In the same sense we may include as trauma the lesions which occur during *labor*, either from pressure of the forceps or from the pressure of the child's head upon the bony pelvis of the mother.

On account of the proximity of the facial nerve (which runs through the petrous portion of the temporal bone) to the tympanic cavity, it is evident that this nerve is likely to be implicated by *acute inflammatory processes of the ear* as well as by chronic suppurations, by caries, and by necrosis. Although the view, held by French authors, that facial paralysis is almost invariably due to an inflammatory disease of the middle ear does not quite agree with the facts, I must state that the history of patients frequently shows that there was difficulty in deglutition for a few days prior to the paralysis, or dull pain in the middle ear, and this accounts for the assumption that an inflammatory process (for example, from the tonsils through the Eustachian tube) has attacked the middle ear.

In the majority of cases, however, the patients tell us that they have taken cold. *Refrigeration* plays a great, if not the greatest, rôle with the laity in explaining the onset of an attack of facial paralysis. The question might be, and has been asked why, if chilling of the body is of such great importance, is facial paralysis not more frequently observed than is actually the case? A number of authors assume that refrigeration is only an occasional cause, that the true origin is to be sought in a certain *congenital or hereditary nervous predisposition* of the affected person. As a proof of this, or, better, as foundation for this view, it has been alleged that facial paralysis occurs in families, among brothers and sisters, or in parents and children, and, furthermore, that certain persons are repeatedly attacked by paralysis of the seventh nerve.

I have lately studied this so-called *relapsing facial paralysis* somewhat more in detail, and find that it occurs in about 7 per cent. of all cases. Usually there is but one relapse, sometimes a second, a third, and, in rare cases, even a fourth. The same side is not always attacked; this may be the case, but the seat of the paralysis may vary, and sometimes the right, at other times the left side may be affected. In an electro-diagnostic sense these relapses are more severe than the original attack.

In about 10 per cent. of the relapsing cases I found chronic inflammatory or purulent affections of the middle ear, 6.63 per cent. occurred in syphilitics, 5 per cent. in diabetics, 13 per cent. in those with hereditary predisposition, but in the majority of cases, over 66 per cent., no causal factor could be determined. No satisfactory explanation has been offered for the repeated appearance of facial paralysis in the same individual.

In the etiology of facial paralysis syphilis plays an important rôle, in so

far as pathologic processes and products at the base of the brain are concerned, such as gummatous tumors, fibrous and gummatous inflammations, etc. Other authors as well as I have observed that facial paralysis may occur in the early stages of syphilis; whether this is mere coincidence, or whether syphilis acts as a predisposing factor by diminishing the resistance of the organism, I cannot answer without further experience.

The important rôle played in the last decades by pathological processes in the nervous system which have been closely studied and aptly designated by the term *neuritis* is well known. Unilateral and bilateral facial paralysis occur in acute and subacute neuritis, as well as in those diseases which during their existence have generated poisons (toxins) in the organism, have led to inflammation of various nerve tracts, and may also lead to similar affections of the nerves of the face. Among these maladies I must mention gout, diabetes mellitus, tonsillitis, diphtheria, influenza, during the course of which, or as a sequel, facial paralysis may appear. To these may be added diseases due to poisons introduced into the organism from without, such as alcohol, lead, toxic gases (carbonic oxid, carbon bisulphid), etc., all of which may be accompanied by facial paralysis; hence there are many etiologic factors to which Bell's palsy may be attributed.

I shall not describe the affections of the brain or spinal cord which are accompanied by facial paralysis as we shall refer to these when discussing the differential diagnosis; and as we shall subsequently describe briefly the hysterical and congenital (not those arising *intra partum*) facial paralyses, we shall now devote our attention to the *course* and *outcome* of peripheral facial paralysis.

Mild cases of prosoplegia usually recover in from three to four weeks; cases of the medium form in a somewhat longer time, or four to six weeks. In the very interesting medium form, we may note that active motility (which is the main thing for the patient) begins to reappear early in the third or fourth week, while there is still decreased indirect contractility upon electric examination, and DeR may still be demonstrated with direct galvanic stimulation. By this time the patients often cease to come for treatment, although recovery is by no means as yet complete. In the severe form of paralysis recovery does not take place until three or four months have elapsed or even longer. Here, unfortunately, simultaneously with the return of active motility, *contractures* are observed, and, according to my experience, these cannot always be prevented even by the most careful treatment.

A patient, aged 45, who four months previously was attacked by severe right-sided peripheral facial paralysis due to cold, showed this condition of contraction. It was noted that the naso-labial fold on the diseased side which had at first been reduced had become deeper, that the mouth was no longer, as in the beginning, drawn toward the healthy but toward the diseased side, and that the palpebral fissure upon the diseased side had become narrower than that of the normal side. Under some circumstances, and in this stage, it is sometimes difficult to decide which is the normal and which the diseased side. In these cases there are also other peculiarities. If the patient is told to close his eyes, the angle of the mouth upon the diseased side is raised and wrinkles appear in the chin. If the patient is asked to open the mouth or to move the lips, the palpebral fissure on the diseased side partially closes. Briefly, there are so-called coördinate *movements* not previously present.

Moreover, there are *brief, lightning-like contractions* from time to time in the affected half of the face, apparently spontaneous. If we watch closely, we note that

these lightning-like contractions invariably appear when the patient, like all of us, from time to time winks. Although I believe that in some cases these contractions occur spontaneously or, more correctly speaking, are due to unknown causes, they are usually coördinate with the closure of the eyelids.

If these symptoms are very prominent in isolated cases, it may be that we are not dealing with paralysis but with a spasm of the facial muscles; careful observation, however, will reveal the true condition. Since we know that when a peripheral nerve is severely damaged not only the peripheral portion but also the center and the nuclear origin of the nerve (therefore, the entire neuron) are histologically changed, we incline to the view that the symptoms last mentioned are the consequence of changes in the facial nucleus caused by the long continued interruption of the nerve tract and the constant irritation of the center by endeavors to move the muscles of the face. The resistance of the cells is decreased and they react with abnormal ease. Their tonic influence upon the muscles is increased, the activity of one portion of the nucleus is transferred to other parts, and the cells show a tendency to spontaneous discharge.

Thus we may understand that when recovery from a severe facial paralysis occurs in the course of six months, it is frequently incomplete. Some muscles never regain their normal motility, others remain spastically contracted, and where, as, unfortunately, sometimes occurs in isolated cases, no regeneration of the interrupted nerve conduction takes place, the paralysis persists.

We now come to the important chapter of differential diagnosis. First we must be perfectly sure whether we are dealing with peripheral or central paralysis. To decide whether facial paralysis exists at all is not difficult after the foregoing description. The decision may be best arrived at by using the diagram (Fig. 187). If the lesion is above the point of origin of the facial (central or supranuclear paralysis), for example, at *A*, the majority of cases dependent upon lesions of the cerebrum show that the orbiculo-frontal branches are nearly exempt and only the nasolabial nerves are affected. Electric contractility remains unchanged, the reflexes are normal. Usually a simultaneous paralysis of the extremity accompanies the facial paralysis. But here, as elsewhere, this rule is not absolute; that deviations from the ordinary type occur cannot be denied; I should, however, digress too far from my purpose of mentioning only what is most essential and important were I to discuss minutely all of the symptoms which occasionally cause perplexity. But I may here mention that there are facial paralyses due to cerebral foci in which the muscles of the face on the hemiplegic side may be voluntarily moved, in which, however, the paralysis affects the motions which give expression to the face. In such cases, as Bechterew has demonstrated, the fiber tract of the cerebral peduncles is probably interrupted while the voluntary impulses (Nothnagel) still pass freely from the cortex to the periphery through the tracts at the foot of the cerebral peduncles. It may also be stated that, following lesions in the *cerebral peduncles*, besides hemiplegia of the opposite half of the body and the implication of the facial nerve which is observed in other cerebral foci, there is simultaneously a characteristic paralysis upon the same side as the focus in the region of the oculomotor nerve (alternating paralysis). Lesions of the *anterior portions of the pons*, so far as hemi-

plegia is concerned, produce the same pathologic picture as other lesions of the cerebrum. If, however, the lesion is situated in the *lower portion of the pons* where the decussation of the facial fibers is already complete, while the tracts for the nerves of the trunk and extremities have not yet crossed, for example, at *B*, an alternating paralysis occurs (hémiplégie alterne) in which the paralyzed facial muscles upon the side of the cerebral focus show the same electro-diagnostic action which we have learned to recognize in the medium forms, and which is never observed in facial paralysis due to lesions of the cerebrum.

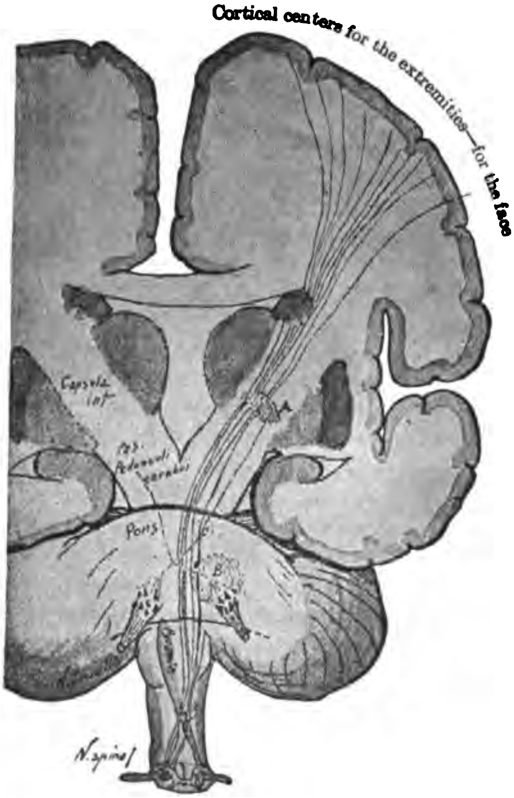

Fig. 187.—Diagram of the Motor Innervation Tract for the Facial Nerves and for the Nerves of the Extremities. Frontal Section through the Cerebrum, the Cerebral Peduncles, the Pons, the Medulla, and the Spinal Cord. (Edinger.)

We know that in chronic progressive bulbar paralysis, therefore in a nuclear lesion of the facial nerve, particularly the nerve branches of the face which supply the lips are implicated; pareses are simultaneously present in the region of other bulbar nerves (the glossopharyngeal, the pneumogastric, the spinal accessory, and the hypoglossal nerves) so that with careful consideration it is not difficult to recognize this form of paralysis, and to avoid error.

Let us assume that the diagnosis of a peripheral facial paralysis is clear. Next we determine the exact location of the lesion in the course of the nerve. We must remember what was previously stated of disturbances of the secretion of sweat, of tears, of the saliva; also of the function of the soft palate and its very questionable dependence upon innervation of the facial nerve; also of disturbances in the sense of taste, after which exact investigation as to the presence or absence of any one of these symptoms will lead to the *localization*.

The farther we advance in knowledge, the more surely will some of our theories be shattered and new principles be established. Fig. 188 shows a diagram first constructed by Erb, which has lately been modified in accordance with the investigations of Köster.

Motor paralysis and possibly disturbances in the secretion of sweat follow a lesion seated below that point at which the chorda tympani branches. If the rupture of continuity is above this point but below the branch for the

stapedius muscle, disturbances of the sense of taste and in the secretion of saliva are added to the other symptoms. If the lesion is above the point at which the stapedius nerve is given off, there is in addition a disturbance in hearing, a hyperacusis; an opinion which Köster did not share.

According to Erb it was formerly assumed that a paralysis of the soft palate followed a lesion of the geniculate ganglion; or, more correctly speaking, paralysis of the velum palati (which can rarely be determined in facial paralysis) is due to a lesion in the region of the geniculate ganglion; to-day we are convinced that the facial nerve bears no relation to the soft palate. On the other hand, according to Köster, we may assume a lesion in this region if there is a disturbance in hearing caused by damage to the auditory nerve, and if the secretion of tears in the eye upon the paralyzed side is diminished or absent. When the seat of a lesion is above the geniculate, we have all the symptoms of absence of function with the exception of disturbance of taste.

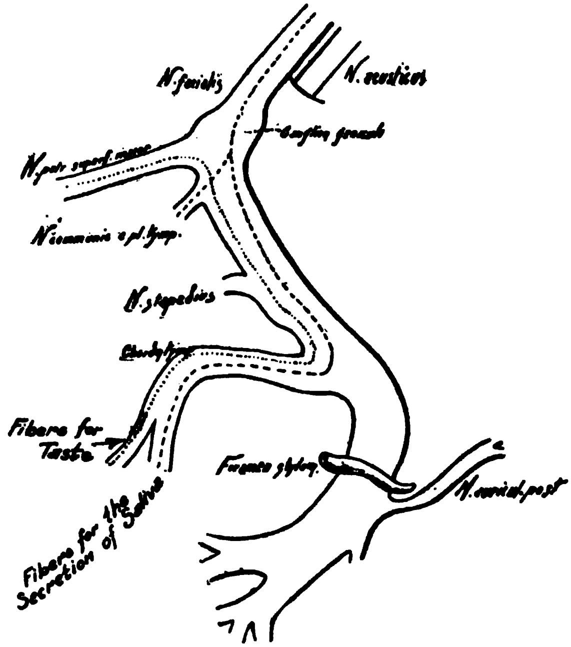

Fig. 188.—Facial Nerve from the Base of the Skull to its Exit at the Stylomastoid Foramen. (Erb.)

The same is true of nuclear lesions, except that other symptoms characteristic of affections of the medulla oblongata are added.

These are the points of view which at present permit the localization of peripheral facial paralysis. But we must bear in mind that science is continually advancing in new paths, and it is not unlikely that future investigations may modify this scheme which at the present time seems absolute.

Pathology.—Finally, if we are asked the *nature of the pathologico-anatomical changes* in the supranuclear, nuclear and infranuclear forms of facial paralysis, we must refuse at this time to answer because a review of this subject would necessitate the description of the entire pathology of diseases of the cerebrum, of the pons, and of the medulla oblongata.

Autopsy reports of purely peripheral paralysis were previously very scarce, but in the course of time have become much more numerous. From these we know that the changes in the nerves are, as a rule, purely degenerative, in the mild cases and those which soon recover mostly very slight, while in the severe cases (in which another cause or some other disease usually terminates the life of the patient) it has been found that degenerative proc-

esses are present which have led to a subsequent degeneration of those portions of the nerve situated below the lesion.

Before concluding this discussion I must state that the facial nerve is implicated not only by diseases of the brain itself but also by a number of pathological processes at the base of the skull (trauma, syphilis, basal tumors).

In diseases of the spinal cord, of course more rarely than in diseases of the brain, the facial nerve may be affected, especially when the pathologic process is communicated by way of the cervical cord to the medulla oblongata and there affects the nucleus of the facial nerve. This may be the case in amyotrophic lateral sclerosis and in spinal progressive muscular atrophy, it is the rule in chronic bulbar paralysis, it may occur in tabes dorsalis, and has been demonstrated in syringomyelia, more rarely in infantile spinal paralysis (acute poliomyelitis) and, a disease we rarely have an opportunity to observe, in leprosy.

In the prognosis of facial paralysis we must differentiate between pure peripheral, so-called rheumatic, and refrigerating paralysis, and that form due to disease of the brain or at the base of the skull. Here the prognosis depends upon the extent of the pathological processes, and is usually less favorable. Syphilitic facial paralysis and that due to basal or cerebral syphilis have a greater tendency to early recovery than that which depends upon nuclear disease. Furthermore, if the facial nerve is not destroyed by caries or by necrosis of the middle ear the prognosis of that form of facial paralysis due to simple inflammatory processes in the ear may also be regarded as favorable.

In rheumatic paralyses the electrical condition of the paralyzed nerves and facial muscles forms a valuable guide to a correct prognosis. If we find on comparison with the normal side after the lapse of eight to ten days that the electric contractility is normal or but slightly impaired, we are justified in regarding the paralysis as of mild type and as likely to yield in from four to eight weeks. But here we must bear in mind what has been referred to before, the late appearance of DeR in the middle forms. A paralysis which was at first regarded as mild may under these circumstances be of longer continuance than was at first thought possible. If, eight to ten days after the appearance of facial paralysis, the contractility is found to be greatly diminished or absent (and I wish to emphasize that this may be demonstrated by the use of the faradic current alone) the case is severe, and three to five months or even longer must elapse before recovery can be expected; even then, unfortunately, recovery is incomplete, inasmuch as contractures and other irritative phenomena such as have been described are liable to appear.

I cannot dismiss this subject without mentioning a symptom previously referred to: namely, the pain with which facial paralysis frequently begins. French authors (Testaz) have maintained that when the pain preceding or accompanying facial paralysis is slight or lasts but a short time the paralysis is apt to be slight and transitory, while under inverse conditions it will probably be several months before recovery takes place. This is true in many cases, but is by no means the invariable rule. The opposite actually occurs, and I attach far greater significance in prognosis to the results of electrical examination than to the symptom just described.

Treatment.—We now come to the most interesting and important part of our discussion, the treatment of facial paralysis. If besides facial paral-

ysis we find syphilis, disease of the ear, trauma, diabetes, etc., our attention must first be given to these conditions and an attempt made to cure them. Whether induced sweating immediately before beginning the treatment, or the use of potassium iodid, of leeches, of hydropathic applications, is of special service, has been less definitely determined than the fact that the pain from which the patient may be suffering can be relieved by small doses of antifebrin or antipyrin (0.25, three times daily). (If seen early attempts to allay local inflammation in the throat, ear, neck or side of the head and face, to reduce congestion in the middle ear and to overcome any rheumatic element, are most strongly indicated. Hot continuous applications to the side of the head and neck and various preparations of salicyl have frequently, to all appearances, resulted in the immediate recession of the paretic symptoms.) As is obvious from our discussion of the electrical reactions, this method of examination is absolutely necessary to enable us to determine in which category the paralysis in question belongs, and may be done if the physician possesses merely an induction apparatus. Although it is true that mild cases may yield without treatment, on the other hand it has been proven that a stabile cathode treatment, applied if possible over the area of the supposed lesion, decidedly ameliorates the pathologic condition, particularly in mild peripheral paralysis. Although, as is generally recognized to-day, severe paralysis can hardly be influenced by treatment with electricity, nevertheless it is wise to give the patient such treatment, at least two or three times a week, the anode being placed over the mastoid process and the cathode applied to the diseased muscles of the face, currents of but moderate strength being employed (2 to 4 milliampères) and electrodes of about 10 cm. in diameter.

Certainly in some of the mild cases a moderately strong faradic current is very serviceable; yet, a very strong faradic current should never be employed, for, not only is it painful to the face, but in severe paralysis it is wholly without effect in the first month. So far as my experience goes, even with most careful use, it does not diminish the very distressing contractures which have been described; they should be treated by stretching and by massage of the contracted muscles, by mild faradization, perhaps by stabile anodal treatment.

Furthermore, the patient should be required, particularly at the first sign of returning active mobility, constantly to innervate and utilize the diseased muscles. Cases which were supposed to be incurable, particularly those due to grave diseases of the ear, have lately been treated by French as well as by German surgeons (so far as I am aware up to this time only in animals) by severing the degenerated facial nerve at its point of exit at the stylomastoid foramen and sewing the central end of the spinal accessory nerve to the peripheral end of the facial nerve, thus producing a new innervation for the paralyzed area. As long as this difficult operation gives no positive results it is not advisable to resort to such measures, no matter how ingeniously devised.

In conclusion, I must refer to some interesting, although less common types of this affection. First, I will mention *congenital facial paralysis*. In these cases the child is actually paralyzed when brought into the world, and the disease does not depend upon external damage to the face or to the nerves of the face occurring *intra partum* from injury during delivery. The following case furnishes an illustration:

Soon after the birth of a child, which is now 10 months old, the mother noticed that it could not suck, it did not cry, did not laugh, but the face remained rigid like a mask. The mouth was continuously open, the lower lip was curved somewhat downward, the eyes did not close and were full of tears, but they rotated freely in all directions. The child was well formed, and except for bilateral facial muscular paralysis there was nothing abnormal in its body; it never suffered from spasms.

Congenital facial paralysis or, more correctly expressed, a defect in the musculature of the face occurs bilaterally as well as unilaterally. Paralysis or a defect in the muscles of the eyes is often simultaneously present; more rarely there are paralytic conditions or defects in other cerebral nerves (the hypoglossal). In the face the muscular tissue disappears either wholly or partially. In many cases the muscles of the lips and chin are absolutely or more or less exempt (here only the muscles of the lower lip on the right side react to strong currents and feebly to intrabuccal irritation, the contractility of the remaining muscles of the face disappearing completely), while in other cases which have been reported the muscles of this region alone were implicated, or at least these chiefly. It is not always easy, especially with adults, to decide whether paralysis is congenital or peripheral, or whether it came on in early youth. At the present time, we are aware of but one anatomical investigation of a case of this kind, that of Heubner, who found a very extensive aplasia of the motor cerebral nerve nuclei as well as of the left olive, and a very meager development of the left pyramid. I must state, however, that congenital facial paralysis may be combined with other congenital defects and deformities as well as with paralysis of the ocular muscles (deformity or absence of fingers or phalanges, web-feet or web-fingers, defective development of the muscles of the chest), and that this anomaly may appear in one or several members of the same family. The abnormality is not always seen in the same muscles, but the conditions may vary; these data form the most important features of the disease of which, at present, we are aware.

An accurate history, the consideration of the points just enumerated, the absence of sensory disturbances, the disappearance of electric contractility, or its great diminution, will lead us to a correct diagnosis. After the foregoing remarks I need scarcely mention that there is no satisfactory *treatment* of this congenital affection.

A special description is also required for *bilateral facial paralysis*. In regard to one form, the congenital, I have already given the important points, and we shall now consider *bilateral peripheral facial paralysis*, which is subdivided into two groups according to whether the cause of the paralysis is at the base of the skull or has its seat external to this. As causal factors for the first category, trauma is important, then syphilitic, carious, or necrotic processes or tumors at the base of the skull. Furthermore, a number of bilateral facial paralyses have been reported in literature for which no other etiology could be determined than that of the unilateral forms; i. e., refrigeration or unfavorable atmospheric conditions. A bilateral affection of the facial nerves may occur in the course of polyneuritis. In such cases, almost invariably, one facial nerve is implicated and paralysis of the other follows in a short time.

In either form the severity of the lesion varies, as well as the presence

or absence of certain symptoms. Double facial paralysis may be due to a bilateral cerebral lesion, to a lesion of the pons, or of the medulla oblongata. Here I must refer to the central form of labio-glosso-pharyngeal paralysis, to pseudobulbar paralysis, to the nuclear form of diplegia facialis in chronic progressive bulbar paralysis, finally, to those forms of bilateral paresis of the seventh nerve which resemble the symptom-complex of asthenic bulbar paralysis or myasthenia pseudo-paralytica gravis.

I shall not discuss in detail the differential diagnosis, as I have already referred to the supranuclear, the nuclear and the infranuclear unilateral forms of facial paralysis.

A detailed history, the investigation of the condition of other cerebral nerves than the facial, the possible implication in the paralysis of the muscles of the extremities, finally, the retained or but slightly impaired electric contractility in cerebral prosopodiplegias will aid us in differentiating these bilateral cerebral palsies from those of purely peripheral origin.

Another form of paralysis is the disease described by Duchenne, which was later referred to by other French as well as German authors, and which is scientifically known either as progressive muscular atrophy of children or as progressive atrophic myopathy (facio-scapulo-humeral type); the muscles on both sides of the face are implicated in the atrophic process.

Finally, those cases of bilateral facial paralysis are especially interesting in which the paralysis upon the side of the lesion differs from that of the other side. Thus, as I observed in a child, a cerebral focus arising in the course of diphtheria may produce hemiplegia and a simultaneous cerebral facial paralysis, while disease of the middle ear due to the same affection may cause a complete peripheral paralysis of the facial nerve upon the non-hemiplegic side. Still other remarkable combinations have been observed; for instance, as in a case described by Oppenheim, hemiplegia due to cerebral syphilis developed in a person who subsequently had a purely peripheral paralysis of the facial nerve upon the same side as the hemiplegia, the palsy being due to a basal, gummatous meningitis. As a rare occurrence I shall mention a case, which so far as I know is unique, of bilateral facial paralysis in a child which, no doubt, resulted from the pressure of the forceps during delivery, and the rare cases of prosopodiplegia in so-called head tetanus in which the wound in the face is in the median line, also a case of diplegia facialis, only one of which has been seen, the person having been bitten by a supposedly rabid dog.

In the clinical picture of diplegia facialis there is no unilateral displacement, no contortion of the face to one side; but the face looks like a mask, since no psychical stimulation whatever produces in it any movement. The eyes are always open and full of tears, and upon trying to close the lids Bell's symptom, previously described, appears bilaterally; the forehead is unwrinkled. Mastication and speech are difficult. Labial consonants and vowels can no longer be enunciated; saliva and mucus dribble from the mouth which the patient cannot close.

The treatment is along the same lines as that described for unilateral facial paralysis; but the prognosis of bilateral facial paralysis is more unfavorable.

Before closing this description I must devote a few words to *hysterical*

facial paralysis, in which there is true paralysis, or, more correctly, paresis only in the rarest cases; on the contrary there is a peculiar spasm of the muscles of the tongue and of the lips, sometimes upon the side opposite to the hysteric hemiplegia, sometimes upon the same side. There is still much skepticism regarding these conditions which are more common in France than elsewhere, and I shall therefore not discuss this form of paralysis. From the foregoing description it is evident that facial paralysis is one of the most interesting and, at the same time, one of the most common and intricate conditions in the pathology of peripheral paralyses.

2. PARALYSIS OF THE TRIGEMINAL NERVE (FIFTH NERVE)

A. PARALYSIS OF THE PORTIO-MINOR; MASTICATORY FACIAL PARALYSIS (ROMBERG)

Paralytic conditions involving the fifth nerve, particularly those which implicate only its motor portion, are very rare. Although in the majority of these cases the motor portion of the nerve is implicated simultaneously with the sensory parts, it will conduce to a clearer comprehension of the subject if we make an artificial division, and separate masticatory facial paralysis (Romberg), or paralysis of the portio-minor, from that form which chiefly implicates the sensory portion of the nerve.

It is well known that the motor portion of the fifth nerve belongs to the third branch and especially supplies the muscles of mastication. The soft palate also receives a small branch which extends to the tensor veli palatini as well as to the tensor tympani.

We have little accurate knowledge of supranuclear central paralysis of the muscles of deglutition due to lesions of the cerebrum; I shall, therefore, at once turn to those *nuclear forms* due to injury of the nerve in its intrapontine course or to lesions of the pons. These are common, and impress their characteristic stamp upon acute, subacute, or chronic bulbar paralyses. Here we must also refer to that disease which has lately so engrossed the attention of pathologists, myasthenia pseudo-paralytica gravis, or asthenic bulbar paralysis without anatomical lesion, in which the weakness of the muscles of mastication, in particular, forms an important clinical symptom.

When I mention that paralysis of the muscles of mastication occasionally occurs also in the course of tabes dorsalis and syringomyelia, I have exhausted the etiologic factors of a purely nuclear paralysis of the motor division of the fifth nerve. [It also occurs in bulbar palsy.—Ed.]

In considering the etiologic factors of paralysis of the trigeminal nerve, especially of paralysis of the muscles of mastication which peripherally affects the nerve, two points are specially significant.

The first of these is the great rarity (in contrast with peripheral facial paralysis) of so-called rheumatic paralysis (we find but few reports of this in literature); the second is the relative rarity of traumatic affections (wounds of the base of the skull from a fall, or injury to the face except stabbing or gunshot wounds). Of course, all pathologic processes affecting the base of the skull, especially its middle grooves, which implicate the bone, the periosteum, and the membranes of the brain, injure the nerve in its infra-

nuclear course just as do pathologic formations which proliferate from the spheno-maxillary fossa into the cavity of the skull.

A congenital absence of both of the upper branches has very rarely been observed, and just as uncommon is atrophy of the muscles of mastication; in one case this may be combined with atrophy of the subcutaneous fatty tissue, in another case with hemiatrophia facialis.

Limitation of the movements of the jaw and a difficulty in deglutition are naturally the most prominent symptoms of paralysis of the motor portion of the fifth nerve. The rise and fall, as well as lateral motion, of the lower jaw, are naturally absent, and in bilateral paralysis this symptom is markedly developed; disintegration of solid food is almost impossible.

In myasthenic bulbar paralysis the masseter and temporal muscles may perhaps be palpated and atrophy is not very prominent, but weakness of these muscles is evident, for they soon become tired and force the patient to desist from mastication and to stop for rest. In regard to the electric reaction of the muscles of mastication, a few reports demonstrate that the atrophic muscles have either lost their contractility or, under galvanism, they show DeR.

In a few cases of paralysis of the fifth nerve, the posterior arch of the palate upon the diseased side was observed to be low down, while the anterior was symmetrically curved on both sides. No severe disturbances of hearing have been noted in paralysis of this nerve, even on complete extirpation of the Gasserian ganglion, as was demonstrated by F. Krause, who performed this operation to relieve severe trigeminal neuralgia; temporarily the patient complained of noises in the ear of the diseased side, that is, the side that had been operated upon.

B. PARALYSIS OF THE PORTIO-MAJOR

A glance at the illustration, Fig. 189, will reveal the portions of the face and head supplied by the three branches of the trigeminal nerve which contain sensory fibers. Besides the skin, the mucous membrane of the eye, of the nose, of the mouth, of the tongue, of the tonsils, of the palate, of the Eustachian tube and of the tympanic cavity, also the pulp of the teeth, the muscles of the eye-ball and of the face, and the dura mater contain sensory fibers.

The majority of etiologic factors which produce trigeminal paralysis affect the motor and sensory fibers simultaneously; but it will conduce to a more accurate knowledge of these conditions if we present the *symptomatology of paralysis of only the sensory portions of the trigeminal nerve* separately.

The main symptom is the hyperesthesia or anesthesia of the affected area, which is gradually accompanied by abnormal subjective sensations. First, in the areas of the skin and mucous membranes which we have mentioned there is sensitiveness on contact, and later, when the affection has advanced, or even immediately if the lesions are severe, sensibility to pain may be lost. Some points which Krause observed in his subsequent examination of patients upon whom he had operated, and which were confirmed by the anatomists Frohse and Zander, are of importance. First, that the middle regions of the skin of the face on both sides receive sensory fibers from the fifth nerve; secondly, as may be seen from the illustration, on the

lateral parts of the face branches of the cervical nerves unite with those of the fifth. Thus it is evident that, even with severe lesions, sensation is not entirely obliterated.

In severe anesthesia the reflexes of the skin and mucous membrane are absent; the secretion of tears may be suppressed, and the sense of smell

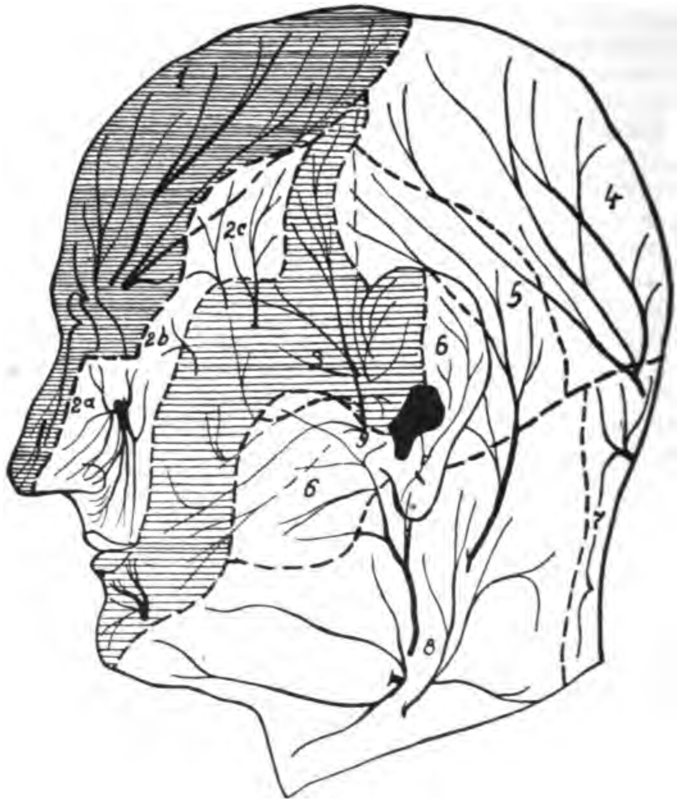

FIG. 189.—DIAGRAM SHOWING THE DISTRIBUTION OF THE SENSORY CUTANEOUS NERVES OF THE HEAD. (After Frohse and Krause.)

The region of the first (*1*) and third (*3*) trigeminal branch shaded, region of the ramus auricularis nervi vagi in the concha auris black. *1*, first trigeminal branch; *2*, second trigeminal branch. *a*, N. infratorbitalis; *b*, N. zygomatico-facialis; *c*, N. zygomatico-temporalis; *3*, N. auriculotemporalis; *4*, N. occipitalis magnus; *5*, N. occipitalis minor; *6*, N. auricularis magnus; *7*, Nn. cervicales posteriores (dorsales); *8*, Nn. cervicales laterales (ventrales); *9*, N. auricularis vagi.

may suffer owing to deficient moisture in the nose on account of the implication of the mucous membrane.

I shall not here discuss the question whether the fifth nerve, as has been assumed, is really the nerve which influences the secretion of the tears or whether this function appertains to the facial nerve. In some cases of isolated paralysis of the fifth nerve the secretion of tears has been found perfectly normal, and when it ceased it could be determined (as it has, for instance, in the case of total extirpation of the Gasserian ganglion) that

the large superficial petrosal nerve (from the facial nerve) was injured either by the operation or subsequently by the formation of cicatrices.

In deep anesthesia of the skin of the face, impaired movement of the muscles of the face has been noted in spite of an absolutely intact facial nerve; this was observed by Bell and Magendie in animals that had been operated upon, and was recently confirmed by the investigations of Filehne and Exner. The patients feel as if their faces were covered by a mask (implication of senso-motility, Exner); when implements are placed in the mouth or upon the tongue the patients either do not feel them or this sensation is indistinct and, with a unilateral affection, they appear as if broken; even mastication is decidedly interfered with on account of the diminished sensation in the mucous membrane of the mouth. I must mention in passing that in unilateral trigeminal paralysis, just as in unilateral affections of the facial and hypoglossal nerves, the tongue may be coated upon the side on which there is anesthesia.

Like facial paralysis, our interest in paralysis of the trigeminal nerve is not exhausted by discussing the anomalies of motion and sensation. *Disturbances of taste* and occasional trophic lesions also invite our attention.

At this point no exhaustive description can be given of the fibers of taste and their course to the central nervous system. Physiologists and clinicians have long assumed that the sensation of taste is conveyed through the lingual nerve from the third branch of the trigeminal nerve, and that the general sensations of the anterior two-thirds of the tongue are simultaneously produced in the same way. If the illustration (Fig. 190) is examined, we will see

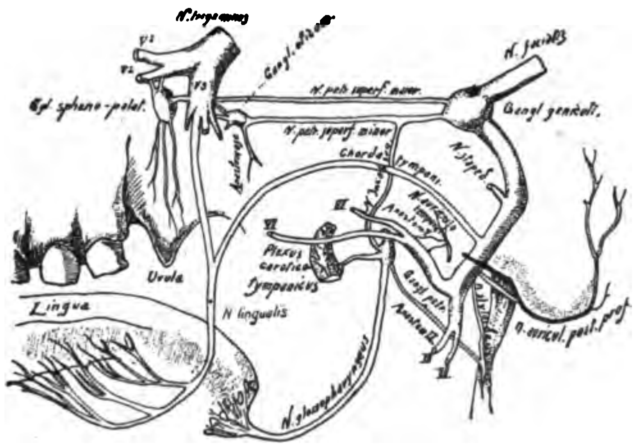

FIG. 190.—THE FACIAL NERVE AND ITS COMMUNICATIONS WITH THE TRIGEMINAL AND GLOSSOPHARYNGEAL NERVES. (W. v. Leube.)

before us the combined results of the latest researches of F. Krause and the neurologists, v. Frankl and Cassirer, as to the supply of individual portions of the tongue with fibers of taste. The fibers of taste for the anterior portion of the tongue are in the basal portion of the trigeminal nerve; in rare cases the whole tongue receives its fibers of taste through this nerve. In some cases no taste fibers are found in the basal portion of the

[illegible]

When we consider, too, that definitely localized lesions of the cerebrum (posterior portion of the internal capsule) produce anesthesia in the areas of the organs of special sense, in the extremities, and also in the fifth nerve, and that even a purely functional disease (hysteria) may cause disturbances of sensation in the course of the trigeminal nerve, we must decide in each individual case whether or not we are dealing with a central affection.

A *peripheral lesion of the nerve* may be positively diagnosticated when the typical symptoms of a trigeminal affection follow a fracture of the skull, a gunshot wound, or any other trauma affecting the skull. In addition to these etiologic factors there are usually tumors at the base of the brain, syphilis or tuberculosis, etc., which implicate the nerve or its ganglia. It must also be noted that this diagnostic principle, first formulated by Romberg, that the seat of the lesion is more likely to be peripheral the greater the disturbance, especially the anesthesia, applies to individual branches.

When a corresponding part of the face is implicated, there is usually a lesion of a main branch; when the entire trigeminal area is involved and trophic disturbances are evident, the seat of the pathologic focus may be assumed to be the ganglion. The implication of other cranial nerves renders it very likely that the seat of the disturbance is at the base of the skull.

The *prognosis* must depend upon the nature of the causal affection. If this be syphilis, if a trauma has preceded, if there is a tumor, antisyphilitic treatment, and, in a few cases, perhaps also operative measures, may bring about amelioration and improvement.

Electricity (galvanization, faradization, franklinization) or irritative inunctions may be used with advantage in anesthetic and paresthetic conditions of the face; for the pain which is occasionally very great lukewarm or hydropathic poultices may be employed; even anodyne remedies may be indicated, perhaps subcutaneous injections of morphin, etc.

The paralyses and enfeebled state of the muscles of deglutition are best treated by galvanization or faradization, the inflammatory conditions in the eye, in the oral cavity, etc., according to the general principles of ophthalmic or surgical therapy.

3. GLOSSO-PHARYNGEAL PARALYSIS

As paralyses of the glosso-pharyngeal nerves alone have not yet been reported, I might limit myself to a description of those pathological changes due to lesions of this nerve which occur clinically. Inasmuch, however, as this nerve may be implicated in all of the pathologic processes which affect the lowest cerebral nerves, it is necessary to relate what is known of its functions, at least briefly, so that these disturbances may be accurately appreciated. The most important physiologic function of this nerve is this, that it supplies the sense of taste to the posterior portion of the tongue, to the mucous membrane of the soft palate, and to the palatine arches. The glosso-pharyngeal nerve terminates at the base of the tongue in the circumvallate papillæ. v. Vintschgau and Hönigschmied maintain that these degenerate after the glosso-pharyngeal is severed, an opinion disputed by B. Baginsky, but concurred in recently by a number of other authors. Besides the sensory fibers which supply taste for certain areas of the tongue, the glosso-pharyngeal

nerve also contains pure sensory fibers which supply the head of the esophagus, the tonsils, the arch of the palate, and the tympanic cavity simultaneously with fibers from the trigeminal nerve. Besides the pneumogastric and the spinal accessory, this nerve also innervates the contrictors of the esophagus, the stylopharyngeal muscle, and perhaps also the glosso-palatine muscle; and it contains motor fibers the disturbed function of which may complicate the clinical picture.

Etiology.—A lesion of this nerve may be caused by pathologic processes which implicate the lowest cerebral nerves at the point whence they originate from the medulla oblongata or the base of the skull. Among these are injuries which affect the cerebral nerves immediately below the base of the skull, tumors of the base or in the cavity of the occiput, syphilitic changes at the base of the skull, or inflammatory affection of the cerebral membranes in this region. We have already spoken of acute and chronic bulbar paralysis due to changes in the medulla oblongata, and of its most typical symptom, difficulty in deglutition. The part which the pneumogastric, the hypoglossal and the glosso-pharyngeal nerves play in these disturbances cannot be estimated in each individual case; it must suffice to know that the glosso-pharyngeal nerve is implicated. When, after diphtheria, an alteration of sensation in the mucous membrane of the pharynx, the palate, etc., is noted, we must consider an implication of the fibers of the glosso-pharyngeal nerve and in some individual, functional, nervous diseases, for example in hysteria, disorder of this nerve and its branches is likely.

Symptoms.—The reports of disturbances of taste in purulent otitis media are especially interesting. When these disturbances are found in the anterior two-thirds of the tongue on the same side as the diseased ear, we know from what has been stated of paralysis of the facial nerve that there is an implication of the chorda tympani. Since it is certain that in these diseases of the middle ear disturbances of taste eventually occur in the posterior third of the tongue and in the palate, they may be attributed to an implication of the tympanic plexus and of the branches of the glosso-pharyngeal nerve contained therein. Furthermore, during therapeutic manipulations within the tympanic cavity, an abnormal secretion of saliva has been observed; if, as is certain, fibers of the chorda tympani supply the sublingual and submaxillary glands, the parotid must obtain its secretory fibers from the glosso-pharyngeal nerve, and Urbantschitsch reports an interesting manipulation of this kind in the middle ear, in which case the saliva was seen to exude directly from Steno's duct.

Little can be said of the **prognosis and treatment.** If in disease of the ear there is a syphilitic affection or a functional neurosis (hysteria), the prognosis is relatively favorable, and under appropriate treatment the malady will disappear. But there is less hope of cure when these conditions are due to disease of the medulla oblongata, or to tumors at the base of the brain, etc. Here also potassium iodid and electro-therapeutic treatment may be tried; temporary improvement may be brought about even though the disease is not cured.

4. PARALYSIS OF THE PNEUMOGASTRIC NERVE

Paralysis of the vagus forms a particularly interesting chapter in the study of paralyses of peripheral nerves. In its extensive course the nerve is subjected to various deleterious influences; as, for instance, at the base of the skull, where it may be affected by inflammatory processes of the cerebral membranes, by neoplasms or similar processes in the bones, or by hemorrhages and aneurysm.

Injuries from blows, or incised and gun-shot wounds of the nerve in the neck are no longer rare occurrences; the nerve is also frequently injured during surgical operations upon the neck (extirpation of tumors, tying of the carotid artery, etc.). A lesion of the trunk of the vagus, particularly its main branch, the recurrent laryngeal, has repeatedly been attributed to tuberculous, scrofulous, or carcinomatous tumors in the neck, in the mediastinum, and in the surroundings of the bronchi. A primary neoplasm of the nerve is rare. More frequently it succumbs to pressure from induration after chronic pneumonia and pleurisy, or, what is particularly interesting, it is compressed in the course of pericarditis, as well as by great dilatation of the left auricle (for example, in mitral stenosis).

Degenerative inflammations of this nerve develop in the course or as sequels of infectious diseases (enteric fever, diphtheria), as well as in metallic poisoning (lead, arsenic) or in chronic alcoholism. That the nerve may be damaged by diseases at its point of origin is apparent. Thus we find the region of the pneumogastric implicated in various affections of the pons and of the medulla oblongata, in acute or chronic lesions in this area, as in tabes, in syringomyelia, in multiple sclerosis, in progressive bulbar paralysis and from tumors; sometimes also in functional diseases of the nervous system, as in hysteria, in which the symptoms point to a special implication of the vagus.

Before proceeding to the *symptomatology of paralysis of the vagus* I must briefly mention two important points: First, the fact revealed by the careful investigations of Navratil, Onodi, Grabower and others that all laryngeal nerves originate from the pneumogastric, and that the spinal accessory is not a laryngeal nerve. According to these researches, the latter nerve is purely a spinal nerve; there is no cerebral accessory nerve. Secondly, the important factor that, on account of the close association of the pneumogastric and the spinal accessory in the jugular foramen, these nerves may be simultaneously involved by pathologic processes, and that with injuries to the vagus in its upper course or disease of the nerve at the base of the skull, the ninth, the eleventh, or even the twelfth cerebral nerve is also frequently implicated. It is well to bear these possibilities in mind so that in the differentiation of these cases, which is not very easy, the physician may more readily comprehend the conditions.

Symptomatology.—Although the symptoms of *paralysis of the larynx* are most frequently due to lesions of the vagus or of its main branch, the recurrent laryngeal, no explicit presentation of the symptomatology can be attempted here; first, because the subject is too extensive for the scope of this article, secondly, because this subject is discussed in a special chapter by a competent authority. I shall only emphasize that paralyses of the vocal cords are due not only to lesions of the recurrent laryngeal, but are also observed in injuries

to the trunk of the pneumogastric; if sensibility of the larynx is retained, we are justified in concluding that the nerve is injured below the point at which it gives off sensory fibers to the superior laryngeal nerve which supplies the upper portion of the larynx.

In injuries to the vagus in its upper course, to the pharyngeal nerves, or to the pharyngeal plexus which passes from the plexus gangliformis to the pharynx, deglutition is seriously impeded. The bolus remains upon the affected side of the pharynx, does not pass into the esophagus but into the larynx, and there causes cough and suffocative attacks.

It is well known that after severing both vagi of young animals they perish, and in older animals pneumonic processes follow. Some authors believe these to be deglutition pneumonia, others assume that, in consequence of injury to the vagus, the lungs are more vulnerable, and a neuro-paralytic congestion occurs. This is hardly the place at which to discuss these still mooted questions; but I should like merely to state that in deciding upon the cause of death after injuries to the pneumogastric, the investigations of Eichhorst, Elias and Hoffmann should be considered, these authors having proven the heart muscle and the endocardium to be severely damaged after such lesions. But the question which most interests us is the following: What are the symptoms on the part of the respiratory and circulatory apparatus which follow lesions of the pneumogastric in man? Different observers, principally surgeons, have given various answers. That a bilateral injury is invariably to be regarded as dangerous to life is self-evident; but, in spite of the objections of some surgeons, I believe that unilateral injuries of the vagus are never to be regarded as slight, since very serious phenomena often appear, and, in any case, the function of the laryngeal muscles is decidedly impaired.

Some surgeons (Deibel, Weidner), in analyzing critically the results of a number of unilateral vagotomies, found no marked symptoms on the part of the heart; others demonstrated an undoubted increase in cardiac activity. On the other hand, prominent clinicians report in disease of the pneumogastric an increased pulse rate occurring as the sequel of infectious diseases (for instance, enteric fever and diphtheria), in alcoholic neuritis, in beri-beri, or in cases in which, post mortem, the vagus is found embedded in a neoplasm, for instance, a mediastinal tumor, or in which it is compressed by caseous bronchial glands.

It is known that, besides the severing of the pneumogastric, paralysis of this nerve may increase cardiac pulsation, and that its mechanical, chemical, or electric irritation may decrease the number of heart beats and that the heart may even be arrested in diastole. I mention this physiological fact because several authors (Czermak, Gerhardt, Quincke, Dubois and others) have resorted to compression of the pneumogastric nerve in the neck in the treatment of paroxysmal tachycardia. I must also state here that tachycardia due to actual disease of the vagus may occur, and, so long as the nerve is not destroyed, especially by the irritation of a neoplasm (carcinomata of the mediastinal glands), may be preceded by a *bradycardia*, a slowing of the heart beat to 50 or 40 per minute; this condition, however, has been only infrequently observed.

Moreover, we know that the pneumogastric nerve is concerned in the

innervation of the stomach and intestines. Except for the previously mentioned difficulty in swallowing, authors have not reported as due to traumatic lesions of the vagus in the neck any marked disturbance in the activity of the stomach and intestines; nevertheless, here and there, abnormal nausea, a decreased motility of the stomach, and an inordinate bulimia have been reported; the gastric crises which are not uncommon in tabes (paroxysmal retching, and vomiting accompanied by severe pain in the gastric region), as well as the rarer esophageal crisis described by Oppenheim, Courmont, Moreira, may probably be attributed to changes in the nerve at its point of origin in the medulla oblongata or in the structure of the nerve itself. I must also state that irritation of the vagus decreases the amount of urine, and would refer to the classic experiments of Claude Bernard that piqûre (injury to the floor of the fourth ventricle between the origin of the acousticus and the vagus) produces melituria and that a similar condition results from irritation of the center of the vagus, also by cutting the vagi; when I state that these conditions as the result of disease of the pneumogastric are certainly very rare in man, if indeed they occur at all, the symptomatology of disease of the vagus, so far as physiologic investigation goes, is complete, and a few words only can be added in regard to the diagnosis, prognosis, and treatment.

Diagnosis.—The diagnosis of paralysis of the pneumogastric may sometimes be easy but is usually quite difficult. If, after injury to, or operation upon, the neck, symptoms appear which point to an impairment of the function of the larynx, of the respiratory organs, of cardiac activity, or of the function of the stomach, we at once recognize a lesion of the vagus. If, however, there is no trauma of these organs, we must elicit a careful history in order to determine whether there has been a previous infectious disease (enteric fever, diphtheria, influenza), must examine the neck for the presence of tumors, and the chest for evidence of pathological changes in the thoracic organs (the lungs, the pleura, the pericardium, the heart, the arch of the aorta, etc.). It must also be borne in mind that the vagus is implicated in the course of many acute and chronic diseases of the prolonged cord (hemorrhage, softening, or neoplasm in this region, also tabes, syringomyelia, multiple sclerosis, and progressive bulbar paralysis), and in every case which indicates a vagus lesion we must seek for proof of the existence of one of these affections. Only in the utter absence of any sign pointing to actual lesion of a nerve, whether at its point of origin or in its peripheral course, and on considering the variability of the symptoms and the temperament of the patient, are we warranted in diagnosticating a neurosis (neurasthenia, hypochondriasis, most often hysteria).

Although the preceding has shown that disease of the vagus may produce changes in phonation, in respiration, in the circulation and in digestion, we know that these symptoms do not necessarily appear in every case. Frequently the muscles of the larynx alone are paralyzed, and this is usually due to a lesion of the recurrent laryngeal nerves. If paralysis of the laryngeal muscles is accompanied by disturbance of heart action and by a change in the rhythm of the respiration, there is every reason to apprehend an affection of the pneumogastric nerve. Finally, if we bear in mind that disturbance of the normal function of the vagus may be produced reflexly by disease

of distant organs (the stomach, the intestines, the liver, the kidneys, the uterus) we shall have considered in the given case every factor that is of importance in the diagnosis of an affection of the pneumogastric nerve.

Prognosis.—The prognosis is always doubtful. If the affection is due to disease of the central organ (medulla oblongata) the outcome depends chiefly on the importance of the underlying affection. Disturbances of heart action and of the respiration after infectious diseases, which may be attributed to implication of the vagus, are always to be regarded as serious complications, and just as serious are those disturbances of the pneumogastric which are caused by disease of the intrathoracic organs, while, as we have seen, *unilateral* destruction of the nerve (due either to the presence of a tumor or to extirpation of neoplasms by operation) offers a much more favorable prospect of recovery, and even of life.

The prognosis in paralysis of the muscles of the larynx naturally depends upon the underlying cause; paralysis of the openers of the glottis (the posterior crico-arytenoid muscles) are the most threatening to life, particularly if the affection is bilateral; here imminent danger can only be averted by tracheotomy speedily performed.

Treatment.—Leaving out of consideration the immediate and great risk of tracheotomy in a bilateral paralysis of the posterior crico-arytenoid muscles, paralysis of the other muscles of the larynx is best treated by electricity with the galvanic as well as with the faradic current. On account of the difficulty of intralaryngeal electrization, and because the possibility of stimulating the nerves and muscles of the larynx by the external application of electrodes to the neck has been demonstrated, the latter method is preferable. Massage of the larynx and similar methodical exercises utilized as auxiliary measures are especially adapted to the treatment of hysterical paralysis of the vocal cords.

If we detect syphilis, scrofula or tuberculosis in a given case of disease of the vagus, appropriate treatment for these underlying conditions should first be considered. If we find acute or chronic disease of the medulla oblongata, this condition must be treated. Disturbances of the respiration and circulation, especially after infectious diseases which produce disease of the pneumogastric, must be relieved by stimulants and a suitable diet to strengthen and tone up the patient. If there are tumors of the neck which interfere with the normal function of the nerve, operation is indicated; for we know from previous experience that a unilateral lesion of the vagus may be borne by the patient without great difficulty, but the removal of malignant tumors is naturally necessary for entirely different reasons.

Neuroses of the pneumogastric such as occur in nervous, hysterical or neurasthenic persons are treated according to the principles on which is based the treatment of the underlying affection. In conclusion, if we have reason to believe that the symptoms indicating an affection of the vagus are attributable to disease of distant organs, the therapy must be directed to the latter; frequently, after the administration of an emetic or laxative, to mention only one of the possibilities, apparently dangerous symptoms may rapidly disappear.

5. PARALYSIS OF THE SPINAL ACCESSORY NERVE (THE NERVE OF WILLIS)

Before entering upon the symptoms of an uncommon affection, *paralysis of the eleventh cerebral nerve*, the *spinal accessory*, a few remarks are in place concerning some recent anatomical and embryologic investigations of this subject. While an internal and external branch of the nerve were formerly mentioned, and it was believed that the internal gave off branches for the heart and the larynx, we have come to the conclusion that the spinal accessory has nothing to do with the innervation of these organs; that, on the contrary, the lowest roots of the vagus control these structures, and the spinal accessory supplies motor fibers to only two muscles, the sterno-cleido-mastoid and the trapezius. It must be added that the trapezius is also innervated by the upper cervical nerves and, according to Sternberg's investigations in monkeys, the junction of the second cervical nerve with the spinal accessory resembles that of a sensory nerve, except that the spinal accessory joins the motor fibers just before it enters the muscle.

When we consider how close the origin between the fibers of the spinal accessory and those of the vagus, how frequently both nerves are damaged by the same cause, also the close proximity of the hypoglossal to the two previously mentioned nerves, we may readily understand that paralysis of the spinal accessory frequently accompanies that of the pneumogastric, perhaps also of the hypoglossal nerve.

Etiology and Symptoms.—In the etiology of paralysis of the spinal accessory, we must first enumerate wounds of different varieties at the back of the head and neck; secondly, operations upon the back of the neck, also in the vicinity of the sterno-cleido-mastoid and trapezius; thirdly, pathological changes in the interior of the upper cervical vertebræ and injuries of the same which may lead to disease of this nerve.

If the upper portion of the cervical cord is diseased, as is the case in tabes, in syringomyelia, in syphilitic affections of this region, or in tumor, symptoms appear which distinctly indicate the implication of the spinal accessory. We know, too, that the musculature of the neck and the back of the neck is frequently implicated in progressive and juvenile muscular atrophy, and this is emphasized as a guide to exact diagnosis.

We shall first consider *unilateral* paralysis of the external branch of the spinal accessory, which produces paralysis of the sterno-cleido-mastoid and the trapezius. Unilateral paralysis of the sterno-cleido-mastoid causes the patient to hold his head obliquely toward the affected side; at the same time the activity of the normal muscle of the other side slightly raises the chin. By the action of the deep muscles at the back of the neck the head may readily be rotated toward the sound side. If the paralysis persists for a long time, the corresponding normal muscle frequently contracts, and atrophy of the paralyzed muscle may ensue, so that instead of the normal contour of the muscle it appears as a thin band; the diseased side of the neck is smooth or even depressed. Bilateral paralysis makes it very difficult to rotate the head while it is elevated; the muscles of the neck on both sides appear to be flattened.

The trapezius muscle has actually three different parts and functions: The

clavicular portion which moves with respiration and at the same time raises the shoulder; the middle part, the true elevator of the shoulder-blade; and the lower part which causes the shoulder-blades to approximate; this is usually implicated simultaneously with the middle portion. If the entire muscle is paralyzed, the shoulder-blade is depressed and appears as if it had slipped down. The internal border of the scapula is oblique, slanting from below inward and from above outward. Upon attempting to raise the arm the internal border of the scapula adheres to the thorax and does not rise. In spite of the fact that the shoulder on the diseased side may be raised through the action of the levator scapulæ, this elevation is not as complete as upon the normal side. According to the recent investigations of Mollier, elevation of the arm anteriorly to the horizontal position is prevented by paralysis of the trapezius; the influence of the muscle may readily be dispensed with; but on weighting the arm thus raised a very light weight will more readily produce fatigue and pain than in the normal arm. While a purely lateral raising of the extended arm to the horizontal position is impossible, the full uplift of the arm is almost perfect; the few degrees lacking upon the diseased side may be concealed by a greater curvature of the vertebral column. It is very difficult to draw the scapula upon the diseased side toward the median line; the contours of the rhomboidei and of the levator scapulæ become very distinct, and put the shoulder-blade into an oblique position (action of the rhomboid muscles).

The *rocking position* (position ou mouvement de bascule) of the shoulder-blade, described by Duchenne, in which the acromial portion of the scapula is depressed, the lower angle deflected toward the median line, and the internal border oblique from above and outward to down and inward is observed only when the middle portion attached to the acromion and the spine of the scapula is implicated in the paralysis.

Although the finding is not the same in all cases, it appears from the investigations of Remak that the rocking position of the scapula does not appear in a pure, uncomplicated paralysis of the spinal accessory. It occurs only when, for example, after an operation, the deeper fibers which are furnished by the cervical nerves and which enter the trapezius at the anterior border of the muscle, are injured.

If there is bilateral paralysis of the external branch, the back is abnormally arched, the thorax is deepened; the clavicles are very prominent, the supraclavicular fossæ form deep cavities. Rotary movements of the head are especially difficult when the chin is raised; the normal outlines of the sterno-cleido-mastoid muscles in the neck have disappeared.

To the symptoms of paralysis of the spinal accessory which have been described, and which affect chiefly the muscles of the neck and the nape of the neck, in numerous cases symptoms are added which were formerly attributed to damage of the so-called internal branch, but which to-day we know to be due to a lesion of the pneumogastric; namely, disturbances in the function of the velum palati, the pharynx, the muscles of the larynx, and anomalies in the beat of the heart.

In unilateral paralysis of the velum of the palate the diseased portion, which appears to be broader than the normal half, is lower, in phonation only the sound half contracts while the paralyzed half remains flaccid, and

on this side there is no power to close the pharyngo-nasal isthmus. Thus the speech becomes nasal, and fluids regurgitate from the nostril on the diseased side. Unilateral paralysis of the muscles of the pharynx forces the patient to incline the head toward the paralyzed side. The same difficulty appears in deglutition, for particles of food which pass into the larynx readily produce cough. When the laryngeal branches are implicated the speech is hoarse and low, and hard coughing is impossible.

Laryngoscopic examination reveals (according to whether there is unilateral or bilateral paralysis of the vocal cord, and according to the law emphasized by Semon which may be observed in many cases) that the posterior crico-arytenoids are the muscles which first show the signs of paresis. In some cases an alteration in the beat of the heart has been observed. Pulse frequency (even in rest) is permanently increased.

The symptoms vary according to the extent of the destructive process and its seat in the individual case; there may be unilateral or bilateral paralyses of the sterno-cleido-mastoid and the trapezius, symptoms which indicate an implication of the so-called internal branch, may be absent or unilateral or bilateral, and may vary according to the involvement of the fibers for the velum of the palate, larynx, etc.

Little can be said in regard to disturbances of sensation and nutrition following paralysis of the spinal accessory. These are very insignificant. If the implication is severe, the paralyzed muscles may atrophy decidedly; thinness of the vocal cord has also been observed, and electrically the different varieties may show altered contractility.

After what has been stated it can scarcely be difficult to recognize the influence of paralysis of one or both of the spinal accessory nerves upon the normal function of the sterno-cleido-mastoid and trapezius. Perplexity can here be occasioned only by the fact that in neuritic processes, in trauma, and in progressive muscular paralysis, the movements of the shoulder-blade and other important muscles are impeded, and thus the true picture of paralysis of the spinal accessory may be masked so far as external muscles are concerned.

Considering the possible implication of the so-called internal branch which supplies the muscles of the palate, the larynx and the pharynx, these structures must be investigated in each individual case, and we should note the condition of the velum of the palate, the muscles of the larynx, etc., as well as test the pulse. Only when the peripheral lesion (for example, after operations upon the neck) is far distant from the intravertebral origin of the nerve, in the middle or posterior third of the neck, may these tests be dispensed with.

If, in a given case, we are assured of the implication of the cervical sympathetic or the hypoglossal nerve, we must seek for a pathologic cause in the interior of the cervical vertebral canal. If this cannot be determined, a high lesion of this nerve in the neck may be excluded. An affection of the base of the brain is suggested if, besides the spinal accessory, the sympathetic and the pneumogastric, paralysis affects the nerves situated more anteriorly, such as the auditory, the facial, or the abducens. The history must always be carefully elicited, and we must bear in mind that, in the given case, we may be dealing with progressive muscular atrophy or with an affection of the central nervous system (tabes, syringomyelia). If syphilis can be

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excluded, we must consider whether there is not a tumor developing slowly in the posterior part of the skull or in the cervical vertebral canal.

Various observers have reported unilateral or bilateral *congenital absence* of the trapezii and of the sterno-cleido-mastoid muscles. If the history be carefully taken this fact may prevent errors in diagnosis.

In disease of the spinal cord, when there are tumors in the vertebral canal, and in progressive muscular atrophy the **prognosis** is naturally very unfavorable; it is brighter when the lesion is of syphilitic character. Improvement or recovery may follow even inflammatory or traumatic affections of the nerve, although the time when this occurs cannot be stated with certainty.

Treatment must be directed to the underlying affection. If a causal therapy, as in syphilis or in disease of the vetrebræ, is possible, if neoplasms can be successfully removed, of course therapeutic measures in this direction are indicated. If there are paralyses due to trauma or neuritic processes, electro-therapeutic treatment should be instituted, and this may be assisted by orthopedic measures.

6. PARALYSIS OF THE HYPOGLOSSAL NERVE; PARALYSIS OF THE TONGUE; GLOSSOPLEGIA

Paralysis of the hypòglossal nerve, in comparison with that of the facial, especially the purely peripheral, is rare. The former is usually caused by gunshot wounds of the nerve, by incised wounds with a knife (attempts at suicide), by glandular tumors and operative wounds of the neck. Paralysis of the tongue is found in a number of pathologic processes at the base of the skull in which the hypoglossal nerve, usually in combination with neighboring nerves (the vagus and spinal accessory) is affected. Here we are prone to find tuberculous, carious, carcinomatous, or syphilitic diseases at the base of the skull, neoplasms in this region or in the occipital foramen, and also in the upper portion of the cervical canal. Not a few cases have been reported of paralysis of the tongue which developed in consequence of disease of the upper cervical vertebræ (caries, necrosis, syphilis). In another series of cases the authors were apparently unable to find any etiologic factor for the unilateral lingual paralysis which they observed. Recent reports of these interesting cases make it appear likely that neuritic processes generated by inflammatory changes in the mucous membrane of the pharynx are the cause of these remarkable paralyses which have perhaps been brought about by glandular disease which implicated the hypoglossal nerve.

The frequent coincidence of such unilateral atrophy of the tongue with unilateral atrophy of the face, the latter being the consequence of infectious disease, particularly of tonsillitis, is significant. Just as in facial paralysis, congenital (usually unilateral) atrophies take place in the tongue but are more rare; and, as in congenital facial paralysis, there is usually a defective formation of other cerebral nerves and anomalies of structure in other parts of the body.

That unilateral atrophy of the tongue is also observed, but more rarely, in scleroderma and in acromegalia is mentioned here for the sake of completeness. In comparison with the rare, purely peripheral paralyses of the

hypoglossal nerve, implication of this nerve from various pathologic changes affecting the brain is relatively frequent. We find this in central hemiplegias due to supranuclear lesions, in progressive paralysis, and most often in all acute, subacute or chronic processes which affect the normal structure of the medulla oblongata. Among these processes I must mention, above all, bulbar paralysis, progressive muscular atrophy, hemorrhages, softening of the medulla oblongata, and syphilitic processes. Some spinal cord affections are occasionally combined with unilateral atrophy of the tongue, and also, it is reported, with tabes and syringomyelia. Isolated cases are found in literature which prove that multiple sclerosis may be associated with atrophy of the tongue.

Symptoms.—In unilateral paralysis of the tongue the diseased half, compared with the healthy, firm side, is much softer, more flaccid, collapsed, fissured, and furrowed; frequently there are fibrillary contractions.

In the majority of cases of unilateral paralysis, the tongue on being protruded deviates toward the paralyzed side, obeying an impulse from the non-paralyzed genioglossus muscle. The concavity of the raphe, too, is directed toward the paralyzed side. If the tongue lies at rest on the floor of the mouth, its tip may deviate toward the normal side, but several cases have been described of unilateral partial lesions of the hypoglossus in which the protruded tongue deviated toward the paralyzed side. Apparently there are variations in the symptoms which depend, perhaps, upon the more or less intense implication of this or that bundle of fibers in the muscles of the tongue.

A fact especially important is the remarkably slight subjective difficulty experienced in unilateral paralysis of the tongue. Although in some cases motion of the tongue may be difficult, and effort is necessary to enunciate certain sounds, the majority of investigators express astonishment over the undisturbed power of speech, of mastication, and of deglutition. In some cases of unilateral atrophy of the tongue the patients are aware of this only because their attention is called to it by the physician. As to *disturbances of sensation* in unilateral paralysis of the tongue, most authors unanimously agree that these are absent or insignificant. This is true not only of sensations of contact, of pain, and of temperature, but also of the sense of taste which, in the main, is found to be unimpaired.

The less essential observation of some authors that the tongue is more thickly coated on the paralyzed side than on the normal side may be mentioned; this probably is explained by its decreased power of motion on the paralyzed side. A curious fact, which so far as I know has been but once reported, is that in paralysis of the hypoglossal nerve severe trophic disturbances and gangrene of the paralyzed side may be observed as well as the previously mentioned atrophy of the paralyzed muscles.

It is well known that the descending branch of the hypoglossus forms a loop with branches coming from the root of the second and third cervical, and thus innervates some of the muscles of the hyoid bone, such as the sternohyoid. sternothyroid, the lower belly of the omohyoid, and perhaps of the thyrohyoid. If these muscles are implicated in the paralysis, as has been observed in a few cases, a flattening of the thyroid cartilage is noted upon the affected side; this is most prominent in the neck upon deglutition, the

larynx deviates laterally, and the electric contractility of the paralyzed muscles reveals characteristic changes.

According to the severity of the lesion, this electric contractility of the muscles of the hyoid bone, as well as of the tongue itself, may denote a mild, medium, or severe degree of paralysis, a fact with which we are familiar, and which need not be minutely discussed after all that has been stated of the phenomena of contractility in peripheral paralyses.

Various observers have demonstrated the galvanic movements on deglutition which were especially studied by Erb; these are produced reflexly by stroking the external half of the larynx with the cathode even when there is complete unilateral paralysis of the tongue, and notwithstanding atrophy.

The **diagnosis** of a unilateral atrophy of the tongue in itself presents no difficulty. If there is a trauma of the neck, the paralysis of the tongue is easily understood; if, besides the hypoglossus, other nerves situated in the posterior fossæ of the skull are implicated in the paralysis and atrophy, syphilis is suggested, disease of the base of the skull and the upper cervical vertebræ are thought of, and, as has been previously stated, the existence of tabes or syringomyelia must be considered. If there is unilateral atrophy of the tongue as well as hemiatrophia facialis, the coexistence of these conditions is readily apparent. When I state that in cases of encephalitis pontis a unilateral paralysis with facial paralysis upon the same side developing during childhood has very rarely been observed, that softening of the medulla oblongata may occur due to disease of the arteries which is also rare and which appears clinically as an alternating hemiplegia (unilateral paralysis of the tongue and atrophy of the tongue with paralysis of the opposite half of the body), I have mentioned all that, in a given case, can aid us in the diagnosis. I may add that if the muscles of the hyoid bone are found to be implicated in the paralysis, we must look for the seat of the lesion in the trunk of the hypoglossal nerve above the point of its anastomosis with the upper cervical nerves. According to Dinkler, the symptoms of a trunk or root paralysis differ only in degree, and the difference between total and partial paralysis is only quantitative. A paralysis of the root is likely when there is partial atrophy with DeR, and when other symptoms denoting disease of the root are simultaneously present.

Before concluding our discussion, *bilateral paralysis of the tongue* must be referred to. Peripheral lesions of the nerves of the tongue causing paralysis have, so far as I know, never been described. But we know that these conditions belong to the symptomatology of acute, subacute, and chronic bulbar paralysis and pseudobulbar paralysis. In these conditions, contrasted with unilateral paralysis of the tongue, speech, deglutition and mastication are impeded to a high degree. The tongue itself is wrinkled and flaccid, it presents fibrillary contractions, its movements are imperfect, the bolus of insufficiently salivated food can scarcely be rolled to the pharynx and saliva collects in the mouth or persistently dribbles from the partially closed mouth. In spite of this extreme disturbance of motility, sensation and taste remain unimpaired or suffer only because the food which is imperfectly masticated no longer comes sufficiently into contact with the surface of the tongue. Electric examination reveals a diminution of contractility or partial DeR.

In a given case it is not difficult to diagnosticate bilateral paralysis of

the tongue, and it is usually attributable to an affection of the medulla oblongata. We must bear in mind that peripheral bilateral paralysis of the tongue is as yet unknown. The implication of the facial nerve, especially of its labial branches, and atrophic conditions in the muscles of the body as well as bilateral paralysis and atrophy of the tongue, must be called to mind; a review of the history will show that bilateral paralysis of the tongue has perhaps only lately been added to progressive muscular atrophy which had existed for a considerable time, after which we may readily diagnosticate bilateral paralysis of the tongue and recognize the pathologico-anatomical disturbance which causes it. We would digress too far to discuss at this point the differential diagnosis of paralysis of the tongue due to unilateral or bilateral lesions of the cerebrum (pseudo-bulbar paralysis).

The **prognosis** in peripheral paralysis of the hypoglossal nerve is much more favorable than in those forms produced by diseases of the brain, of the medulla oblongata, and of the spinal cord. There is more hope of recovery in paralyses due to syphilis and disease of the cervical vertebræ than in those due to central lesions.

The **treatment** of paralyses of the tongue which follow an affection of the cerebrum, of the medulla oblongata, or of the spinal cord must be directed to the underlying affection. Appropriate surgical or orthopedic treatment may be instituted in paralysis of the tongue caused by disease of the vertebræ, and antisyphilitic treatment when the affection is attributable to syphilis. Electricity is always beneficial in central paralysis as well as in that due to purely peripheral conditions, either because the muscles of the tongue or, indirectly, the nerves are stimulated by faradism or galvanism, and because the galvanic current is applied to the brain, the medulla or spinal cord.

7. MULTIPLE CEREBRAL NERVE PARALYSIS

After the consideration of paralysis due to lesions of individual cerebral nerves, we must describe those not infrequent *paralyses* in which the lesion cannot be located definitely in this or that nerve but in which *a number of cerebral nerves* are implicated. I refer to the so-called *paralysis of multiple cerebral nerves*. In the preceding description I have frequently referred to the fact that, besides one special cerebral nerve, occasionally this or that neighboring nerve or even several may be implicated in the process.

In the **etiology** of such cases we find a history of injuries to the head, the neck, and the back of the neck, and of fractures of the skull, especially at the base. In addition, tuberculous and syphilitic changes at the base of the skull and tumors in this region play an important rôle. Even if the cerebral membranes at the base alone are implicated, or, as in the case of tumors (sarcomata, carcinomata) if the bones also are injured, several cranial nerves, as a rule, are affected, although this depends upon the seat of the lesion. Congenital conditions (insufficient development?) very rarely affect several of the cerebral nerves; nevertheless there are a few reports of this in literature (Schapringer, Schmidt).

It may here be in place to call attention again to the **symptomatology** of paralysis of the individual cerebral nerves which has been heretofore mentioned.

In the majority of cases multiple cerebral nerve paralysis is unilateral, and implicates three or four, often even more of the nerves; in syphilitic affections and in tumor the pathologic conditions may implicate either one side or both.

While disease of the olfactory nerve is rarely mentioned, inflammation of the optic nerve and a consequent impairment of the sight plays a far more important rôle. Ophthalmoscopically we usually find neuritis of this nerve, and only when there is direct pressure upon the chiasm or the optic nerves such as is produced by tumors of the pituitary body, do we observe white atrophy of the papillæ without preceding inflammation. While, notwithstanding existing inflammation of the nerve, the sense of sight may be more or less retained, direct pressure of a tumor in the region of the hypophysis producing atrophy causes a well-defined bi-temporal hemianopsia, and the subsequent implication of the optic nerves may occasionally aid us in the diagnosis.

It must be borne in mind that inflammation of the optic nerve as well as the other symptoms of the patient (headache, etc.) are the general expression of a limitation of space within the skull, and they have a local diagnostic value only when the inflammation is unilateral and corresponds to the symptoms upon the affected side produced by injury to the other cerebral nerves.

If a neoplasm ruptures the anterior cranial fossa, exophthalmos and complete paralysis of all the muscles of the eye may follow. If the lesion is in the middle of the cranial fossa it causes a conspicuous paralysis of the muscles of the eye (especially of the oculomotor, or the trochlear nerve, more rarely of the abducens). Moreover, symptoms attributable to the damaged trigeminal nerve appear (rarely paralysis of the muscles of mastication), particularly as paresthesias, anesthesias, pain in the facial branches of this nerve, to which naturally a neuroparalytic inflammation of the corresponding eye may be added. If the pathologic processes at the base involving the facial and the acoustic are very extensive, the last four cerebral nerves (glossopharyngeal, vagus, spinal accessory, and hypoglossal) may sometimes be implicated. If, however, these nerves are affected while the nerves of the ocular muscles and the fifth nerve remain undamaged, we may regard it as certain that the lesion is situated in the posterior cranial fossa.

We have now enumerated the fundamental laws upon which to base a diagnosis, and it is unnecessary to recapitulate the individual points.

As to the *nature* of the affection which leads to multiple cerebral nerve paralysis, it may be stated that young persons who are predisposed to tuberculosis, or who suffer from tuberculous affections of the bones or joints, are prone to tuberculous disease of the cerebral membranes or of the bones at the base of the skull.

The probability of syphilitic affection is more frequently assumed in later life than in the young; the history combined with other characteristic symptoms of syphilis will justify a suspicion of this disease at the basis cranii. The question, often difficult to decide, whether in a given case we are dealing with syphilis or a neoplasm, is frequently determined by the results of antisyphilitic treatment. Nevertheless, this cannot be depended upon with certainty, for cases have been reported in which, in spite of antisyphilitic treat-

ment, the subsequent autopsy revealed the presence of a syphilitic process at the base of the brain.

If there has been a trauma of the skull, particularly a fracture at the base, or an operative wound in the upper part of the neck, there can hardly be a doubt as to the diagnosis.

In the **diagnosis** of multiple cerebral nerve paralysis, preexisting disease of the spinal cord must be considered (tabes, progressive paralysis, syringomyelia), in which case numerous cerebral nerves may be paralyzed simultaneously; when I state that in so-called nuclear paralysis of the muscles of the eye other cerebral nerves, chiefly the facial and the trigeminal, may be implicated, I have enumerated all the points which are necessary in a given case for the diagnosis of multiple cerebral nerve paralysis.

In conclusion I must briefly remark that a paralysis which involves several cerebral nerves situated in the posterior cranial fossa, and which develops gradually, may occasionally be confounded with Duchenne's progressive bulbar paralysis. Another fact is that, especially in pathologic processes in the posterior cranial fossa, besides paralysis of the nerves of this region, polydipsia and polyuria may appear, and may confirm the diagnosis of a pathologic process in this area.

In the **prognosis** and **treatment** of multiple cerebral nerve paralysis, it may be said that the cases due to syphilis or external injuries have a much better chance of recovery than those due to tuberculosis or to tumors at the base of the skull. Notwithstanding the oft quoted failure of antisyphilitic treatment, it is wise to institute this, for even in some cases of undoubted neoplasms a period of improvement, at least, was thus brought about. Only an unfavorable prognosis can be given when multiple cerebral nerve paralysis occurs in the course of chronic diseases of the spinal cord, and here the treatment must be based upon the underlying affection. In cases in which the implication of the trigeminal nerve produces severe neuralgic pains, narcotics should be administered to the patients.

8. PARALYSIS OF THE DIAPHRAGM

Of the paralyses affecting the region of the cervical plexus the practitioner is interested only in *paralysis of the phrenic nerve*, paralysis of the diaphragm, which is seldom observed. The nerve of the diaphragm extending from the region of the third and fourth cervical nerves may be injured in its course through the neck and within the thorax by trauma or neoplasms (aneurysms) but this is more rarely the case than its degeneration after some infectious disease, such as diphtheria and scarlatina, or by polyneuritis, occasionally from exposure to cold, more frequently from various poisons (alcohol, lead, carbonic oxid, opium, etc.).

The branches distributed to the diaphragm may be affected by inflammation of the pleural or peritoneal lining of the same, and in injuries of the upper cervical vertebræ and of the upper cervical cord this chief nerve of respiration may be damaged by various pathologic processes as well as in chronic diseases of the cervical cord (bulbar processes, progressive spinal muscular atrophy) and in acute inflammatory affections of this region. In rare cases, the nerves has been found implicated in tabes; functional (hysteri-

cal) paralyses also occur. In very rare cases there is congenital absence or partial absence of the diaphragm.

The **symptoms** of paralysis of the phrenic nerve depend upon the absence of contractions of the diaphragm in inspiration. The thorax is dilated by the contraction of the intercostal muscles and the scaleni, but while it expands the hypochondriac and epigastric regions retract. The respiration, hardly increased during rest, becomes very rapid on exertion and dyspnea and a sense of suffocation follow. The patient avoids deep respiration because it often causes pain and a feeling as if the abdominal viscera were rising into the thorax. Since the abdominal cavity is enlarged by the high position of the diaphragm and by paralysis of the muscle, the strength of the abdominal press (prelum abdominale) is decidedly decreased, and all expiratory acts (cough, evacuation of the intestines, etc.) are accomplished with difficulty.

Litten's Diaphragm Phenomenon.—I must here call attention to a phenomenon discovered by Litten which may be demonstrated in every normal person; it consists of an undulatory movement which, beginning bilaterally at the height of the sixth intercostal space and extending as a straight line or slight furrow with deep inspiration descends several intercostal spaces, then rises to the same height during expiration. This phenomenon is best seen in a person who assumes a horizontal position with his back toward the light; the sign is most clear upon the right side of the thorax anteriorly, less well marked upon the left side and upon the back. In children and women the phenomenon is less distinct than in men. Litten's phenomenon has already been employed diagnostically; for example, in the hemiplegia of adults as well as in that of children, the phenomenon is less well developed upon the paralyzed than upon the normal side.

The electric contractility of the normal phrenic nerve is the same as that of all other motor nerves. In asphyxia (chloroform or charcoal) the electric contractility is greatly diminished; the same is true of all paralyses of this nerve due to neuritic processes, particularly in cases of alcoholic neuritis.

If we bear in mind the points just stated in regard to paralysis of the diaphragm, symptoms arising after injury or other disease of the upper cervical vertebræ may correctly be referred to a lesion of the phrenic nerve. Similar conditions arise in neuritic affections of the nerve, in the case of alcoholics, or in persons who have just recovered from severe infectious diseases. In chronic affections of the cervical cord and the medulla oblongata, especially in their terminal stages, the action of the diaphragm may be paralyzed, and it has already been mentioned that this may occasionally occur in tabes and in poisoning from lead and gas. We should always consider a more or less severe implication of the diaphragm when we are dealing with a hysterical person, in lead poisoning, or if respiratory difficulty appears in the course of peritonitis or pleurisy.

In the rare unilateral paralyses of the phrenic nerve in consequence of tumors in or injury to the neck, Litten's phenomenon, the decreased or almost inaudible inspiratory murmurs, the anxiety and dyspnea of the patient, his low speech, and the absence of laryngeal stridor may assist in the diagnosis.

The **prognosis** of paralysis of the diaphragm depends upon the underlying condition, therefore in injuries to the vertebræ of the neck and in acute or chronic diseases of the cervical cord and the medulla it will always be dubious.

All diseases of the phrenic nerve must be regarded as serious. Although, while the patient remains quiet, the difficulty in respiration is not decided, this may at once become dangerous if the slightest affection of the bronchi or lungs is superadded. Aside from the form occurring in diphtheria, alcoholic paralyses of the phrenic nerve are the most serious; at all events, they necessitate more care and are less favorable than those which occasionally appear in persons suffering from lead poisoning or syphilis.

We should not be too sanguine in the prognosis of paralysis occurring in the course of hysteria, for we must bear in mind the not infrequent experience that death may occur suddenly.

I must admit that in a recent article two American authors, Schröder and Green, have attempted to prove that the cases described in literature of injury to the phrenic nerve with fatal consequences depended upon erroneous observations, that severe injury of important organs was usually found, and that the unfavorable outcome could not be ascribed *solely* to a lesion of the phrenic nerve. Their own investigations and the study of the literature of the subject led these authors to the conclusion that a unilateral injury of the phrenic nerve is not always followed by a fatal termination or even serious consequences.

Treatment.—Paralyses of the diaphragm due to wounds of the neck or to trauma of the cervical vertebræ naturally require careful surgical treatment. Paralysis due to syphilis, to lead poisoning, etc., must be combated by treating the underlying condition with the measures now to be described. Paralysis of the phrenic nerve on a psychic basis (hysteria) should be treated by suitable physical and psychical therapy. If we are dealing with asphyxia, as in the new-born, in poisoning with toxic gases, in chloroform narcosis, or that which occurs in severe hysterical attacks, the use of electricity, especially the faradic irritation of the phrenic, will sometimes be very beneficial even in apparently desperate cases.

The electrodes, which should not be too small, are placed on both sides of the neck at the external border of the sterno-cleido-mastoid, some pressure being exerted from without inward. (This method of treatment has been evolved by v. Ziemssen.)

The current should be moderately strong; we need not fear to stimulate other inspiratory muscles; on the contrary this is rather desirable. The shoulders, the upper arm, and the head are to be fixed; after a stimulation of one or two seconds in duration, expiration should be practised by making pressure upon the abdominal wall, both laterally and from below upward. From time to time we suspend the electricity, and note whether respiration is taking place. It must be remembered that a long time may often pass before respiration occurs spontaneously. There are reports in literature which make it appear that, often after hours during which there was apparently no reaction to electric irritation, contractility returned and the patient was saved from imminent death.

Finally, another caution. Deficient diaphragm action, which is not rarely seen in alcoholics, and especially in patients convalescing from diphtheria, requires exceedingly careful treatment. Here the patient must refrain from all exertion, and his strength must be maintained by nutritious food and the administration of wine or other stimulant. This condition of debility and

paralysis of the most important nerves of respiration may be combated also by subcutaneous injections of strychnin, by cautious galvanic treatment of the neck, and by systematic faradization of the phrenic nerves.

9. PARALYSIS OF THE SUPRASCAPULAR AND POSTSCAPULAR NERVES

Paralyses rarely isolated are those of the *suprascapular* and *postscapular nerves.*

Hardly a dozen cases have been observed of paralysis of the first mentioned nerve, which innervates the supraspinatus and infraspinatus. Among the **etiologic factors** we recognize the pressure of heavy loads upon the shoulder, shock to the shoulder from a fall, torsion of the nerves of the brachial plexus, inflammation of nerves (neuritic conditions) in consequence of exposure to cold and, in a curious case observed by Bettmann, a congenital absence of these muscles.

The supraspinatus is less used in rotating the arm outwardly than the infraspinatus; as Duchenne emphasized, the muscle is an active ligament of the humeral joint, and when paralyzed the arm droops, hence throws additional labor on the actual lifter of the arm, the deltoid, which is most active in raising the arm. This produces a certain weakness of which the patient complains when he attempts to raise his arm. After it has been raised to a horizontal position, a visible effort as though to overcome some obstruction seems necessary before it can be further elevated to a vertical position.

The function of the infraspinatus which rotates the arm outwardly is more clearly developed; if the muscle is paralyzed this rotation becomes difficult but is never impossible.

Here the teres minor and the posterior portion of the deltoid may perhaps render assistance. Duchenne reported that paralysis of the infraspinatus prevented the movements for writing, embroidering, the drawing of long threads in sewing; I once observed this in a man who, although he experienced no difficulty in writing, was unable to draw a needle outward in sewing.

Symptoms.—Besides the functional disturbances just enumerated under the symptomatology of paralysis of the suprascapular nerve, ***atrophy*** in both *grooves of the shoulder-blade,* particularly the lower, is evident. This atrophy is less readily detected in the supraspinatus because of the intact trapezius; however, the infraspinatus fossa may look as if excavated and the bone may be directly palpated.

Among the *sensory disturbances* which may be experienced, pain is rarely absent at the onset of the disease and is usually of most importance, while objectively demonstrable disturbances of sensation are either insignificant or wholly absent.

The *electric contractility* of the paralyzed muscles is more or less decreased according to the severity of the affection. This is readily determined in the infraspinatus for the muscle may be directly irritated, and it may also be directly caused to contract from Erb's point; in severe lesions of the nerves, this reaction is, of course, entirely absent.

In the **diagnosis** of paralysis of the suprascapularis not only is pain in the region of the shoulder-blade significant but inspection of the shoulder-blades is also important, for here the excavation of the infraspinatus fossa may be at once discovered. Limitation of power to raise the arm freely, the difficulty of outward rotation of the arm, and the intactness of the other muscles of the shoulder-girdle facilitate the diagnosis of an isolated paralysis of the suprascapular nerve.

Although we have stated that isolated paralyses of the muscles of the shoulder-girdle are rare, it must nevertheless be noted that this form of paralysis is not infrequently combined with paralysis of other muscles of the shoulder. These muscles, as we shall see later, may be implicated in Duchenne-Erb's plexus paralysis, in the so-called obstetric paralyses of children, in neuritic processes which affect the nerve areas of the shoulder and arm, and in the so-called humero-scapular form of spinal progressive muscular atrophy; finally, a fact which Hoffmann has clearly demonstrated, these muscles are also implicated in syringomyelia, and this must be borne in mind (also the characteristic sensory disturbances of this affection) to protect us from error.

No severe disturbance is produced by paralysis of the nerves which innervate these muscles, and the patient of Bettmann's mentioned above was even said to be an athlete; nevertheless, the difficulty in raising the arm and the possible prevention of its outward rotation will make the patient seek professional aid in order to rid himself as soon as possible of these disabilities.

Treatment.—I shall not here discuss the *treatment* of a possible neuritis which causes such paralysis as we have been considering; the sequels of neuritis or trauma, producing paralysis of the muscles of the shoulder-girdle, are treated according to the fundamental principles of electro-therapy, with the addition of all procedures, such as massage, etc., which in similar cases have an abortive or curative influence on the disease.

Still more rare than paralysis of the suprascapular nerve is *isolated paralysis of the nervus dorsalis scapulæ.* The nerve supplies the levator scapulæ, the rhomboideus and the superior serratus. Of these muscles the posterior serratus draws the upper ribs upward and posteriorly, and during respiration assists the thorax to expand. The levator scapulæ, as its name indicates, raises the shoulder-blade; this acts simultaneously with the upper portion of the trapezius and the rhomboideus, the latter muscle drawing the shoulder-blade upward, backward, and inward toward the vertebral column.

Aside from the occasional implication of these muscles in the paralysis and wasting of progressive muscular atrophy, these muscles, or, rather, the dorsalis scapular nerve, is implicated in neuritic processes which attack the cervical nerves after infectious diseases, syphilis, etc. A paralysis of the serratus posterior can scarcely be determined externally. In paralysis of the levator scapulæ the shoulder can be only partially raised; if the rhomboidei are implicated, the maximal approximation of the shoulder-blade to the vertebral column is incomplete and, as Mollier has shown, the power of dropping the arm with force is reduced by the insufficient fixation of the scapula.

When the arm is at rest, paralysis of this nerve is denoted by the low position of the shoulder-blade and its remoteness from the vertebral column.

The *treatment* of such paralysis does not differ from that of other forms; considering its etiologic factors (syphilis), electro-therapy should first be tried.

10. PARALYSIS OF THE ANTERIOR THORACIC AND SUBSCAPULAR NERVES

Paralysis of these nerves alone is exceedingly rare.

The first mentioned nerves innervate the pectoralis major and minor, as well as the clavicular portion of the deltoid; these muscles are occasionally implicated in progressive muscular atrophy, in unilateral paralysis of a cerebral nature, and in injuries of the brachial plexus.

If they are paralyzed, particularly the pectoralis major, the arm cannot be thrust forward or pronated; and it is either difficult or impossible to place the hand upon the opposite shoulder. In atrophy which follows severe paralysis of the muscles in question, the infraclavicular fossa becomes so deep that the upper intercostal spaces may be seen and palpated.

We know that various muscles of the body may be congenitally absent. This fact has been previously mentioned in discussing paralysis of the trapezius. Absence of some of the muscles of the chest has also been noted.

An interesting fact which occasions surprise is that most authors who have seen these conditions emphasize the very slight motor disturbance produced by this congenital absence. The persons affected have developed into fine gymnasts and swimmers, which is perhaps accounted for by the vicarious function of muscles that have hypertrophied, such as the deltoid, the subclavius, and others. In the *differential diagnosis*, when deciding whether in the given case we are dealing with a congenital defect or with progressive muscular atrophy, the absence of muscles in other parts of the body, the absence or distortion of individual members (curvature of the fingers, absence of individual phalanges, the union of the fingers, webbed fingers, etc.) may be a guide as well as a carefully elicited history which may prove that the malformation existed from birth.

Without minutely discussing this condition, I must mention that competent authorities have expressed the opinion that besides the so-called congenital defects of individual muscles there is a rudimentary form of progressive muscular dystrophy.

Like the anterior thoracic, the subscapular nerves are rarely paralyzed alone. When this occurs, we have paralysis of the subscapular muscles, the teres major, the latissimus dorsi, and the inferior serratus.

Nearly all of these muscles rotate the arm inwardly; the latissimus dorsi also draws the arm backward, and lowers it after it has been raised. Paralysis of the previously mentioned muscles, by the action of their antagonists, especially the infraspinatus and teres minor, produces an outward rotation of the arm which decidedly hinders all those movements in which the arm is rotated inwardly.

An important etiologic factor of paralysis of the muscles under consideration is progressive muscular atrophy (perhaps also excessive use), which causes abnormal distention or torsion of the latissimus dorsi.

In paralysis of the latissimus dorsi alone, only a few cases of which have

been observed, we know from what we have learned regarding the function of the muscle that the arm can only with difficulty be thrown backward or lowered. As the upper border of this muscle covers the angle of the scapula and presses it upon the thorax, it is apparent that when paralyzed the lower angle of the shoulder-blade will escape from this fixed position and be drawn downward, a movement which is assisted by the action of the coracobrachialis, the pectoralis minor, and the short head of the biceps, all of which draw the shoulder-blade forward from the coracoid process.

11. PARALYSIS OF THE THORACICUS LONGUS NERVE (PARALYSIS OF THE SERRATUS)

Among the most interesting paralyses of the upper extremities are those of the *long thoracic nerve* which innervates the *serratus anticus major* (serratus magnus).

In the study of paralysis of these muscles, our views have lately been considerably modified and our knowledge supplemented by the investigations of many authors, among whom I must mention Mollier and Steinhausen. For instance, we know to-day that there may be not only complete paralysis of this muscle but paresis, that is, partial paralysis; the upper portion of the serratus is of unique interest inasmuch as it frequently does not become paralyzed. The upper fibers of the nerve most often escape the injury which damages those extending down to the supraclavicular fossa. In the discussion of the etiology this will be more minutely considered.

The muscle draws the shoulder-blade outward and forward, thus pressing it against the thorax, and draws the inner median border of the scapula away from the vertebral column. Here we observe (and this is evident in normal persons) that the serratus contracts long before the arm reaches a horizontal plane; the elevation of the arm is not due, as was formerly assumed, to the combined and equal action of the deltoid and serratus, but the latter combined with the trapezius supplies only about one-third of the necessary force, the other two-thirds being conveyed by the abductors of the arm.

The **etiologic factors** may be any injury which can possibly affect the long thoracic nerve in its course through the supraclavicular fossa. Besides those due to gross trauma (such as incised wounds, direct blows, gunshot injuries) the cases are interesting in which compression of the nerve occurs from the pressure of heavy loads carried upon the shoulder, or in which the nerve is unilaterally damaged by the continuous movement of the arm in some definite occupation (as in shoemakers, carpenters, farm hands, seamstresses, etc.) and by the contraction of the scalenus medius through which the nerve passes.

The stretching and torsion to which the nerve is subjected in some movements (as lifting down of heavy objects from a considerable height, torsion during gymnastic exercises, sudden spasmodic action of the muscle when a threatened fall is prevented by extending and bracing the arms) may occasionally produce paralysis of the serratus.

Other cases are known in which paralysis immediately followed wetting or chilling, infectious diseases (enteric fever, diphtheria, influenza, and in the puerperium), and severe rheumatic affections of the shoulder-joint. Paralysis of the nerve not due to wounds in the supraclavicular fossa but to trauma

affecting other regions (incised wounds of the axillary cavity or extirpation of diseased lymph-glands in this area) is rare. Besides other muscles of the shoulder-girdle, the serratus magnus is implicated in progressive muscular atrophy and in infantile spinal paralysis; paralysis of the serratus occasionally occurs in hysteria.

Symptoms.—In the cases of progressive muscular atrophy just mentioned, whether of spinal origin or occurring in the course of dystrophia muscularis progressiva, the paralysis of the muscles is usually bilateral; on the other hand, peripheral bilateral paralysis of the serratus seldom occurs.

In the majority of cases paralysis of the serratus causes no conspicuous disturbance of sensation; but at the onset of those paralyses which appear as neuritic inflammation, and also after so-called rheumatic damage, severe pain may arise from the implication of other sensory nerves belonging to the cervical plexus, and may subsequently radiate upward and downward.

There was a time when authors were not unanimous in regard to the position of the shoulder-blade during rest with the arm hanging down at the side of the body. The most diverse opinions were held as to the possibility of an oblique position of the scapula during rest; Berger was of the opinion that the medial border of the shoulder-blade extended obliquely from above, and outward, downward, and inward; other authors quite as distinguished maintained that the medial border was parallel with the vertebral column. According to the latest investigations these conditions alternate. A slight rocking motion is observed in quite isolated paralyses of the serratus; in any case, they fail to prove a special implication of the lower portion of the trapezius, which in fact is frequently affected simultaneously by the same damaging factors.

The condition is different when the position of the scapula is changed (with an intact trapezius); this approximates it to the vertebral column, and shows it to be higher than that of the normal side; even in rest the spinal border of the shoulder-blade, especially its lower angle, ranges somewhat posteriorly. The antagonists of the serratus, the rhomboidei, the trapezius, and the levator anguli scapulæ, are the ones which produce this higher position and the approximation to the vertebral column; the muscles which are inserted into the coracoid process, the pectoralis minor, the coracobrachialis and the biceps, draw the shoulder-blade forward and lift it out of the encircling upper border of the latissimus.

The conditions in paralysis of the serratus to some extent resemble those of paralysis of the facial nerve. When the patient is quiet some physicians are unable to discern anything abnormal, but they perceive the anomaly as soon as the patient makes any movement. I saw a young seamstress who, a few weeks before she presented herself at the Clinic, suffered from very severe pain in the right shoulder region and was soon afterward unable to raise her arm, or, when I requested her to do so, she could merely raise it to a horizontal plane. Instead of removing the internal border of the shoulder-blade from the vertebral column, as on the normal side, it was, on the contrary, approximated, and displaced by the rhomboideus and the trapezius. When I told her to move the arm from the frontal to the sagittal plane, she raised the internal border of the scapula and the lower angle from the thorax,

and the shoulder-blade projected posteriorly like a wing. The inner surface of the scapula could readily be palpated.

This position of the shoulder-blade in the sagittal plane of the horizontally raised arm is a pathognomonic sign of serratus paralysis. The muscle which, in normally performing this movement, holds the shoulder-blade to the thorax during the contraction of the deltoid, that is, the serratus, cannot perform its function because the antagonists alone, those muscles arising from the coracoid process as well as from the vertebral column, act upon the shoulder-blade.

Such conspicuous conditions as in the case just detailed are not always found; we know to-day that the arm may be raised above the horizontal, even to a vertical position. Here the muscles which act vicariously are usually hypertrophied, especially the middle and lower portions of the trapezius, also the infraspinatus and the posterior portions of the deltoid which compensate for the deficient or imperfect function of the serratus; at other times there is only partial paralysis of the serratus alone; a condition which Steinhausen observed in soldiers, and which may be present for years without the individuals in question or even the examining physician suspecting its existence.

How important the function of the serratus is for the complete elevation of the arm is shown by the fact that when the shoulder-blade is pressed against the thorax and simultaneously shoved downward and outward the raising of the arm, previously impossible, is found to be very easy.

That derangements of motion from serratus paralysis are disabling for the person in question is shown by the following: Upon raising the arm it cannot be projected forward with any power, hence the disease must in many cases be regarded as severe, and greatly reduces the patient's capacity for labor.

If neuritic pains, to which I have already called attention, have preceded, no characteristic disturbance of sensation is subsequently noted in serratus paralysis. In the peripheral paralyses which run their course with complete DeR (aside from the cases due to progressive muscular atrophy) we frequently perceive an atrophy of individual areas of the serratus on the diseased side which is absent in milder cases.

As in all peripheral paralyses, mild, medium and severe forms occur in which the electric contractility may be readily determined by an electro-diagnostic examination, and this should never be omitted.

A point in the **diagnosis** is the peculiar change in the position of the shoulder-blade on attempting to raise the arm to the frontal plane; this is even more marked in the sagittal. Contrasted with this, the change in the position of the scapula when the arm is at rest is difficult to recognize and unimportant. Since it has been recently demonstrated that mild and also partial paralyses of the serratus certainly occur, the patient's chest must be bared during the examination if we wish to avoid error.

We know that in partial paralysis, just as in normal conditions, the arm may be raised far above the horizontal plane, and we know, too, that when the function of the serratus is absent the middle and lower portions of the trapezius, the supra- and infraspinatus, and the deltoid vicariously perform these functions; hence, if the patient does not uncover his chest and shoul-

ders, the actual condition may be readily overlooked. We must bear in mind that paralysis of the serratus is often associated with a similar condition in the middle and lower areas of the trapezius, because the injury which affects the long thoracic nerve may also implicate the neighboring branches belonging to the cervical plexus. The history and a minute inspection of the affected thorax will soon show whether the paralysis of the serratus is merely an accompanying process affecting other muscles (as in progressive muscular atrophy from various causes) or whether we are in fact dealing with a *paralysis* of this nerve and muscle *alone*. It must be observed that this form of paralysis rarely occurs in children, it is much more frequent in men than in women, the working classes are particularly prone to be affected, and the right side, as will readily be understood, is more often implicated than the left.

Serratus paralysis can never be regarded as a mild affection, for it may seriously impair the function of the arm. We have stated that the vicarious action of other muscles of the shoulder may compensate for the loss of power in the serratus, and although the **prognosis** is by no means favorable, yet when electric examination shows only a slight or moderate degree of paralysis, recovery may occur in the near future.

Of course the prognosis is more unfavorable when electric examination reveals a profound paralysis or when the paralysis is an accompanying symptom of advancing *muscular* or *spinal* disease.

Treatment.—In the prophylaxis we must *prohibit* the immoderate use of the arm in any special way, also all exercises which cause prolonged tension of the nerves of the neck (gymnastic exercises). If, as often happens, there is pain in the early stages, this may be combated by hydrotherapy or antineuralgic remedies. In any case electro-therapeutic treatment should be instituted by the daily application for two or three minutes of the galvanic current (5 to 6 milliampères, the cathode in the supraclavicular fossa, the anode to the nape of the neck) and by labile stroking of the muscles in the axillary region with the cathode; finally the attempted stimulation of the nerves and muscles by the faradic current.

In bilateral paralysis of the muscles of the shoulder-girdle due to progressive muscular atrophy, the question whether operative procedures such as were once attempted by a prominent surgeon (who sewed together the shoulder-blades) will find their way into practice must be decided in the future.

In the course of this description I have several times referred to Mollier's [1] investigations of the statics and mechanics of the shoulder-girdle; for obvious reasons I must refrain from describing minutely the results which this investigator obtained. Yet I trust enough has been said to enable the practitioner in a given case to diagnosticate correctly a paralysis of the trapezius and serratus. Very important work has been done in this field. Mollier's original book is not of easy access to all, but there are more or less detailed references to it which give a synopsis of the most important points.

[1] S. Mollier, Jena, 1899, Fischer.

12. PARALYSIS OF THE AXILLARY (CIRCUMFLEX) NERVE (DELTOID PARALYSIS)

Isolated *paralyses* in the course of the *circumflex brachialis or axillary nerve* are not so frequent as paralysis of the radial nerve, nevertheless they are not rare. Thus, paresis and disturbance of function in that muscle most involved in paralysis of the axillary nerve, that is, the deltoid muscle, are the most frequent accompaniments or sequels of rheumatic or traumatic inflammation of the shoulder-joint. As in all paralyses of peripheral nerves, so also in those of the axillary nerve, injuries form the most important **etiologic factor**, whether the muscle be directly injured by a fall, a contusion or a blow, or whether the immediate cause be a blow, a gunshot wound or stab of the nerve itself, compression from fractures of the upper arm, or luxations of the upper arm. Like other peripheral nerves, the circumflexus brachii is attacked by neuritic processes after infectious diseases, intoxications, or constitutional diseases; for example, after enteric fever, influenza, lead poisoning, gout or diabetes. It may be readily understood that tumors in the axillary region or in the supraclavicular fossa may occasionally damage the nerve either alone or in association with other nerves of the arm.

Occasionally unilateral, more frequently bilateral, paralyses of the muscle occur after prolonged operations under anæsthesia during which one or both arms have for a long time been hyperextended and abducted; paralyses have occurred during sleep in consequence of one or both arms being extended above the head, or from the direct pressure of the body upon this muscle during sleep or in following an occupation (for instance, that of miners who lie upon the left side continuously while loosening ore, Seeligmüller).

I must also mention paralysis of the deltoid as an accompanying symptom of other neuritic or central processes (progressive muscular atrophy, syringomyelia, etc.) and the implication of this muscle in paralysis of the plexus or lead paralysis, in hemiplegia and paralysis of hysterical character.

Symptoms.—The *principal symptom* of axillary paralysis is paralysis of the deltoid muscle. This nerve gives off branches to the teres minor and the capsule of the shoulder-joint, also main branches such as the posterior cutaneous brachial nerve, but the lesions of these branches are so insignificant in comparison with paralysis of the deltoid that they scarcely merit description. In severe paralysis of the deltoid it is impossible to raise the arm or to lift it above the thorax, at least at first; the performance of this act necessitates many important movements of the arm which have become impossible. If the profound paralysis persists for a long time the muscle emaciates, the arm glides downward and is no longer fixed in the glenoid cavity.

If paralysis of the nerve is due to neuritic processes, pain may be present from the first (and this occurs also in paresis of the deltoid due to inflammation of the shoulder-joint). If an accurate examination be made in those cases in which paralysis is attributed to a lesion of the axillary nerve, we find a *more or less developed* hyperesthesia (or anesthesia) which has frequently been described; this consists in a cutaneous disturbance of sensation in the upper and posterior third of the upper arm. The patients themselves complain of it but little and the disturbance can only be detected by some effort.

The *anomalies of electric contractility* in the damaged nerve and muscle

are usually parallel with the severity of the lesion; thus electro-diagnostically we sometimes find slight, sometimes moderate, at other times severe forms of paralysis. In electric examination, those pareses of the deltoid which follow inflammation of the shoulder-joint usually show only a quantitative diminution in electric contractility. It must be remembered that even in severe paralysis the median bundles of this muscle often remain exempt because supplied by fibers of the anterior thoracic nerve. Two other facts must be mentioned which occasionally perplex those not familiar with them; first, that in exceptional cases lead paralysis (which otherwise first affects the extensor muscles of the hands and fingers) may immediately attack the deltoid without this muscle showing any change in electric reaction. Secondly, what is perhaps more rarely observed, in lead paralysis the activity of the muscle is apparently unimpaired, nevertheless electro-diagnostic examination reveals the existence of marked reactions of degeneration. But these most interesting conditions cannot at this point be minutely discussed.

When it is borne in mind that by a carefully elicited history and electric examination (even though only with the faradic current) deltoid paralysis has been diagnosticated in persons who applied for treatment of a joint inflammation or trauma, we should also note whether pain has preceded the onset of paralysis; and on considering the occupation of the patient (a worker in lead, etc.) it can scarcely be difficult to recognize the cause of the deltoid paralysis and with the aid of the symptomatology to make a correct diagnosis. If infectious diseases have preceded the paralysis, if the examination of the patient reveals symptoms of a chronic affection of the spinal cord (progressive muscular atrophy, syringomyelia, etc.), it is apparent in the given case that paralysis of the muscle is only one phenomenon of an extensive clinical picture.

Finally we must remember that hysterical paralyses after trauma are not very rare, even in men. By the retention of electric contractility these may be readily differentiated from severe paralysis which characteristically implicates the entire arm and shows sensory disturbance. They are also often marked by a surprisingly rapid disappearance of all the symptoms, and this again distinguishes them from paralysis of other origin.

The **prognosis** and **treatment** of deltoid paralysis vary according to the severity of the inflammation of the shoulder-joint or the trauma. The direct treatment of the nerve in muscular paralysis must be preceded by proper surgical measures. Only after inflammation has subsided, a dislocation has been reduced, or a fracture has been properly set, may we begin massage, baths, etc., combined with electro-therapeutic treatment. We need scarcely emphasize that any existing anemic conditions, diabetes, or lead poisoning, must not be overlooked in the treatment.

In conclusion, if rest or support is to be given to an inflamed joint the patient may be ordered to wear a brace; in other cases of longer existing paralysis, it is sometimes advisable, as various authors have recently recommended, for the patient to practise systematic exercises which increase the activity of the humero-scapular muscles (the supra- and infraspinatus, perhaps also the pectoral muscles, the trapezius, the coracobrachialis, etc.) and thus compensate for the loss in muscular power due to paralysis of the deltoid. Very satisfactory results have been obtained in this way.

13. PARALYSIS OF THE MUSCULOCUTANEOUS NERVE

Isolated *paralyses of the musculocutaneous nerves* (perforans Casserii) are rare occurrences. Fourteen cases are reported in literature. The **causes** of this paralysis are those with which we are already familiar: Compression of the upper arm, a fall upon the shoulder, tumors in the supraclavicular fossa, gunshot wounds, etc. In a few cases the cause may have been excessive use of the arm or a neuritis limited to this particular nerve. Everything previously stated applies also to isolated paralyses of this nerve. In association with injury of other nerves of the upper extremity, the musculo-cutaneous nerve is also frequently damaged; as we shall see, it plays an important rôle in the symptomatology of the so-called Duchenne-Erb paralysis.

Symptoms.—We shall first consider the symptoms due to *motor loss*, and here we find that in paralysis of this nerve the muscles which it innervates, the coracobrachialis, the biceps, and the brachialis internus, lose their function. The flaccid lower arm cannot be supinated, and can be flexed upon the upper arm only by the vicarious action of the supinator longus. Of course the brachialis internus is not wholly innervated by the musculo-cutaneous nerve; the musculo-spiral also sends it branches. In regard to the paralytic implication of the coracobrachialis, which is also supplied by this nerve, its intact state is particularly emphasized in the majority of the reports which mention it at all.

As the *sensory* portion of the perforans Casserii supplies the skin on the radial side and the internal and external surface of the forearm as far as the ball of the thumb and the palm of the hand with sensory fibers, injury of the nerve causes a subjective sense of numbness in these areas; in isolated cases the volar side of the thumb is also implicated.

If the damage to the nerve is severe, this is manifest by changes in the electric contractility; of course, these may be either simple, quantitative diminutions of contractility, moderate degrees, or well developed reactions of degeneration. It has been repeatedly observed that the portion of the brachialis internus innervated by the musculo-spiral nerve may react normally.

The *atrophy* of the affected muscles which always appears in severe paralysis gives to the upper arm of the patient a characteristic appearance: From the normal deltoid to the supinator longus the upper arm is emaciated owing to atrophy of the biceps and a large portion of the brachialis internus.

After what has been said of the disturbance of motion and sensation in these cases, the **diagnosis** of isolated musculo-cutaneous paralysis cannot be difficult. Even a superficial examination reveals to us whether the paralysis is independent and isolated or is a part of other processes; I refer particularly to the combined paralyses of shoulder and arm of the Duchenne-Erb type, which will later be described. Of course, neuritic processes may affect this nerve simultaneously with others in the upper extremities; and by a fracture of the humerus or luxation of this bone it may be injured simultaneously with other nerves.

I must call attention to the possibility that isolated paralysis of this nerve may be confounded with the muscular atrophy which accompanies Erb's juvenile dystrophia and particularly affects the flexors of the forearm. To avoid error we must bear in mind that in Erb's disease there is no history

of trauma; but, on the contrary, the affection exists from early youth, or at least for many years. The careful examination of the patient, the comparison of the atrophic muscles with normal or hypertrophied muscles in the same member, and the absence of sensory disturbances, etc., will soon reveal the true condition.

The **prognosis** of an isolated paralysis of the musculo-cutaneous nerve depends upon the severity of the original injury and the degree of electrical reaction; the treatment should be chiefly electric, and with perseverance on the part of the physician and the patient this will often lead to satisfactory results.

14. MUSCULOSPIRAL (RADIAL) PARALYSIS

In my experience the most frequent form of paralysis in the upper extremity is *musculo-spiral paralysis.* A common, if not the most common, cause is the pressure to which the arm is subjected during sleep because the head or the whole body of the sleeper rests upon the exit of the musculo-spiral or the area a little above this point while the arm rests on a hard surface. In rare cases this pressure acts lower down. The **clinical picture** may be best illustrated by quoting the history of a young workman who was attacked by right-sided musculo-spiral paralysis which occurred in the manner described.

When I directed the patient to extend both hands and at the same time the fingers he succeeded in accomplishing this only on the left side; the right hand as well as the fingers hung down in volar flexion. Neither hand nor fingers could be raised to a horizontal position, to say nothing of dorsal flexion; the thumb was flexed on its phalanges under the other fingers and could neither be abducted nor extended. On account of paralysis of the extensor carpi radialis longus and brevis and the extensor carpi ulnaris there was difficulty in abducting and adducting the hand, these movements being normally performed by the muscles just referred to with the combined action of the corresponding flexors. Here I must mention a fact which in my experience is frequently overlooked; namely, that the inability to extend the fingers is actually true only of the basal, the proximal phalanges, for when I passively extended them the patient at once extended the middle and distal phalanges, the interossei muscles which are supplied by the ulnar nerve and which control this movement not being implicated in the process.

The musculo-spiral nerve supplies both spinators as well as the extensors of the hand and fingers. When I told the patient to supinate the extended lower arm on the upper arm he could not perform this movement on account of paralysis of the supinator brevis.

That the supinator longus is involved in the paralysis is most evident when the patient flexes the arm and tries with all his might to keep it flexed while the physician attempts to extend it. During this procedure the contours of the supinator longus become very prominent in the normal arm of the patient, but not on the paralyzed side; we there feel only the flaccid belly of the muscle.

We must now investigate the function of another extensor of the arm which is innervated by the musculo-spiral nerve, the triceps. In the case under discussion this muscle escaped paralysis, and the patient could extend both the raised arm and the flexed lower arm. This is not surprising when we remember that the causative factor, pressure, was exerted below the point where branches are given off for the triceps, as is usually the case in radial paralysis. If, as sometimes occurs, there is compression on the trunk of the nerve in the axillary cavity, for example, from an insufficiently padded crutch, from gunshot wounds or stabs in the axillary cavity, or from high fractures or dislocations of the humerus, the triceps as well as other muscles innervated by the musculo-spiral nerve may be paralyzed; in rare cases the triceps may be the only muscle involved.

Before considering the history of this patient further, I must state that to-day even less than ever do we ascribe to exposure to cold a predominant rôle in the etiology of radial paralyses. The majority of those afflicted with radial paralysis belong to the working classes, and besides the etiologic factor of pressure upon the nerve a tendency to the abuse of alcohol here comes into question, the nerves of these persons being more susceptible to external damage than the nerves of non-alcoholics. Besides from pressure on the musculo-spiral nerve during sleep and its injury from fractures and dislocations of the upper arm, from blows, from gunshot wounds or stabs, pressure paralysis occurs in nurslings from their being too closely wrapped in clothes, in the arms of persons who are tightly held by the police when arresting them, from tight bandages after surgical operations, from too great constriction of the arm by an Esmarch bandage, and in some occupations in which the axillary cavity of the workman is subjected to too prolonged or abnormally great pressure. In this category probably belong a number of so-called anesthesia paralyses of the radial nerve which occur during prolonged anesthesia from pressure of the head of the abducted and hyperextended humerus; the nerve near its exit may be pressed against the hard surface of the operating table; or it may occur in other ways. We must also include among these musculo-spiral paralyses those cases which are caused by suspension, as in Motschukowski's method of treating tabes dorsalis. Here I must mention that in the course of tabes, apparently without external cause and spontaneously, paralysis of various nerves, among them the musculo-spiral, may occur, and these like other peripheral paralyses may improve while the underlying affection, tabes, persists.

We have already spoken of radial paralyses from various injuries of the upper arm, and we must now consider those which follow a fracture of the bones of the lower arm, especially of the head of the radius in adults, as well as that occasionally observed in the new-born.

It was noticed in my patient, as well as in many others, that radial paralysis due to pressure during sleep is only partial, for, as I have stated, the triceps is always exempt. This muscle is implicated in the paralysis only in the case of trauma of the axillary cavity or in injuries of the entire plexus.

In a patient who suffered from lead paralysis some of the muscles innervated by the musculo-spiral nerve characteristically escaped paralysis. Before giving his history, I shall briefly refer to the not very rare radial paralyses due to the subcutaneous injection of ether in the forearm. These paralyses in which the supinators and extensors of the hand are usually exempt are due to the deleterious effect of ether upon the nerve substance; we should therefore be extremely careful when injecting this substance into areas where there are superficial nerves which may be easily damaged.

Other poisons as well as lead may attack the musculo-spiral nerve either alone or conjointly with other nerves; among these I must mention arsenic, alcohol, carbonic oxid, carbon bisulphid, etc. Neuritis and polyneuritis, which frequently occur after various infectious diseases, such as enteric fever and pneumonia, also attack the musculo-spiral nerve, and abnormal distention of the nerve, as when one falls with the hand in volar flexion or when the nerve is pressed against the humerus by the forcible contraction of the triceps after an abnormal and sudden movement, may result in radial paralysis.

We must still consider the *sensory disturbances* in the case of the first patient, which also occurred in other patients attacked by the same disease. Usually these are very slight. They affect the area of the superficial radial nerve, and are manifested by abnormal sensations, numbness, formication, etc., rather than by demonstrable hyperesthesia; severe anesthesia occurs only in exceptional cases. Some of the skin on the ball of the thumb, the skin on the radial side of the root of the hand, and the middle hand as far as

the third metacarpal bone, also the skin over the proximal phalanges of the thumb and first two fingers and perhaps of the third on the radial side is implicated. In high lesions of the nerve, the disturbances of sensation are observed in the skin of the posterior aspect of the upper arm, and possibly down to the wrist on the extensor side of the forearm (the superficial cutaneous nerve, external branch).

It should be remembered that, even in severe musculo-spiral paralysis, there is sometimes no sensory disturbance, a condition which may perhaps be attributed to the extensive compensatory innervation, particularly in the nerves of the hands and fingers.

There are no marked *trophic disturbances* except a decided emaciation of the extensor muscles on the posterior surface of the lower arm in severe cases.

Among these we should perhaps reckon the swelling of the tendon sheaths of the extensors of the fingers on the back of the hand, which was first described by Gubler; this was conspicuous in the patient with lead poisoning whose history is soon to be related. Whether these are really trophic disturbances or whether the swelling is due to torsion of the tendons and their sheaths from the drooping of the paralyzed hand cannot be positively determined.

Before considering the electric reactions of musculo-spiral paralysis, which, with certain modifications, are also observed in all peripheral paralyses, I will refer to the history of a painter who was attacked by *lead paralysis* (*paralysis saturnina*).

Although the motility of the left hand of this patient was not quite intact, yet the right hand was chiefly affected. When I told him to flex dorsally the hand which was made into a fist he could do so but the hand deviated to the ulnar side. Both the radial extensors of the hand were at least paretic. In extending the fingers the second and third drooped below the horizontal plane, and the index and little fingers barely reached this line. On the other hand, the movements of the thumb were quite free. In regard to the active movements of the triceps and both supinators, it was observed that in this patient these muscles moved normally.

When we considered that this man was a painter (often working with lead paints), that he had not suffered from trauma, that the paralysis came on during the day while he was at work and not after sleep, that he was pale and cachectic with a lead line on his gums, even without an electric examination we readily diagnosticated a case of radial paralysis due to lead poisoning.

It is well known that radial paralysis is rarely the first symptom of lead poisoning. In the majority of such cases it is a later symptom, it attacks the patient years after he begins to work with lead, and other signs of lead intoxication (attacks of colic, pains in the limbs, especially in the joints, tremor, etc.) have usually preceded. Besides the occupation of the patient, we find a guide to the diagnosis in the almost invariable exemption of the supinators and the triceps, and particularly in the bilateral nature of the radial paralysis and the results of electric examination. Naturally, the bilateral character rarely develops to such an extent that the sides are affected with equal severity; as a rule, the extremity most used is the one most severely implicated.

When we examined diagnostically the electric contractility of the paralyzed radial region in the patients whose histories have been given, a marked difference in reaction could be perceived. In the patient with pressure paralysis from sleeping on his arm I obtained a good reaction with both currents at the point where the musculo-spiral nerve winds around the bone. Contractility, direct as well as indirect, was retained for both currents.

This was true also below the point of pressure; the conditions above this point will be described after discussing the electric contractility in the patient with lead poisoning.

Here it was soon apparent, both by direct and indirect irritation with the faradic current, that only those muscles could be stimulated which could still be actively moved, the paralyzed ones not responding at all. The indirect galvanic contractility of the paralyzed muscles had been lost, but, when directly stimulated by the galvanic current, they responded with a slow, sluggish contraction, especially marked to the anode, and soon showed the reaction of degeneration.

In the case of lead paralysis the electric contractility of the supinators and the triceps had not suffered.

I must now call attention to some very interesting points of this examination which are of differential-diagnostic significance.

All paralyses due to pressure on the musculo-spiral nerve during sleep are not slight; they may be very serious, although more rarely than from other severe traumata. It is easily understood that the electrical conditions then resemble those in severe paralysis of other nerve regions. When, therefore, as in the majority of cases of pressure paralyses occurring during sleep, contractility is retained at the point on the upper arm where the nerve curves around the bone, this is due to the fact that pressure has been operative upon the nerve above this point. If, as in rare cases, the lesion is below this point, even with mild paralysis no reaction is obtained. In both of these patients I demonstrated this by faradically irritating Erb's point in the supraclavicular fossa (this will subsequently be considered more minutely). In the patient with lead paralysis it then became evident that the supinator longus which had been exempt from paralysis, together with other muscles which need not now be mentioned, contracted powerfully while this did not occur in the patient with pressure paralysis.

Diagnosis.—After the foregoing it can scarcely be difficult to *diagnosticate musculo-spiral paralysis.* Paralyses due to trauma, to pressure during sleep, and to injections of ether, may readily be recognized from the history; the occupation of the patient (a worker in lead in its broadest sense), the implication of both radial areas, the results of electric exploration, the absence of paralysis in definite muscle groups, so characteristic of paralysis saturnina, will protect us from error. It must, however, be remembered that other metallic poisons such as arsenic and silver produce pathologic pictures which resemble lead poisoning, although more rarely; and in the given case there need be no difficulty in determining whether we are dealing with a musculo-spiral paralysis which is not isolated but is a part phenomenon of polyneuritis or of cerebral or spinal paralysis if we always make it a point to examine the patient systematically and thoroughly.

The **prognosis** of radial paralysis depends upon the cause and the possibility of removing the deleterious factor. This is evidently most true of injuries to the nerve from trauma.

Most paralyses from pressure during sleep prove to be mild, and to a certain extent are self-limited. They are somewhat relieved by electro-therapy; the better the electric contractility the more favorable, as a rule, the prognosis, though to this there are, of course, exceptions. But lead palsy is among the most severe forms of paralysis, and yields only slowly to treatment.

Musculo-spiral paralysis from pressure usually terminates in recovery without **treatment**, but the return of active motility may be decidedly hastened by proper electro-therapy, and if only a week be gained in restoring to health one who maintains his family by manual labor, this is important and well worth striving for. According to Remak, the activity of the nerve may be stimulated by stabile treatment with a not too strong faradic current, the cathode being at the point of pressure, the other electrode being used

to stroke the muscles in the paralyzed areas. I have several times treated in this way paralysis of the musculo-spiral nerve which had existed for several weeks, and recovery followed in a relatively short time.

Unyielding paralyses (in an electro-diagnostic sense) as well as lead paralysis, require such galvanic treatment for a long time, often for months. If possible the patient with lead poisoning should be removed from the deleterious influence of the poison, the alcoholic should be warned to break up his evil habits, etc.

If the paralysis is due to trauma, we must endeavor to remove by surgical means any obstruction caused by the fracture, dislocation, or compression of the nerve, etc., before beginning electrical treatment. If the nerve has been completely severed various methods, even *nerve suture,* must be resorted to.

I should like to call attention here to two points: First, that if nerve suture has been performed late, electrical treatment should not be too early abandoned; that is, the operation is not to be regarded as fruitless if motility is not restored as soon as was expected. Others, as well as I, have known active motility to return a year, and even more, after the patient was discharged from the hospital as incurable. Secondly, from my own experience and that of others, I think we must beware of performing a bloody operation too soon in musculo-spiral paralysis caused by a fracture of the humerus. Several of my patients for whom operation had been advised have recovered under a systematic and prolonged galvanic treatment.

I must also mention that in addition to neurotomy, primary and secondary nerve suture, etc., surgeons have lately added *transplantation of tendons* to their operations in paralysis, and have thereby increased the possibilities of cure even in apparently desperate cases. For various reasons, I cannot allow myself to discuss this interesting theme; I will merely state that sometimes by uniting the tendons of a normal and strong muscle, even although it be an antagonist, with the elongated or more frequently shortened tendon of a paralyzed muscle, satisfactory functional activity has been restored after all other remedies had proven fruitless. [Nerve transplantation promises better results in such cases.—ED.]

In conclusion, I will add that a special *prosthetic apparatus* has been invented (Heussner, Goldscheider) to improve or compensate for deficient extension of the fingers, with what results I am unable to state.

15. PARALYSIS OF THE ULNAR NERVE

Etiology.—Although not as frequent as musculo-spiral paralysis, *paralysis of the ulnar nerve* is by no means rare. Pressure or compression of the nerve during sleep is not so frequently a cause as in radial paralysis. Because of the protected position of the ulnar nerve in comparison with other nerves some authors question the possibility of its paralysis by direct pressure. On the contrary, they attribute it to the pressure of the head of the humerus on the nerve in the axillary cavity during sleep with the arms extended upward and backward. If during anesthesia this position of the arm is long maintained, *anesthesia paralysis* of the ulnar nerve may readily occur; this has been pointed out by Braun.

Duchenne has called attention to *occupation paralysis* in the course of the

ulnar nerve; namely, paralysis of the nerve occurring in persons whose occupations force them to use their elbows as a brace while working upon a hard under surface. Many authors, of whom I shall mention only Leudet, Ballet, Bruns, Menz, Gessler, Salomonson, have observed this in glass-workers, wood-carvers, telegraphers, and diamond-cutters, also in bakers, who knead the dough for bread with the ulnar side of the right hand and forearm, and thus constantly exert pressure upon a hard surface (Huet and Guillain). This has also been reported by the earlier authors (Cöster and Remak, as well as by myself) who noted, particularly in laundresses, cigar makers, etc., that the small muscles of the hand became weak and atrophic. In these cases the etiologic factor of occupation neurosis was probably the direct pressure exerted upon the small muscles or upon the nerve.

In a certain sense we may perhaps add to these the not infrequent paralyses of the ulnar nerve on the left side which are seen in bicyclers, and are caused by exposure of the ulnar nerve at the wrist-joint to pressure between the handle of the bicycle and the pisiform bone; this has been described by Destot and myself.

Another and less common cause of paralysis of the nerve of the elbow is compression from the use of an Esmarch bandage, from handcuffs, or from the pressure of a crutch in the axillary cavities. More rarely tumors seated at or near the nerve, or syphilitic affections, will produce this paralysis. Among the most common causes are injuries to the bones of the shoulder and the elbow-joint. Here I must refer to conditions long known but which have only recently attracted special attention; namely, ulnar paralysis which occurred years afer a preceding injury to the elbow, and also the so-called *luxation of the ulnar nerve* which may be either congenital or acquired, this nerve being normally protected (in its groove between the internal condyle and the olecranon) from the danger to which the elbow-joint, being without this natural protection, is exposed.

This nerve is often injured by blows, stabs, or cuts at the wrist, more rarely by isolated injuries of the brachial plexus; I once saw paralysis develop after a subcutaneous injection of ether clumsily given in the area of this nerve on the inner side of the upper arm.

Inflammatory degeneration in the region of the ulnar nerve is not rare after infectious diseases, especially after enteric fever; at the same time I must state that the peripheral paralyses occurring during the puerperium frequently implicate the nerves of the hand, usually the ulnar nerve. When I state that ulnar paralysis is observed in leprosy, and that the small muscles of the hand are implicated in many cases of lead poisoning, and, as I have seen, even without a primary radial paralysis, all of the important factors which may lead to ulnar paralysis have been enumerated, and it need only be added that paralysis in the course of this nerve, especially in the small muscles of the hand, is also met with in tabes, in syringomyelia, and very often in progressive muscular atrophy.

Symptomatology.—In complete paralysis of the ulnar nerve the use of the hand for the execution of finer movements, as in writing, etc., is markedly impaired. Flexion of the wrist and the last three fingers is incomplete. The little finger is motionless. The interossei muscle, the separators of the fingers, the muscles which draw them toward each other, and the adductor

pollicis are innervated by the ulnar nerve; since the proximal phalanges of the fingers are flexed by the interossei, the middle and terminal phalanges, however, being extended, in injury of the nerve above the wrist-joint (a frequent occurrence), and with a normal condition of the upper portion of the ulnar which supplies the branches for the long flexors of the finger, the characteristic *claw* or *talon hand* (main en griffe) develops, a peculiar position of the affected hand and fingers with which we are familiar from Duchenne's description. By paralysis of the interossei which flex the basal phalanx and extend the middle and terminal phalanges, and with intact flexors of the middle and terminal phalanges, as well as the extensors of the basal finger-joints (the extensor communis digitorum which is innervated by the radial nerve) this resemblance of the hand to the claw of a bird is particularly marked in extreme atrophy of the interossei muscles. There is decided *disturbance of sensation* in the hollow of the hand, most marked in the little finger, especially in the ball of it, as well as in those parts of the third finger and the palm of the hand which are situated on the ulnar side of an imaginary line drawn through the middle of the ring finger.

On the dorsum of the hand the entire little finger is implicated, the ulnar side of the third, the proximal phalanges of the second and third upon the ulnar side, and finally the region of the fourth and fifth metacarpal bones.

Among *trophic disturbances* I have already mentioned atrophy of the interossei and hypothenar muscles which is often extreme and gives a sunken appearance to the metacarpal spaces. With or without the formation of vesicles ulceration appears in the cutaneous areas more or less deprived of sensation, particularly by the action of a low temperature, and in a few cases contracture in the palmar fascia (Dupuytren's contracture) has been regarded as a trophic disturbance. In a few cases, hyperplasia of the subcutaneous cellular tissue of the palm of the hand has been noted (Löwenfeld), and has been regarded as a trophic disturbance in the course of the damaged ulnar nerve.

In ulnar paralysis *electric contractility* reveals nothing special; according to the severity of the lesion we are dealing with a slight, a moderate, or a severe form of peripheral paralysis.

As to **prognosis**, little can be stated except that the more severe the trauma which has affected the nerve the more difficult the cure. If the nerve is compressed, and it is possible to relieve this by a surgical operation and restore its normal function, the prognosis is favorable; the outlook is least promising when we are dealing not with a peripheral lesion but with the implication of the nerve in a pathologic central process, which is the case in progressive muscular atrophy and in syringomyelia.

Considering the deformity which it produces the *diagnosis* of paralysis of the ulnar nerve occasions no perplexity. If there are signs which point to a possible trauma (fracture of bones, dislocations, wounds, etc.), the cause of the affection is readily recognized, and appropriate treatment should be instituted. If these conditions are not present, a carefully elicited history may enable us to determine in the given case whether any infectious disease has preceded or whether we are dealing with an occupation paralysis, and a thorough examination of the patient will perhaps reveal that the paralysis

is but the symptom of a grave derangement of the nervous system, for example, syringomyelia or progressive muscular atrophy.

In regard to **treatment**, paralyses due to the fracture of bones, dislocation, etc., indicate surgical measures by which the damaged nerve may be released from the pressure of any bone, foreign body or cicatrix; later, even when the nerve has been severed by trauma and reunited by suture, electricity may be employed, and this treatment is also applicable in those cases in which the paralysis may be attributed to a neuritic affection. no matter what the antecedent cause.

16. PARALYSIS OF THE MEDIAN NERVE

Before discussing the pathology of *paralysis of the median nerve,* which is less frequent than radial paralysis yet by no means rare, I desire to give the history of two patients.

Case 1.—A girl, who had fallen upon glass a few weeks prior to her appearance in the Clinic, received a wound two or three fingerbreadths above the right wrist-joint and several centimeters in length. This severed the median nerve; the ulnar nerve was uninjured. When the hand was examined it was remarked that the index and middle fingers were smooth, glistening, and cool to the touch; in contrast with the other fingers they never perspired. This smooth, glistening appearance is an illustration of what is known as "glossy skin." The cicatrix and the areas above it were insensitive to pressure; but pressure upon the nerve below the cicatrix caused a painful sensation in the palm of the hand at the base of the first and second fingers. The sensory disturbances were then investigated. Test with a needle showed profound anesthesia on the volar side of the middle and distal phalanges of the first and second fingers and upon the radial side of the third finger as well as in the distal phalanx of the thumb; marked hyperesthesia was found in the basal phalanges of the thumb and first two fingers and on the radial side of the third, and over the ball of the thumb and the adjacent areas of the palm of the hand. At the cushion of the middle finger a cicatrix from a burn was noted; and this injury had when received *not been felt* by the patient, but was only *perceived later.*

The disturbances of sensation upon *the back* of the hand are extremely interesting, and still not sufficiently understood. Absolute anesthesia was found over the entire distal and middle phalanges of the first and second fingers, and on the radial side of the third; the sensibility of the skin over the basal phalanges on the dorsal side of these fingers was but little altered; the thumb and back of the hand were in this respect absolutely normal.

It was surprising to find that but little *disturbance of motility* could be determined. The first, second and third fingers as well as the distal phalanx of the thumb could be flexed and the hand clenched into a fist. Careful investigation revealed a slight flattening of the ball of the thumb in the region of the abductor pollicis brevis; there was no marked atrophy of the muscles of the ball of the thumb.

All movements of the thumb were free; it could be brought into apposition with all the fingers without flexing the distal phalanx. Articles could be held between the distal phalanges of the thumb and index finger, although not with as firm a grasp as upon the normal left side.

The *results of the electrical examination* formed an apparently remarkable contrast to the generally well-retained motility. Unlike the conditions on the normal left side, no effect could be produced upon the affected right side even with the strongest faradic currents, neither with direct nor with indirect irritation; this was true also of indirect galvanic irritation. The musculature of the ball of the right thumb, however, reacted slowly and sluggishly to direct irritation with a current which upon the normal side had no effect whatsoever, the anodal closure contractions being equal to the cathodal, contrary to the normal.

Case 2.—I will now relate the case of a patient who had been married six years, had borne three children, and, according to report, during every labor had complained of tearing pain in the shoulder. After the last labor, during which the patient suffered from pain in the right shoulder, she noticed a gradually increasing emaciation of the right hand. This affected the interosseous spaces (especially the first) and the ball of the thumb; along the entire ulnar border of the right lower arm and in the little finger there was numbness. The power to spread the fingers and draw them together again was decidedly limited, as well as movements of the thumb. Electric examination showed a moderate reaction of degeneration in the area of both nerves.

The history of these two patients discloses the **etiological factors** which most frequently produce paralysis of the median nerve—trauma and degenerative neuritis. In my experience, incised wounds of the nerve immediately above the wrist-joint, most often by a fall upon glass, etc., are the common causes of paralysis either of the median nerve alone or combined with the ulnar nerve. Paralysis also occurs after gunshot wounds, stabs, or operations on the upper arm, and somewhat more frequently in consequence of fractures and dislocations.

While tumors of the nerve have several times been described as causes of paralysis of the median, paralysis of the nerve from the pressure of a crutch, pressure during sleep, or after anesthesia is a rare occurrence. Paralysis of the median nerve after dislocation of the wrist-joint (Gowers) has been only occasionally reported; E. Weber published the case of a young man in whom the sensory branches of the median nerve were affected while dancing because of a marked hyperflexion and pronation of the hand and lower arm. As with other nerves of the arm, the pressure of an Esmarch bandage or any other compression may paralyze this nerve either alone or combined with other nerves of the arm; for instance, when handcuffs are applied just above the wrist-joint.

The second case belongs to the form not infrequently seen after ***infectious diseases***; alone or in association with the ulnar nerve the median nerve may be affected during the puerperium, as was first stated by Kast and subsequently emphasized by Möbius and other authors; such cases have been observed after variola and enteric fever, and lately by Eulenburg and myself after influenza.

A degenerative neuritis is probably the cause or consequence of overexertion in an occupation; the so-called *professional* or ***occupation paralysis*** occurs in laundresses, carpet beaters, carpenters, locksmiths, cigar and cigarette makers, perhaps in dentists (in filling teeth) and in those persons who milk many cows (milker's paralysis).

I must also call attention to the fact that paralysis of the median nerve may be an accompanying symptom of a widely distributed neuritis in paralysis due to lead, to arsenic, or occasionally to phosphorus and carbon bisulphid.

I omitted to mention among the so-called occupation paralyses a few cases of paralysis of the thumb in drummers. Bruns first called attention to this isolated affection of the flexor pollicis longus. Later investigations (v. Zanders, Düms, Steidel) have proven that in such cases the extensor pollicis longus is just as frequently implicated, and that the condition is often not one of nerve or muscle paralysis but is due to a laceration of the tendons of these muscles.

In conclusion I must state that atrophy and paresis of the small muscles

of the hand, therefore also in the distribution of the median nerve, are observed in spinal progressive muscular atrophy and in syringomyelia.

The **symptomatology** of paralysis of the median nerve varies according to the seat of the lesion. If, as is often the case, the nerve is injured above the wrist-joint, and provided the tendons of the long flexors of the fingers are not also injured, the muscles of the ball of the thumb only are paralyzed. Flexion and apposition of the thumb to the fingers becomes impossible. The extended thumb, adducted from the fingers and with its normal position of apposition lost, shows its volar side anteriorly. The hand loses the characteristics of the human hand, becomes flat, and resembles the *claw* or *paw* of the lower *animals,* for instance the *monkey.*

When the injury to the median nerve is higher up, the conditions differ. It is well known that, besides supplying both pronators, this nerve also innervates the palmaris longus, flexor digitorum sublimis, the radial portions of the profundus, the flexor pollicis longus, the three first lumbricales, and, except the abdùctor pollicis which is innervated by the ulnar nerve, it supplies all the other small muscles of the ball of the thumb.

It is evident, therefore, that if the lesion is high, pronation of the hand is imperfect or impossible, flexion of the hand also is incomplete, and when attempted the hand is drawn medially by the flexor carpi ulnaris which acts alone. As the flexor digitorum sublimus flexes the middle phalanges of all the fingers, the digital phalanges of the second and index fingers, and the flexor digitorum profundus which is also innervated by this nerve, these movements are impossible if the lesion is high. Of the altered movements of the first three lumbricales after injuries to the median nerve we have little clinical knowledge. Flexion of the basal phalanges of the four fingers (not the thumb) is produced by the activity of the interossei. Flexion of the distal phalanges of the last two fingers depends upon the flexor digitorum profundus, which here receives its innervation through the ulnar nerve. If we add to these disturbances the lost power of apposition in the thumb, further details of the loss of power in the hand and fingers from paralysis of the median nerve are unnecessary.

Disturbances in sensation following lesions of the median nerve were referred to in the history of the first patient; it was also emphasized that the dorsum of the hand, the nail, the middle phalanx of the index and second fingers, and the radial side of the same members and of the third finger receive sensory fibers from the median nerve. In some persons the dorsal side of the distal phalanx of the thumb appears to receive its sensory fibers from the median nerve. The distribution of the sensory fibers is more easily traced in the palm of the hand: The most lateral parts of the ball of the thumb receive their sensory innervation from branches of the superficial radial and musculo-cutaneous nerves; the bulk of the skin covering the thenar eminence and the vola manus is innervated by sensory fibers from the median, as well as the volar side of the thumb, of the index and second fingers, and the radial side of the third finger.

These fibers are not implicated to the same extent in all cases of injury to the median nerve, for the lesion may be slight; the disturbance of sensation just described is more or less extensive according to the severity of the injury.

Among the trophic and vasomotor disturbances which occur in almost all severe peripheral paralyses we here find a flattening of the ball of the thumb, and in lesions higher up a flattening of the radial side of the forearm. Hand and fingers are cool to the touch and often show a livid discoloration; the skin is readily injured and reacts even to slight injuries by the formation of vesicles and ulcers. As was seen in one of my patients the skin of the finger is smooth, glistening and insensitive; upon it herpetic and pemphigus-like vesicles readily appear. Marked trophic disturbances in the nails are not uncommon; these become irregular, curved or fissured, but, so far as I have been able to observe, their growth is but little affected.

The *changes in electric contractility* vary according to the severity of the injury; they may be slight, moderate or extensive. To avoid repetition, I shall not enumerate these conditions, but I must call attention to one peculiarity observed very frequently in paralysis of the median nerve, and which was clearly demonstrated in the case of the first patient.

While in peripheral paralysis with a severe implication of the motor nerve fibers the sensory fibers are but slightly or not at all affected, I have observed (and I note from literature that other authors also have noted) that in median paralysis these relations are often reversed. Contrary to the usual condition very decided sensory disturbances may appear after an apparently slight implication of active motility; this was evident from the history of the first patient. This remarkable condition may be explained by the anastomoses (which have been understood for some time but have recently been more clearly demonstrated by Frohse) which take place between the median and the deep branches of the ulnar in the hand. At all events we may assume that this anastomosis does not take place in all persons, at least not to the same extent, so that in the majority of cases median paralysis runs its course according to the ordinary diagram of peripheral paralysis.

After the foregoing it can hardly be difficult to diagnosticate median paralysis, especially if in the examination of the patient we elicit a history of trauma. It need only be premised that paralysis of the muscles supplied by the median nerve may be an accompanying condition of cerebral, and even more frequently of spinal, disease; I need merely refer to syringomyelia, to progressive spinal muscular atrophy, and to similar processes. The prognosis depends upon the severity of the lesion. Compression is much more favorable than complete severance. Electro-diagnostic examination will clear the prognosis.

The **treatment** should include the use of electricity, but it must be remembered that, as in all injuries produced by complete severance of the nerve, this should be employed only after the divided nerve-ends have been united by suture.

17. COMBINED PARALYSES OF THE NERVES OF THE SHOULDER AND ARM

Having studied paralysis of the individual nerves of the arm, we must still discuss those paralyses which attack several nerves of the upper extremity simultaneously. These *combined paralyses of the nerves of the shoulder and arm* develop from different causes. Usually they are due to injuries

of the bones or joints which produce a lesion of the nerve: Fracture of the head of the humerus, of the shaft of the bone, of the clavicle, or dislocation of the head of the humerus. Next we consider injuries made by abnormal pressure in the axillary cavity (pressure of the crutch) or by a too great constriction of the arm during operations (with Esmarch's bandage), or by constriction from ropes around the upper arm. Paralysis which occurs after prolonged anesthesia is specially interesting. Later we shall more minutely discuss the forms of paralysis designated by Braun *anesthesia paralysis*; we may here state that such paralysis need by no means implicate all the nerves of the upper extremity, but that besides complete paralysis there may be every possible degree according to the injury which the nerves have sustained.

An excellent example of combined paralysis of the shoulder and arm occurred in the case of a man, aged 28, who sustained a fall, his right shoulder striking the ground. He at once observed that he could not move his arm. It was rotated inwardly, could be extended at the elbow-joint, but could not be elevated to the frontal plane and only a few degrees in the sagittal plane; outward rotation of the arm was impossible. With the lower arm supine the patient could not flex it toward the upper arm; actual supination of the lower arm was impossible. The right shoulder appeared flattened, as well as the supra- and infrascapular fossæ. On the anterior and outer side of the deltoid muscle there was an area 4 to 5 centimeters long and 2 centimeters in breadth which, like the posterior surface of the thumb and the extensor surface of the lower arm along the radius, was somewhat less sensitive to touch than the corresponding region of the left lower arm.

The electric contractility showed a moderate reaction of degeneration, for muscular contraction occurred quite readily as compared with the left side on direct and indirect faradic irritation with but a slightly increased current; muscular contraction was also sharp and lightning-like with indirect galvanic irritation, while in the biceps and the posterior and external bundle of the deltoid, with direct galvanic irritation the contractions were sluggish and the CaOC which is otherwise difficult to demonstrate could be readily produced. Before entering upon the pathology of the form of paralysis, of which this patient furnished an almost classic example, I must call attention to a symptom which in this case was readily demonstrated. I performed the following little experiment: I passively flexed the lower arm of the patient upon the upper arm, and asked him to keep the arm flexed while I attempted to extend it; it was evident that I could easily do this; not only did the true flexors of the lower arm, the biceps and the brachialis internus, fail to contract, but during this process the supinator longus on the right side remained flaccid while in the left normal arm it contracted distinctly and energetically. Was it not remarkable then that the patient could flex his arm? It was noted that he slightly pronated the lower arm while extending the hand upward; if the flexor and ulnar side of the right lower arm was grasped with the fingers a strong contraction of the muscles from the internal condyle, especially of the pronator teres, the flexor carpi radialis and the ulnaris, was distinctly felt; and the tendon of the palmaris longus became very tense. If pressure was made with the fingers alongside the quite flaccid supinator longus, this was followed by contraction of the extensor carpi radialis. It was observed, too, that it was unnecessary to flex the hand dorsally in order to flex the lower arm, for this could easily be done with the hand completely flexed.

Such vicarious functions of the flexors of the hand and fingers (more rarely of the extensors) are not always so evident in paralysis of the flexors of the lower arm, hence I took occasion to mention this case which I had observed years ago. To complete the history of this patient it is only necessary to add that the oculo-pupillary fibers (of the sympathetic) were intact as well as the regions supplied by the median and ulnar nerves; also that with the exception of the supinators the radial region was normal, as was also

evident from the ease and promptness with which movements of these areas were executed.

This form of paralysis, although known before Erb described it, was, nevertheless, only then fully appreciated. In these paralyses the deltoid, the biceps, the brachialis internus, the supinator longus (sometimes also the supinator brevis), also, although not in all cases, the supra- and infraspinatus are simultaneously paralyzed while other nerve regions are usually exempt.

The question arises, Where must the damaging factor fall to produce such a *plexus paralysis?* Naturally, at that point in the plexus where the motor tracts for the involved muscles have not yet divided into the different nerve trunks. Erb showed that the fifth and sixth cervical nerves in particular assist in the formation of the branches of the brachial plexus here under consideration, and he indicated the supraclavicular fossa as the point in which, under normal conditions, the paralyzed muscles may be induced to contract (Erb's point).

According to Hödemaker this point is found by drawing a line from the sterno-clavicular joint to the spinous process of the seventh cervical vertebra, somewhat anteriorly to and 1.5 cm. from the border of the trapezius muscle.

Some points in the *etiology* have already been mentioned: In addition to trauma of the supraclavicular fossa, a fall upon the shoulder, operative wounds in this region, blows, gunshot wounds, or stab wounds, this form of paralysis may be caused by tumors or neuritic processes in this area as in the case of tumors of the cervical vertebræ.

Following Duchenne, Erb and still later many other observers called attention to the paralyses which not infrequently occur in the new-born, paralyses which, according to Duchenne, are designated as "*paralysies obstétricales infantiles due membre supérieur sans complication*"; our knowledge of these forms of paralysis has been added to and elucidated by the investigations of more recent authors. The following is the history of such a case:

A child, aged 5 weeks, delivered without instruments. The labor was tedious owing to the non-appearance of the shoulders, and paralysis was noted immediately after birth and still existed at the time the infant was examined. It was evident that the left upper extremity rotated internally (from paralysis of the supra- and infraspinatus), being extended upon the thorax. In contrast with the activity of the right upper extremity, the left as far as the arm and forearm were concerned was immotile, only the hand and fingers moving. Testing the electric contractility in such small children is, as is well known, extremely difficult, but in this case it was enormously diminished, especially in the deltoid, the flexors of the lower arm, and the supinator longus.

It is exceedingly interesting to review the various theories which have been proposed to explain this Duchenne-Erb paralysis of the plexus of the arm. The etiology of these paralyses in the adult is easy to understand if trauma has affected the supraclavicular region. But concerning those which follow a blow upon the shoulder, the opinions of authors are wide asunder. While some maintain that the clavicle is raised and approximates the vertebral column in consequence of the forcible adduction of the shoulder which so presses upon the cervical vertebral column as to affect the middle portion of the transverse processes of the sixth and seventh cervical vertebræ and to

injure the nerves located there, others reject this explanation, and believe that the injury is done between the clavicle and the first rib, while still others take a middle ground and admit both possibilities.

What I have previously stated is particularly true of the traumatic paralyses of adults during anesthesia.

In regard to such paralysis in the new-born, Erb first called attention to the fact that in Prague, according to custom, the fingers of the obstetrician encircle the neck of the child and pull with such force that this region of the supraclavicular fossa is subjected to great pressure. At other times the fingers of the obstetrician hooked in the axillary cavity of the child may produce compression, or the condition may be due to torsion and pressure when in a breech presentation the arms of the child are forcibly pulled. Recent investigations made upon the cadavers of children, indicate that it is not so much the direct pressure as torsion of the fifth and sixth nerve roots which causes this paralysis, and also ascribe to this etiologic factor so-called anesthesia paralysis.

The majority of paralyses occurring *intra partum* are of the Duchenne-Erb type; the internal rotation of the arm is invariably attributed to paralysis of the outward rotators, particularly of the infraspinatus.

Aside from the immediate damage to the nerves by injuries to the bone or joint, we occasionally find paralyses which implicate the median and ulnar nerves yet differ from those ordinarily observed.

There may be profound paralysis which implicates the oculo-pupillary fibers. It is characteristic, however, that in a case of paralysis of the new-born recently described by Jolly in which there was a bilateral paralysis of the pectoralis major, of the latissimus dorsi, and the triceps, and in which nearly all of the muscles of the forearm and of the hands were implicated, the function of both deltoids and biceps was retained. Both arms were raised, the forearms strongly flexed; the hands drooped and, as Jolly expresses it, were in a pump-handle position. In this case torsion had occurred between the sixth and seventh cervical vertebræ and had damaged the point of exit of the sixth and seventh pairs of roots.

Before passing to another equally important and interesting form of plexus paralysis, I must call attention to the fact that during labor, besides injury to the nerve, other lesions may be sustained which occasionally result in the paralysis of one or several nerves in the arm of the child. I refer to injuries of the bones and joints such as are not infrequent in difficult deliveries. Paralyses of this kind must receive the same consideration as traumatic paralyses of adults, for they are due to similar causes.

We will now discuss those paralyses which affect in a typical manner only one or another of the muscles ordinarily paralyzed in common with other muscles. It has been observed that sometimes, in trauma or neuritic affections of the plexus, not all but merely a few of the muscles implicated are paralyzed, or even only one, yet minute investigation has shown the point of origin of this apparently isolated paralysis to be in the plexus. I mention this solely for the sake of completeness, since such peculiar conditions as this are rarely met with.

The *bilateral paralyses of the Duchenne-Erb type* need more minute investigation. These occur bilaterally in anesthesia if the arms are drawn upward and back for a considerable time during prolonged operations. Cases of this kind have been reported by Braun, Jolly, Mally and others.

A number of *neuritic plexus paralyses* have become known in the course of years; here infectious diseases such as pneumonia and influenza, or other affections such as anemia, perityphlitis, tuberculosis, etc., play a causative rôle. Bilateral plexus paralysis has also been observed in persons whose occupations forced them to carry heavy loads on the shoulders (coal, stones), the supraclavicular fossa thus being subjected to heavy pressure for weeks or even years.

For the sake of completeness I must mention that such paralysis is now and then produced by excessive use of the muscles of the upper extremities, also that torsion of the arm or gymnastic exercises which abnormally stretch the arms may be the cause. These paralyses have also been observed after hemorrhage in the supraclavicular fossa (in alcoholics) and in isolated cases from gout. A case reported by Raymond is unique from the fact that there was merely an affection of the sensory plexus roots without implication of the motor ones; this rare condition was probably due to a tuberculous pachymeningitis.

So far we have considered only plexus paralysis of the upper roots. There are also forms which have long been known but have only lately been explained by the studies of Déjerine-Klumpke who considered these to be *paralysis of the lower roots of the plexus.* In Klumpke's paralysis the regions of the median and ulnar nerves are chiefly implicated; these show paralysis and atrophy of the musculature of the ball of the thumb and the little fingers, also of the interossei; accompanying this are sensory, especially *oculo-pupillary,* disturbances, manifested by the narrowing of the palpebral fissure of the affected eye, the retraction of the eyeball, and the contraction of the corresponding pupil (myosis). These signs of disturbance in the function of the sympathetic indicate an injury to the communicating branch of the first dorsal nerve, as Klumpke proved by experiments upon animals, and Oppenheim in man by an extremely interesting case.

Etiologic factors for this paralysis may be wounds in the affected region, but it is usually caused by tumors which, starting from the lungs or from the vertebræ, damage the lower cervical and upper dorsal vertebræ and the nerve roots between them.

Upper or lower plexus paralysis of this kind may not always exhibit from the onset a characteristic type. A paralysis which immediately affects all the fibers of the plexus may yield in the course of weeks or months so that all that remains of the once complete paralysis is the affection of the upper or lower plexus. This is true not only of the traumatic cases, which are at once recognized, but also of those which occur *intra partum*; we know to-day from a series of observations, Heubner's, for instance, that plexus paralysis of Klumpke's type is seen in children and is due to caries of the vertebræ or tumors of the vertebræ.

There are, of course, exceptional cases which deviate from the types here described. Plexus paralyses implicate the median and ulnar regions without paralytic phenomena on the part of the sympathetic; cases have been seen in which the symptoms of Erb's paralysis were combined with those of sympathetic involvement; these, however, may be due to the fact that the tumor which compresses the upper roots presses also upon a branch of the sympathetic in the neck; and here and there are other deviations which, in the given case, must be especially investigated.

I shall not touch upon the disturbances of sensation, nor the vasomotor and trophic symptoms in paralysis of this kind, nor shall I describe the electrical reactions which vary according to the severity of the individual case, since these do not differ from those ordinarily observed in isolated paralysis of this nature.

The *diagnosis* of these cases usually occasions no perplexity if there is a demonstrable trauma, if close inspection reveals a tumor in the upper or lower part of the supraclavicular fossa, or if there are caries of the upper or lower cervical vertebræ. If infectious disease has preceded the appearance of plexus paralysis, or there has been severe pain, neuritis must be considered. We can easily decide from the history whether in the given case the paralysis was obstetrical.

We must be particularly cautious in the diagnosis of paralyses of this plexus which occur early in life. I have previously referred to those pseudoparalyses which follow injuries to the bones or joints in instrumental deliveries and which have no direct association with a damage to the nerve. I must also refer to the epiphyseal loosening which is observed in children with congenital syphilis and which may resemble paralysis (pseudo-paralysis, Parrot).

Further de Péters has called attention to a form of paralysis which he regards as a spinal cord affection; this occurs in children with congenital syphilis and, particularly if the radial region is involved, gives a peculiar appearance to the hand and fingers. The wrist-joint and the pronated forearm are so flexed and abducted that a singular position results which resembles the paddles of a seal, and was called by de Péters the "*paddle position*."

Moreover, we observe in children affections of the spinal cord which are more or less familiar to us as spinal infantile paralysis.

This acute poliomyelitis of children may closely resemble the Erb-Duchenne paralysis, the same muscles are paralyzed, in the same combination, and with the same electric reactions. In comparing these with paralyses occurring *intra partum* we have as differential factors the history, the more advanced age of the child, preceding infectious diseases or a febrile condition, and the absence of marked disturbance of sensation.

I must admit that Remak has recently described an *amyotrophic plexus neuritis* in children which may very readily be confounded with poliomyelitis; its differential diagnosis from circumscribed poliomyelitis may under some circumstances be exceedingly difficult, and perhaps only by its more favorable outcome can amyotrophic plexus neuritis be distinguished.

In adults we may find an acute, subacute, or chronic spinal paralysis which is described in literature as acute poliomyelitis or chronic poliomyelitis of adults; if, in the given case, we suspect lead poisoning, we must ascertain whether any of the symptoms can be referred to this etiologic factor.

Finally, we may observe paralyses resembling those described in persons predisposed to nervous derangements after trauma. These local *hystero-traumatic paralyses* may be differentiated from those due to a peripheral nerve injury by the peculiarly distributed disturbances in sensation almost invariably present, by the retention of electric contractility, and by the other symptoms of hysteria.

The *prognosis* of plexus paralysis depends upon the underlying affection.

Tumors of the vertebræ or carious processes in the cervical vertebræ which produce plexus paralysis are more serious than slight injuries of the plexus itself. In electric contractility we have a test which rarely fails to reveal the severity of the attack and its probable duration. Cases of obstetrical paralysis usually improve with time, nevertheless recovery is incomplete; in any case it is wise, in a prophylactic sense, to avoid during labor all unnecessary pulling or tension upon the limbs and the vertebral column of the child. During prolonged anesthesia, too, we should not hold the arms of the patient too firmly nor abduct them too strongly. It need hardly be emphasized that in gymnastic exercises and in certain occupations there should be intervals of rest to prevent abnormal tension, torsion and strain. If operation is indicated this should first be performed before we proceed to treat the paralysis by means of baths, massage, exercises and electrotherapy.

Parrot's pseudo-paralyses, as well as de Péters paralyses due to syphilis, should be treated with antisyphilitic remedies which are usually very beneficial.

18. PARALYSIS OF THE MUSCLES OF THE NECK, BACK AND ABDOMEN

Before describing paralysis of the lower extremity, we must briefly consider those pathological conditions due to *paralysis of the muscles of the neck, back and abdomen,* which rarely occur singly but are partial phenomena of other pathologic processes.

Paralysis of the muscles of the neck is noted in progressive muscular atrophy, in infantile spinal paralysis, in asthenic bulbar paralysis, in syringomyelia, in polyneuritic processes, and in caries of the vertebræ of the neck. The head droops upon the chest, and can be raised only by the aid of the hand. In the muscles of the neck, which are usually atrophic, electric contractility is either markedly decreased or is lacking.

Isolated paralysis limited to the *deep anterior muscles of the neck* has been observed only once, in a case of hysteria, the patient's head being bent backward, and he was unable to bring it forward without the aid of his fingers introduced within the mouth.

Paralysis of the auxiliary muscles of respiration may follow lesions in the upper part of the spinal cord; this has been seen also in hemiplegic conditions.

If the *muscles of the back,* especially the extensors of the vertebral column, are paralyzed, it is impossible to raise the trunk, that is, to extend it. To maintain the equilibrium of the body the upper part must be bent far back, the lumbar vertebral region showing lordosis. The center of gravity for the body is behind the promontory of the sacrum. If the patient bends too far forward he falls. The actions of these patients on rising from a recumbent posture are especially characteristic: They place both hands upon the knee and slowly draw them up the thighs, and, as it were, *climb up upon themselves;* then with the help of their arms and shoulders they raise the trunk. In sitting, the weak extensors of the back no longer hold upright the lumbar vertebral column which is bent backward, and shows kyphosis. In walking the trunk waddles, for the weak extensors of the back no longer hold it firm. When the patient is in the recumbent posture the kyphotic curvature of

the vertebral column no longer appears. It is easy, therefore, to see that unilateral paralysis of the muscles of the back causes lateral displacement of the vertebral column (scoliosis).

This description is especially applicable to a disease described by Duchenne, pseudohypertrophy of the muscles; of course, the symptom-complex will be identical even if other pathologic processes have caused the same weakness of the muscles of the back. The condition is found in infantile spinal paralysis, after enteric fever or diphtheria, in severe lesions of the spinal cord, also when the back has been injured or is diseased by trauma or rheumatic processes. It is well known that paresis of the muscles of the back occurs in weak anemic children, even without a conspicuous nervous affection, and gives rise to curvatures of the vertebral column (scoliosis, kyphosis).

Paralysis of the muscles of the abdomen may generally be referred to the same etiologic factors as the forms of paralysis just described. In the course of enteric fever the abdominal muscles are often severely damaged. As the vertebral column and the trunk are bent forward by the muscles of the abdomen (also the ilio-psoas muscle), when there is loss of function in these muscles the patients readily fall backward on slight extension of the trunk.

According to Duchenne, such patients markedly flex the pelvis anteriorly so as to support the trunk by the extensors of the vertebral column which have remained normal. With the consequent curvature of the vertebral column, the point of equilibrium falls in front of the promontory of the sacrum.

If the abdominal muscles are paretic or paralyzed, expiration and all the functions dependent upon it (coughing, sneezing, expectoration, etc.) suffer, above all intra-abdominal pressure (difficulty in emptying the bladder and the rectum).

The abdomen is distended; the patients cannot rise from a recumbent posture without aid.

To complete the symptomatology I must state that upon tapping the surface of the abdomen various reflexes (Rosenbach's, also Geigel-Dinkler-Bechterew's reflex) are absent; in the extremely uncommon *unilateral* paralyses with forced expiration the paralyzed half of the abdomen is drawn toward the sound side.

A few researches into the possible change in the electric contractility of the paralyzed muscles have revealed the same changes which occur in diseases of other muscle regions.

The points upon which to base the *diagnosis* of these conditions were enumerated in the description of the symptoms.

When we remember that these paralyses rarely occur singly, a most careful examination of the patient's entire condition is necessary in order to determine the relation of the symptoms which I have described to an affection of the spinal cord, of the muscles, etc.

That diseases of the spinal cord, progressive muscular atrophy or pseudohypertrophy of the muscles, may be followed by paralysis of the muscles of the neck, the back and the abdomen, and that these do not permit a favorable prognosis is obvious; it is also apparent that paralysis of the muscles which facilitate expectoration may seriously threaten life.

In the treatment of these forms the underlying condition must receive

myelitic paralysis, the knee-jerk may be absent; this has been observed after diphtheria, and repeatedly in the case of diabetes.

While, however, in the majority of cases of tabes (it is well known that in this disease the absence of the knee-jerk, the so-called Westphal's sign, is one of the first and most constant symptoms) the knee-jerk which has disappeared may exceptionally return; and in anterior poliomyelitis it usually remains absent, it may happen that after diphtheria or diabetes the knee-jerk which has been lost may in the course of weeks or months be restored with an improvement in the general condition.

Furthermore, on firm pressure above the internal femoral condyle between the sartorius and the vastus internus, the testicle on this side rises (cremaster reflex, Jastrowitz). The large saphenous nerve and the external spermatic nerve which innervate the cremaster muscle are branches of the crural nerve; in hemiplegia this reflex is absent on the paralyzed side; whether it is also absent in peripheral paralysis of this nerve will be disclosed by investigation of such cases.

As an extreme abnormality I must mention *congenital absence* of the extensor of the leg; whether this is due to persistent or beginning muscular dystrophy is as yet unknown.

If paralysis of the crural nerve is produced by trauma of the vertebral column, or by tumors in this region or in the pelvis, the prognosis is usually unfavorable. In cerebral or spinal pathological processes its curability is more unlikely than when due to a peripheral lesion. When electrical examination reveals conditions deviating but little from the normal, the more favorable is the prognosis. Neuritic paralysis after diphtheria, in diabetes, or from chronic alcoholism affords a more hopeful prognosis because good results may be attained by means of tonics and general strengthening measures, and sometimes by prohibiting injurious elements (such as alcohol).

In many cases, at least in the first stages, the **treatment** should be purely surgical. This is especially true when the paralysis is due to injuries of the thigh, the vertebræ, the bones of the pelvis, or to abscesses, tumors, or other affections of the hip- and knee-joint. Atrophy of the thigh which is often so extreme in affections of the knee-joint, is favorably influenced by orthopedic treatment, by massage, and particularly by electro-therapy which cannot be too strenuously advocated.

Atrophic paralysis which frequently persists after severe poliomyelitis and resists all attempts at cure, and which as yet has been treated with very little success, has lately in a few cases yielded to careful surgical treatment.

Without entering into details, I may state that in a case of severe infantile paralysis in which the quadriceps femoris was almost wholly atrophic, F. Krause by a successful operation transplanted the flexors of the leg into the anterior side of the thigh, and thus almost wholly compensated for the loss of muscle by atrophy.

20. PARALYSIS OF THE OBTURATOR NERVE

As a supplement I mention *paralysis* in the *course of the obturator nerve.* Of course this nerve may occasionally be injured by trauma; more frequently, although rarely, it is damaged by a difficult labor or by a hernia (cases of

this have been mentioned in literature) or by a lesion of the lower thoracic or upper lumbar vertebræ.

The **symptomatology** is soon apparent if the divisions of the nerve and its branches in the muscles of the thigh are considered.

This nerve gives off branches to the adductors of the thigh as well as to the rotators; it also supplies the pectineus and gracilis. The adductor magnus receives a branch from the sciatic nerve, the pectineus one from the crural nerve, and the obturator nerve not only supplies the external obturator muscle but, at least in part, the internal obturator also.

In paralysis of the adductors, it becomes impossible to draw the legs together or to throw one leg over the other; walking is fatiguing as well as horseback riding, because firm pressure of the thighs can no longer be exerted. Some *sensory disturbances* appear upon the inner surface of the thigh; if a hernia becomes strangulated there is sometimes burning pain in this area.

It is impossible to satisfactorily explain vasomotor and trophic disturbances, either from my own experience or from the reports in literature, nor can I throw much light upon the electric reactions; nevertheless we may assume that electric anomalies occur here as in other peripheral paralyses.

The **prognosis** and **treatment** of paralysis of the obturator nerve is the same as that explicitly described for paralysis of the crural nerve.

21. PARALYSIS OF THE EXTERNAL CUTANEOUS FEMORAL NERVE

Symptoms.—Before concluding the discussion of paralyses in the region of the crural nerve we must briefly consider a peculiar symptom-complex due to an *affection of the external cutaneous femoral nerve,* which has only lately attracted the attention of physicians.

This nerve, which carries sensory fibers to supply the skin of the anterior and external surfaces of the thigh, is subject to disturbances which cause those who are affected to complain of abnormal sensations in the region it supplies; for instance, of numbness which, upon objective testing of sensation, frequently shows a diminution in the various qualities of sensation.

Actual pain, particularly when the patients sit or lie down, there is none; but on standing or walking about the feeling of numbness is often increased to seemingly actual pain. This affection which was first described by me and immediately after by Roth, occurs in both sexes but in women much less frequently than in men. Roth gave to this affection the name *meralgia parœsthetica*; it is usually unilateral but may be bilateral, and among its causes we may enumerate the effect of cold in this region of the body, infectious diseases, lead poisoning, trauma, syphilis, chronic alcoholism, gout, pregnancy, too firm pressure around the brim of the pelvis (from tight-lacing or pressure of the belt which supports the trousers). The anatomical course of the nerve in the fibrous canal of the fascia lata perhaps explains the abnormal tension and high degree of pressure upon the nerve, which, in the few cases in which autopsy has been performed, has been found thickened and spindle-shaped with the characteristic signs of neuritis and perineuritis.

It is remarkable that this paresthesia, limited to the external cutaneous femoral nerve, should occur so frequently in physicians; a number of descrip-

tions of this peculiar affection have been written by colleagues who have suffered from it. Pal recently called attention to the fact that a certain proportion of those who suffer from this affection also suffer from obesity and flat-foot, due to a displacement of the static relations of the leg, and probably to an over-distention of the hip-joint.

The **prognosis** is generally favorable. In the majority of cases we are dealing with an affection of only *one* nerve, and the symptoms of which the patients complain and which they regard as indicating a severe or progressive nervous disease, may be relieved.

It is always wise to examine the patient carefully since there are cases reported which show that the disease may occur in tabes, in hematomyelia, in malignant affections in the cavity of the pelvis, and in paralysis.

In **treatment**, *prophylaxis* is of the greatest importance. Tight lacing must be prohibited, and also the use of cold douches; persons whose occupations force them to stand for a long time (I have noted the affection particularly in waiters and the police) should be advised to take periodic rest; if there is flat-foot, measures should be taken to relieve this affection, apparatus being manufactured from firmly resistant material, and this must extend from the heel to the tip of the great toe.

Operation (resection of the nerve) is advisable only when very severe pain makes walking impossible. This will stop the pain; naturally, it is succeeded by complete anesthesia in the region of the resected nerve. But in the reports of such operations in literature we note that amelioration is not always immediate, and that relapses occur.

Of course, if there is a severe affection of the spinal cord, no operation can be undertaken. Besides treating the probable underlying affection (gout, rheumatism, alcoholism, etc.), inunctions, lukewarm salt baths, massage and treatment with a faradic brush are to be recommended.

Often the disturbances are so slight that the affection is only accidentally discovered by the physician.

22. PARALYSIS OF THE SUPERIOR AND INFERIOR GLUTEAL NERVES AND OF THE POSTERIOR CUTANEOUS FEMORAL NERVES

Before describing *paralyses of the sciatic plexus* and of the most important nerve of this plexus, the sciatic and its branches, I must briefly refer to those *paralyses* in the course of the *three upper nerves* belonging to this plexus; namely, the superior gluteus, the inferior gluteus, and the posterior cutaneous femoral nerves. The inferior gluteal nerve supplies the gluteus maximus; the superior innervates the gluteus medius and minimus, the tensor fascia lata, and the pyriformis; while the posterior cutaneous femoral is chiefly a cutaneous nerve which is distributed to the skin over the buttock, the internal and upper portions of the thigh, and over its posterior surface down to the popliteal space.

An isolated paralysis of this nerve can scarcely occur; but it is implicated in lesions of the conus of the spinal cord or of the cauda equina if these are due to injuries of the lowest portions of the back or the upper lumbar vertebræ, or to a fall upon the buttocks. These lesions, as well as direct injury

to the sacrum or tumors of this region, also lead to paralysis of the muscles supplied by the gluteal nerves. While, however, isolated paralysis in these nerve and muscular regions is very rare, paresis, particularly of the muscles of the buttock, occurs in progressive muscular atrophy, especially in the affection known as pseudo-hypertrophy. If the gluteus maximus, which draws the thigh backward and outward and when the thigh is fixed extends the trunk, is paralyzed, climbing steps and rising from a sitting or recumbent posture become exceedingly difficult. The "climbing up upon himself" which is so characteristic of pseudo-hypertrophy, is also seen when these patients rise from a recumbent posture, and is due to paralysis of the large muscles of the buttock, combined, of course, with a co-existing weakness of the muscles of the back. If the gluteus medius and minimus which abduct the thigh are paralyzed, and simultaneously the pyriformis (the gluteus medius with the posterior portion of its fibers), these movements are weak or cannot be made at all. In paralysis of the gluteus medius and gluteus minimus the abduction of the leg and its rotation inwardly is defective; and as the tensor fascia lata assists the ilio-psoas in flexing the hip and at the same time rotates the thigh a little inward'v, flexion of the thigh, if this muscle be paralyzed, will be less complete and in walking the leg will be rotated outwardly.

Among the *trophic disturbances* we recognize an apparent increase in volume, and, upon tapping, a distinct tension of the muscles of the buttock which in progressive muscular atrophy become extremely emaciated; this may also be observed in severe sciatica and in chronic affections of the hip-joint. Aside from the previously mentioned pathologic conditions in the course of the posterior cutaneous femoral nerve, little is known of sensory disturbances of other nerves; on electric examination the muscles generally show a diminished power of contractility, which varies according to the severity of the affection. With widely distributed muscular disease or a spinal lesion causing paralysis of the muscles of the buttock, the *prognosis* is less favorable than when these are due to sciatica or to a disease of the hip-joint which causes atrophy; the treatment is the same as that described in paralysis of other peripheral nerves.

23. PARALYSIS OF THE SCIATIC NERVE

In describing *paralysis of the sciatic nerve,* I must mention at the outset that paralysis of the entire nerve trunk may occur, but that much more frequently the paralyses which come under professional treatment are due to damage of one of its main branches, either the peroneal or tibial nerve.

The nerve trunk which traverses the pelvis may be injured by fractures of the lower vertebræ or of the sacrum, by injuries to the cauda equina, by tumors of the pelvis, particularly those of the uterus (in difficult labor by pressure of the child's head), which impair its integrity; or it may be damaged by injury to the femur or by tumors which have developed within it or in its vicinity. It is self-evident that it may also be injured by direct lesions (a blow, a stab, etc.). Among the chief etiologic factors of paralysis of an entire nerve or one of its branches are inflammations which may be purely rheumatic, neuritic, infectious, or septic, these affecting the nerve after various infectious diseases, in the puerperal period, in chronic suppuration, in the

course of acute or chronic intoxication from alcohol, carbon dioxid, arsenic, and lead, and also in the course of diabetes.

The paralyses of the peroneal nerve which appear early in tabes and, independent of the progress of the underlying affection, result in recovery are especially interesting. Similar conditions in progressive paralysis have been noted by different authors; lately reports have been published of paralysis which occurred (more readily than in the healthy) from the pressure of one leg being thrown over the other or from the arthritic affections which not so rarely occur in tabes (in these cases, naturally, those of the knee-joint). In the description of peroneal paralyses which we shall soon discuss other no less interesting etiologic factors will be mentioned.

In describing paralysis of the two main branches of the sciatic nerve, the peroneal and the tibial, symptoms due to paralysis of the upper branches of the trunk must be enumerated, and here the outward rotators of the thigh and the flexors of the leg are particularly involved.

The outward rotators are the quadratus femoris, the gemelli, the obturator internus. A lesion of the branches which innervate these muscles impairs the motion as previously described. If the flexors of the leg (the semimembranosus, semitendinosus, and the biceps femoris) are paralyzed the leg cannot be flexed. By hyperextension in the knee-joint, the leg becomes stiff.

Even if the peroneal nerve remains exempt in such a paralysis, the short head of the biceps femoris innervated by it may cause a weakened power of movement.

(a) PARALYSIS OF THE PERONEAL NERVE

Prior to a description of the symptomatology of *peroneal paralysis,* I must mention the etiologic factors which may induce such paralysis. It is prone to occur in women with a narrow pelvis after the expulsion of a very large child (*maternal* obstetric paralysis) and resembles that occurring in new-born infants who have been delivered by the feet. In rare cases subcutaneous injections of ether in the posterior side of the thigh may cause paralysis of the peroneal nerve (analogous to radial paralysis). Those cases are particularly interesting which occur in persons who are much upon their knees, for instance, farm laborers, asphalt workers, plumbers, etc. The tibial nerve is, of course, sometimes implicated, but never to the same extent as the peroneal nerve. Among these occupation pareses, so-called, we may include the paralyses occurring in workmen who hold the article upon which they work between the knees, which sustain a heavy pressure.

Strong pressure upon a nerve during anesthesia is also a cause of paralysis; isolated cases have been reported from sudden torsion of the leg while slipping manacles around it (analogous to paralysis of the radial or other nerves of the arm from handcuffs), in persons who walked upon stilts, and after severe injuries while playing football. I shall not describe the other rare and exceptional paralyses of this nerve; for example, paralysis after chorea; more important are those which, like radial paralysis from pressure during sleep, attack the peroneal nerve after sleep, and those which Oppenheim and Stein have recently described as senile forms of multiple neuritis. Finally, I must acknowledge that in many cases no etiologic factor can be detected, provided

that in cases of this kind we do not regard pressure effects to be increased by chronic alcoholism.

Certainly in some cases such a predisposition may be assumed; in other cases we must accept a report of exposure to cold, of rheumatism, etc. But in such cases we should never omit the examination of the urine for albumin and sugar; a positive finding of either of these will often clear up a case etiologically obscure.

The *symptomatology* of peroneal paralysis is readily understood if we consider which muscles are supplied by this nerve. The most conspicuous feature is the lost power of flexing the foot dorsally and of extending the basal phalanges of the toes. The foot which hangs down flaccidly glides over the floor upon its tip; to obviate this the patient strongly flexes the thigh and the leg and foot are thrown forward. This gait is very characteristic; Charcot calls it the *steppage gait* (démarche des steppeurs), equine gait. It is due to paralysis of the following muscles: the tibialis anticus, the extensor communis digitorum, extensor hallucis longus, extensor digitorum et hallucis brevis, and of the first dorsal interosseus muscle.

In complete paralysis of the nerve the peroneus longus and peroneus brevis are both inactive. The first named muscle flexes the foot toward the sole, abducts it, and at the same time lowers the anterior portion of the inner border of the foot; it raises the external border of the foot, at the same time abducting it by the action of the peroneus brevis.

Disturbances of sensation in peroneal paralysis are rarely marked; the patients complain of a feeling of cold and numbness; objective examination reveals hyperesthesia or anesthesia at the outer anterior side of the leg, upon the dorsum of the foot, and the posterior surface of the toes. Implicated in this are the cutaneous branches of the peroneus (the posterior, middle and external cutaneous crural, the internal and middle cutaneous dorsi pedis, and a few branches of the peroneus profundus). In profound paralysis the muscles chiefly implicated are more or less conspicuously atrophied, and electric contractility reveals distinct reactions of degeneration; of course, many cases are reported in literature of less severe paralysis with less marked electric alterations.

Among *vasomotor disturbances* we may mention the bluish, livid discoloration of the member which has been described in some cases, as well as the abnormal growth of the nails and hair, and the formation of bedsores in the areas subjected to great pressure (the heel, trochanter, etc.). But these symptoms are rare, and are observed only in consequence of severe traumatic lesions of the trunk of the sciatic nerve.

An especially interesting and important fact is that traumatic paralysis of the peroneal nerve, and also that which is regarded as the sequel of neuritis, *does not always implicate all of the fibers.* The branches intended for the tibialis anticus are sometimes exempt, while in other cases these, also those supplying the other extensors, are likewise affected, the peroneal muscles remaining intact. I shall revert to these conditions in considering the diagnosis.

(b) PARALYSIS OF THE TIBIAL NERVE

Isolated *paralyses of the tibial nerve* are much more rare than those of the peroneal. In paralysis of the tibial nerve the function of the muscles

innervated by this nerve naturally suffers, particularly the triceps suræ, that is, the gastrocnemius and the solius muscles.

These extend the foot downward and abduct it. In paralysis of this nerve it is impossible to flex the toes or to stamp vigorously upon the floor. In paralysis affecting the long and short flexors of the toes these movements are likewise impossible, as well as individual movements of the large and small toes which depend upon the abductor hallucis, the adductor hallucis, the flexor brevis, the flexor digiti minimi, and the abductor digiti minimi. If the tibialis posticus is paralyzed the power to turn the internal border of the foot upward and to abduct the foot is lost. By paralysis of the interossei pedis muscles which (together with the lumbricales pedis) extend the middle and terminal phalanges and flex the basal phalanges, a deformity is produced, the "pied en griffe," which resembles "main en griffe," the basal phalanges being dorsally flexed, the other two phalanges showing marked plantar flexion.

Disturbances of sensation caused by paralysis of the tibial, like those in paralysis of the peroneal nerve, affect the skin of the calves, the region of the ankle, the external border of the foot, and, finally, the under part of the toes. The nerves affected are the suralis, the cutaneous plantar branches, and the external and internal plantars.

Before discussing the diagnosis, prognosis and treatment of these paralyses I must state that, particularly in partial peripheral or spinal paralysis of the lower extremities, *abnormal positions of the foot* are assumed in consequence of loss of function of individual muscles; these we cannot here comprehensively describe, but I must mention a most significant fact. In partial paralysis (which is chiefly found in children as the consequence of infantile spinal paralysis) pes varo-equinus results from contracture of the plantar flexor. If the flexors of the foot are paralyzed, from contracture of the dorsal flexor, pes calcaneus follows; modifications are noted if the peroneus longus is implicated in the paralysis. These conditions, in themselves highly interesting, are especially so to the surgeon and the orthopedist, for reasons which cannot here be minutely given. This is particularly true in children in whom the conditions are chiefly observed as the result of spinal cord disease, for they not only suffer from muscular paralysis but the growth of their bones, etc., is impeded.

The *diagnosis* of paralysis in the course of the sciatic nerve must be based upon the function of individual muscles, and this can hardly be difficult if there is a history of trauma. If external injury can be excluded, the etiologic factors before enumerated must be sought for, and we must consider in the given case whether we are dealing with paralysis following a difficult labor, or one that has occurred during the puerperal period, whether febrile infectious diseases have preceded, or intoxications from organic or inorganic poisons (alcohol, lead, arsenic, sugar, etc.).

Especial significance must be attached to paralysis of the lower extremities which is so common in children in consequence of acute poliomyelitis. It is true that peripheral paralysis of the lower extremities also occurs in children, but here an accurate history will be a valuable aid. If no trauma can be detected, if other members are implicated, if individual muscles are exempt, if there is extreme atrophy of the affected limbs and decided diminution of electric contractility, the diagnosis of infantile spinal paralysis can cause no

perplexity. But I must mention a peculiarity often observed of late, that lead paralysis in the lower extremities is also observed in children, and that in children these members appear to be more frequently implicated than the upper extremities, while, as is well known, quite the reverse is true in adults.

We must also bear in mind that in paralysis of the lower extremities from poliomyelitis individual muscles are frequently exempt (for example, the sartorius in the region of the crural nerve, the tibialis anticus or the peroneus in the field of the sciatic); this peculiarity, however, is noted not only in spinal but also in peripheral paralysis. But this fact alone does not justify us in forming conclusions as to the seat of the lesion.

Pathologic conditions are occasionally observed in the form of juvenile dystrophy or progressive neurotic (spinal neuritic) muscular atrophy. Here an accurate history, the occurrence of the disease in several members of the family, or heredity, the symmetry of the paralysis in the two sides of the body, and the greater advance of the pathologic process in the upper extremities will prevent us from confounding conditions of this kind with peripheral paralysis.

If the vertebral column has been injured (shock, fractures, dislocations of the lower dorsal and upper lumbar vertebræ, etc.) we may regard paralysis of the muscles of the lower extremities as due to a lesion of the spinal cord or the cauda equina. At this point I cannot fully discuss the special diagnosis; here our knowledge of other diseases of the spinal cord must be our guide or the reader is referred to other articles of this book.

Before considering the prognosis and treatment of paralysis in the course of the sciatic I must mention a rare condition occasionally reported in literature, appearing particularly in paralysis of the peroneal nerve, which is due neither to a lesion of the peripheral nerve nor to disease of the spinal cord, but is an affection of the upper portions of the central convolutions in the brain. *Cerebral paralyses in the region of the peroneal nerve* are characterized by the fact that they are usually of traumatic origin, that the paralysis although prominent is not the only symptom (weakness in the areas of the facial nerve and in the upper extremities will always appear), and that the electric reaction of the paralyzed muscles is absolutely retained.

In regard to *prognosis* it may be stated that traumatic paralyses or those due to neuritic processes are less intractable than those attributable to disease of the brain or spinal cord, or which develop in the course of progressive muscular atrophy.

In peripheral, traumatic and neuritic paralyses of the peroneal nerve the old law so frequently emphasized is still applicable, that when the electric contractility is retained or but slightly altered there is a better prospect of restoration to health than when it shows marked change.

In the *treatment*, traumatic lesions of the nerve necessitate the same surgical procedures as are indicated in fractures, dislocations, impacted foreign bodies, etc. If it is possible to reunite by suture a nerve which has been severed, this must, of course, be done, and, in fact, has been done several times. Surgical treatment is also necessary when the vertebral column is injured or when, as in rare cases, intravertebral tumors have formed. If we remember that many paralyses in the course of this nerve are due to neuritis from toxic causes, it becomes our duty to discover this cause, and to preserve

the patient from its injurious effects (lead, arsenic, alcoholic intoxication, etc.); if there is diabetes appropriate treatment must be instituted.

When all of these indications have been fulfilled, the electric current in its various forms offers a mode of treatment not only in peripheral but in spinal paralysis which if persevered in is sometimes very satisfactory. In spinal paralysis it is most necessary to combine peripheral with so-called central treatment, i. e., to subject the diseased spinal cord itself to the influence of the electric current. It is not my purpose to indicate here how this may be accomplished; text-books upon electro-therapy should be consulted.

If, as unfortunately often happens in paralysis of the lower extremities, all our efforts are unavailing, the aid of the orthopedist must be invoked, and apparatus must substitute for the loss of function in the muscles. This idea of substitution has recently led surgeons to unite the muscles capable of function with the diseased ones by a tendon transplantation, with remarkably good results.

24. PARALYSIS IN THE COURSE OF THE PUDENDIC AND COCCYGEAL PLEXUSES.

Paralysis in the course of the *pudendic* and *coccygeal plexuses* and their nerves is of very rare occurrence, and in the majority of cases is very difficult to differentiate from that of the cord in which they originate.

In the **etiology** we usually find the history of an injury to the lowest portion of the vertebral column (the lower thoracic, lumbar and sacral vertebræ) sustained by a fall from a height which has produced either a fracture, a dislocation, or a severe contusion. These parts may also be directly injured by the falling upon them of heavy weights. Besides wounds, tuberculous and syphilitic diseases of the vertebræ and congenital anomalies (spina bifida) may be a cause. In the act casuistically described as "falling on the feet" there may be no demonstrable lesion of the vertebræ yet compression or hemorrhage into the lumbar cord or its lowest portion, the conus medullaris, may have taken place. A purely inflammatory affection limited to the lower thoracic, lumbar or sacral portions of the spinal cord is very rare.

Syphilitic affections of the lowest portions of the cord and the membranes which envelop and compress the cauda equina are more common, above all, neoplasms in the sacral canal by which the nerves are compressed and rendered incapable of function.

From a purely scientific standpoint as well as with a view to treatment, it is most necessary for the physician or surgeon to differentiate a lesion in the lowest part of the lumbar cord or the conus terminalis from one in the cauda equina. In the last twenty years authors have directed special attention to this differentiation.

The following points must be borne in mind: First, affections of the cauda are produced much less rapidly than those of the conus, as may be inferred from the preceding statements as to the cause of these diseases. In the overwhelming majority of cases a lesion of the conus produces no pain, while in an affection of the cauda, particularly at the onset, there is acute pain, localized in the sacrum, the bladder, the rectum and the buttocks, and this often radiates. Furthermore, in caudal affections motor irritative

phenomena are absent (fibrillary contractions) while they are common in diseases of the conus.

Symptoms.—The most constant disturbances in lesions of the lower portions of the spinal cord, as well as of nerves originating in this region, are those which affect the innervation of the bladder and the rectum, these organs

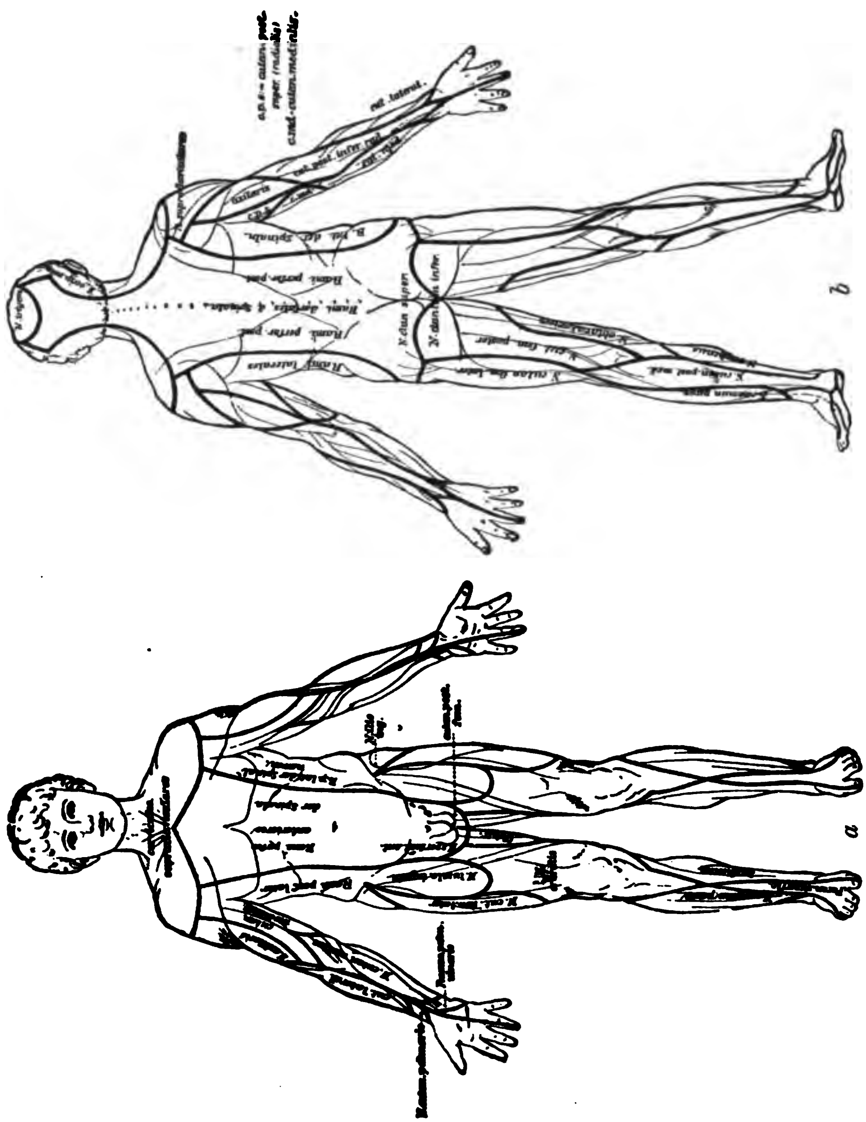

FIG. 191.—DISTRIBUTION OF THE SENSORY (PERIPHERAL) NERVES IN THE HEAD, THE TRUNK, AND THE EXTREMITIES. (After Freund.)

being paralyzed and their normal functions suspended. This paralysis of the bladder and rectum is rarely found without a simultaneous disturbance of sensation along the course of the nerves originating from the pudendic and coccygeal plexuses.

This disturbance in sensation affects the perineum, the posterior surface of

the scrotum, the penis, or the labia majora in the female. There is also anesthesia in the region of the anus, the anal groove, in the buttocks, and upon the posterior surface of the thigh to the upper border of the middle third. The mucous membrane of the genital organs and the rectum is also implicated in the anesthesia.

Before describing the motor symptoms in these cases, I must call attention to the fact, most important in the differential diagnosis, that these disturbances of sensation may be distinguished by the time of their appearance and the degree of completeness (an important guide). If the conus is injured, anomalies of sensation soon develop without preceding pain. Furthermore, the disturbances of sensation in the majority of the pure cases present the characteristics of so-called dissociated sensory paralysis, i. e., tactile sensation is retained in contrast with the almost utter absence of pain and temperature sensibility. In opposition to this the sensory disturbances which appear gradually, and if there be disease of the cauda equina usually accompanied by very severe pain, are marked by complete anesthesia—facts which may occasionally be of the utmost significance in the differential diagnosis and in treatment.

The sexual function is usually impaired; there may be complete impotence; but with retained libido cœundi only the property of ejaculation of the semen may be implicated, yet, in spite of existing libido and the power of erection of the member, impotentia generandi may be produced.

If the lesion is above the point where the last three sacral roots branch off, therefore in the upper portion of the conus, in addition to the symptoms enumerated there is paralysis of the muscles of the buttock, the flexors of the leg (the muscles upon the posterior side of the thigh) and the entire musculature of the leg and foot. Then the anesthesia is more pronounced; the entire posterior side of the thigh is involved as well as the posterior and lateral portions of the leg, and the foot with the exception of a small portion on the internal border.

The paralyzed muscles are atrophic, their electric contractility severely damaged.

When the focus of disease is still higher (implication of the vertebral cord at the height of the twelfth thoracic or first lumbar vertebra) all the muscles of the lower extremity are paralyzed, are atrophic, and their electric contractility seriously impaired; the reflexes disappear, anesthesia extends to the mons Veneris. Marked trophic disturbances in the form of bed-sores upon the sacrum, the trochanters and the heels, are not uncommon. I have now described the symptomatology of these interesting paralyses as well as called attention to a few guiding points in the **differential diagnosis** between lesions of the conus and the cauda. I must also state that in the majority of cases this fine discrimination is impossible because the cord as well as the nerves of the cauda are often simultaneously affected by the injury or the disease. Fr. Schultze describes another important point: Whether we are dealing with disease of the conus or cauda the diagnosis in the given case depends not only upon the height of the vertebra injured, but also upon the transverse distribution of the original disturbance. In a lesion of the lower portion of the lumbar enlargement without implication of the caudal roots, a paralysis may appear which implicates only the sciatic distribution (seat of the lesion at the

height of the twelfth thoracic and first lumbar vertebra); on the other hand, with a transverse lesion at the height of the second lumbar vertebra (for example, from tumors which fill the vertebral canal), there may be complete motor and sensory paralysis of all the nerve tracts of the lower extremities, so far as the roots of the crural and obturator nerves are here concerned.

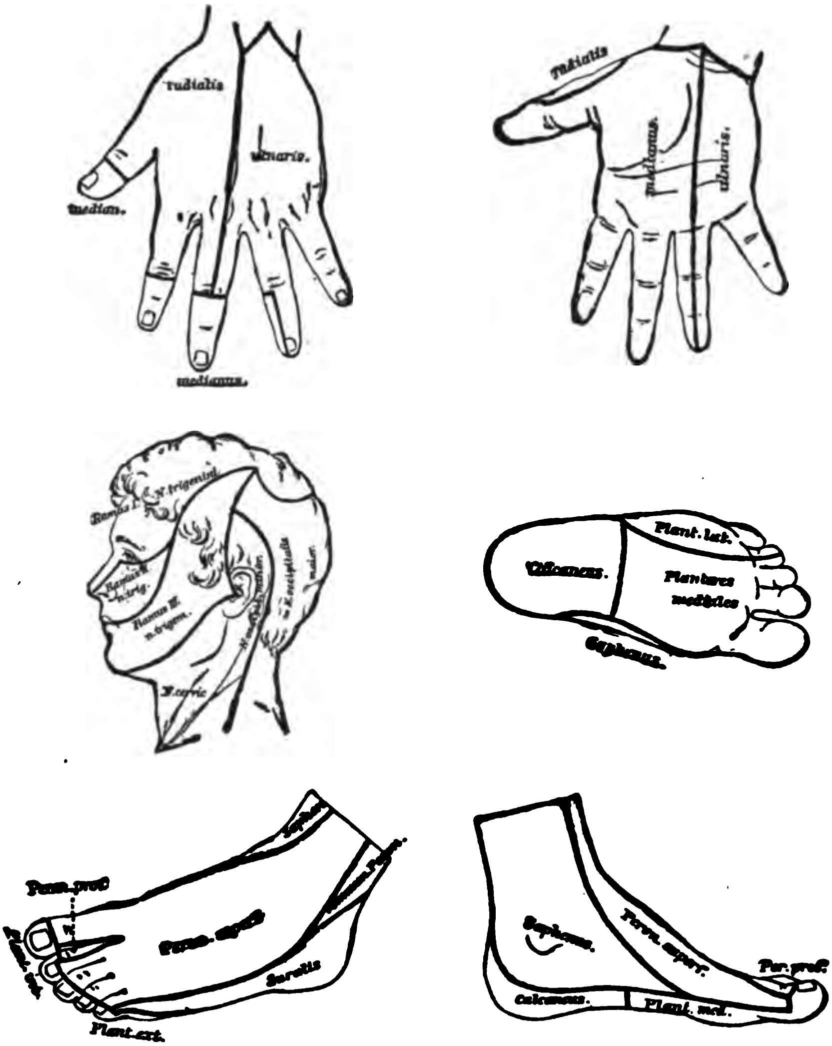

Fig. 192.

These nerve tracts may remain unaffected after a complete transverse lesion extending the entire length of the vertebral canal if the affected roots have already emerged from the vertebral canal (when the lesion is below the third lumbar vertebra).

In the **prognosis** of disease of the lower portions of the spinal cord and of the cauda equina we must always consider the severity of the damage. An

injury or disease of the cord is usually serious, although this is not invariably the case, for there are instances reported in literature which show that lesions of the cauda more often terminate seriously than those of the conus, although the latter are generally regarded as more dangerous. In not a few cases the local paralysis in certain areas of the lower extremities may in the course of weeks gradually disappear so that merely paralysis of the bladder and rectum and a disturbance of the sexual function remain. If the disease is due to

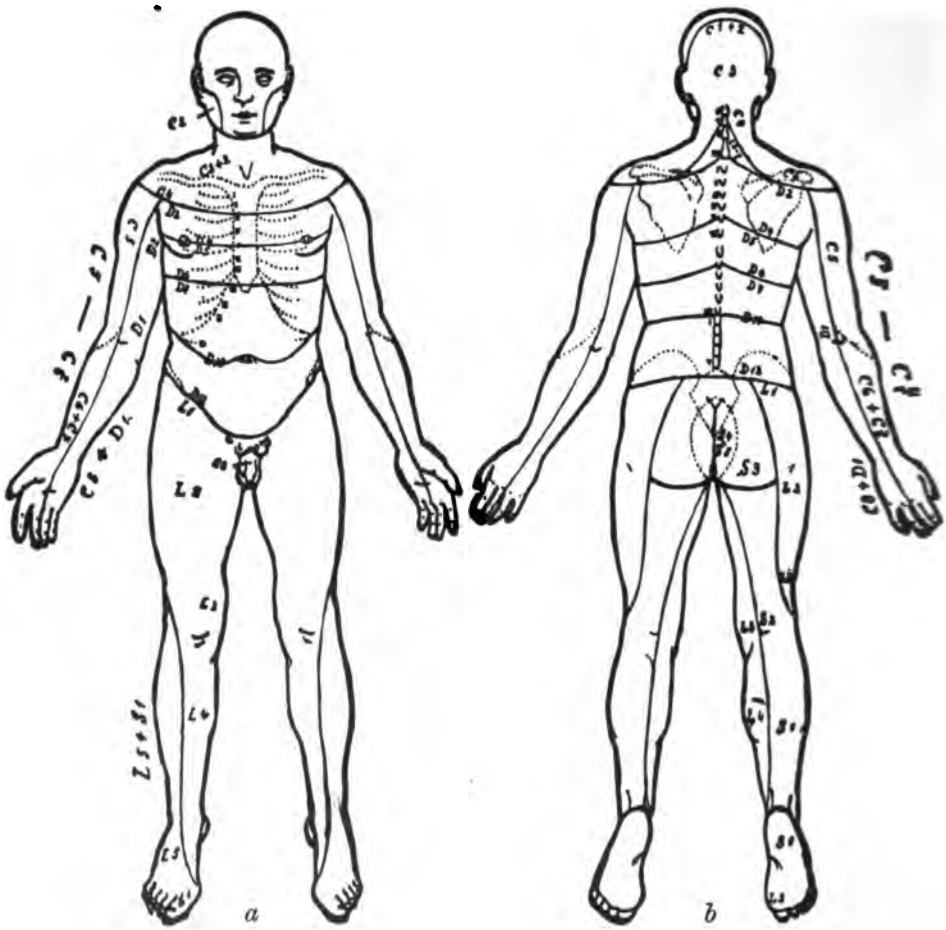

Fig. 193.—Diagram of Spinal Sensation. (After Seiffer.)

syphilis the prognosis is somewhat more favorable; when there is certainly a tumor of the cauda equina, there is some prospect of recovery if proper surgical aid is at hand.

Treatment.—In any case of injury to the vertebræ, surgical treatment is the first indication. In fracture or dislocation of the vertebræ the surgeon must decide whether anything can be done to relieve the compression of the spinal cord and its lower roots. It is the duty of the physician to see that such patients are kept in a proper position and, above all, he must endeavor

to prevent bed-sores. In most cases catheterization must be resorted to on account of the paralysis of the bladder usually present, and one of the physician's most important duties is to prevent infection of the mucous membrane of the bladder.

If syphilitic infection has preceded, antisyphilitic treatment is, of course, in order. Lukewarm baths, suitable electric treatment, and subcutaneous injections of strychnin are useful in many cases. If preceding syphilis is excluded, and the affection progressively develops, the severe pain and other diagnostic factors which have been described will convince us that we are dealing with a slowly growing tumor in the lowest part of the spinal cord, and the necessity and feasibility of operation must be considered.

Admirable results have several times been obtained by the combined skill of the neurologist and surgeon.

NEURITIS AND POLYNEURITIS

By R. CASSIRER, Berlin

NEURITIS

Case 1.—The patient under consideration, a laborer aged 30, states that two weeks ago he had an attack of influenza accompanied by fever, headache, lassitude, cough, and coryza; he was confined to bed for three days. When he left his bed he noticed a numbness on the side of the right little finger with stabbing pain on the ulnar side of the forearm extending from the elbow to the hand, and soon after a feeling of weakness when performing certain movements with the right hand. The numbness and tingling were especially marked when the forearm rested on a hard surface. The patient has sustained no injury in the course of his occupation, does not use alcohol, and has not had syphilis. He reports that he several times suffered from lumbago. Examination reveals the following conditions:

The right ulnar nerve is very painful when pressure is made upon the internal condyle, the pain radiating throughout the entire hand; the radial nerve also is somewhat sensitive to pressure, but not the other nerves. The right ulnar nerve on comparison with the left appears rather thicker. The right hand shows the so-called claw-hand (*main en griffe*), the basal phalanges of the fingers being hyperextended, the middle and terminal phalanges flexed. The anomaly is more conspicuous in the fingers from the index to the little finger. The interosseous spaces are greatly depressed, especially the third and fourth. The region of the adductor pollicis is also depressed. The patient is unable to spread his fingers or to adduct them; neither can the thumb or little finger be abducted to any extent. The basal phalanges cannot be energetically flexed, nor can the terminal phalanges of the fingers be forcibly extended; this is most noticeable in the last three fingers. The terminal phalanges of the third and fourth fingers cannot be vigorously flexed. On the contrary, the apposition of the thumb is good, and the remaining muscles of the right arm are normally strong. On brush contact there is hypesthesia, and needle pricks in the areas innervated by the ulnar nerve (that is, in the hollow of the hand, the little finger, especially the ball of the little finger, and the ulnar surface of the third finger) are less painful than in normal areas. Upon the dorsum of the hand these disturbances of sensation are somewhat more extensive, for they partially implicate the radial portion of the third finger and the ulnar distribution of the second. The tendon reflexes of the arm are preserved. Electrical examination reveals contractility of the right ulnar nerve to the faradic as well as to the galvanic current, while in the left it is moderately decreased. In addition, the faradic contractility of the paretic muscles is diminished; stimulation of the muscles with the galvanic current reveals quantitatively normal conditions. The anodal contraction does not exceed the cathodal closure contraction, but no remarkable sluggishness is observed. The patient is a strong healthy man.[1]

This case is quite simple. As is evident from the history, the affection is strictly limited to the course of the right ulnar nerve, and presents the typical symptoms of *neuritis occurring immediately after acute influenza.*

[1] This case, like the majority of those to be detailed, is from the Clinic of Professor Oppenheim, to whom I am greatly indebted for the privilege of using this material.

Accordingly the diagnosis is *neuritis of the right ulnar nerve following influenza.* In fact we here find all the symptoms which are requisite or important in neuritis.. The patient first observed *tingling, numbness,* and *pain.* Our patients usually describe this neuritic pain as *drawing, tearing, stabbing,* or *boring,* or, at least in local neuritis, as limited to the *definite distribution of the nerve.* The pain is most severe in this region, but it is sometimes felt in one, sometimes in another area, and is subject to periodical variations; at the onset and at the height of the disturbance it is more or less constant, and this differentiates it from the lancinating pain of tabetics, as well as from true neuralgia. It is increased by all manipulations calculated to remove the nerve from the median position which is instinctively assumed by the patient. If, for example, I passively extend the elbow of the patient and hyper-extend his wrist, thus pulling upon the ulnar nerve, the pain is increased. Pressure upon the nerve also intensifies the spontaneous pain. This tenderness of the nerve to pressure is another significant symptom, but one that must be utilized with care as we shall soon see. The pain is extreme when we press the nerve against a bony under-surface, such as the internal condyle; the sensation immediately over the wrist when we press the ulnar nerve against the styloid process is exceedingly disagreeable, and in many cases the affected nerve is sensitive to pressure throughout its entire course. The sensitiveness may be limited to the point of compression; usually pressure produces pain peripherally over the course of the nerve, but it may often radiate centrally. Valuable as is this symptom for diagnosis, one point must not be overlooked. Even pressure upon a normal nerve will produce an unpleasant sensation; for instance, as we all know by experience, when we accidentally strike the internal condyle, and thus cause shock to the ulnar nerve. This sensitiveness of the nerve is frequently extreme in persons with neurasthenia and hysteria. In the peripheral nerves of neurotic individuals there is not rarely a pathological increase of tenderness on pressure, appearing at one time in one, at another time in another nerve.[1] We cannot base our diagnosis of neuritis (as Franke[2] incorrectly assumed to be possible) on a few patients who have recovered from influenza. It has also been pointed out (especially by Remak[3]) that when secondary nerve degeneration takes place—for example, after poliomyelitis—the nerve may be sensitive to pressure. Therefore, in my opinion, when differentiating neuritis from poliomyelitis too great value should not be attached to this symptom.

It is difficult to decide whether we are justified in speaking of *swelling* of a peripheral nerve; when the affection is unilateral comparison with the normal side aids us. Little significance can be attached to slight differences, although the swelling, usually spindle-shaped, is occasionally most distinct. This is commonly observed in leprous neuritis, but Remak saw it also in syphilitic neuritis, Meyer and Henschen observed it in facial paralysis, Oppenheim found it in an alcoholic who complained of severe pain in the course of the peroneal nerve, and in whom the inner side of the biceps tendon was so

[1] Oppenheim, "Zur Differentialdiagnose der Neuritis." *Journal für Psychologie und Neurologie,* Bd. I, 1902, p. 129.

[2] Franke, *Mittheil. aus dem Grenzgebiete der Medicin und Chirurgie,* Bd. V. 1900.

[3] Remak, "Neuritis und Polyneuritis." Wien, 1900, p. 92.

swollen that its circumference was more than double that of the normal nerve of the other side. In another patient he found the median nerve almost wholly transformed into a thick hard cord.

Of subjective symptoms the only important one is paresthesia in the area innervated by the diseased nerve; this is usually described as tingling or numbness. Such paresthesias are rare under normal conditions, and patients describe them variously: for instance, they say the sensation is as if ants were crawling over the skin (formication), or they feel as if an electric battery were being applied to the skin, etc. These paresthesias are most marked in the terminal distribution of a nerve. In comparison with the sensory, the *motor* irritative phenomena are less numerous, but are most common in traumatic neuritis. They consist of slight contractions and spasmodic and fibrillary movements in the paralyzed areas. These phenomena may occur at the onset of paralysis as well as during the time of restitution. In cases of severe facial paralysis from which there has been only partial recovery, we quite often find not only contractures upon the paralyzed side but also conjoined movements and clonic contractions.[1] Our patient did not present this symptom.

Among the cardinal symptoms of neuritis is *paralysis,* which rarely fails to appear as soon as the pathologic process attacks a mixed nerve. Its onset may be sudden or gradual; even the entire region of a plexus may be attacked by an acute, almost apoplectiform, paralysis, and in localized neuritis pareses may implicate one muscle after another. In the majority of cases of localized neuritis, paralysis is almost uniform in the entire area of distribution. It is frequently noted that the muscles most distally situated are the ones most severely damaged. In ulnar neuritis the flexor carpi ulnaris and the ulnar portion of the flexor digitorum profundus are usually less markedly implicated.

For various reasons paralysis affecting the different muscles innervated by one nerve may be unequal. The exciting cause may affect a nerve in an area where it gives off fibers for certain muscles—a so-called crutch paralysis in which the pressure producing neuritis affects the axilla and implicates all the muscles supplied by the radial nerve including the triceps. When injuries affect the radial nerve in the upper arm, the triceps remains intact, and the supinator longus may be exempt even when the lesion is more distally situated. Furthermore the pathological factor may have a directly selective action upon the muscles supplied by one nerve; a typical example of the elective and, we might almost say, of the systematic influence of such pathologic causes is radial lead paralysis in which the supinator longus is always exempt, and frequently the abductor pollicis. Therefore the diagnosis of neuritis of a single nerve cannot be based upon the fact that all of the muscles which it innervates are uniformly implicated.

If the disturbance of conduction is less intense, there is weakness or paresis instead of paralysis. In our patient there were distinct atrophic and qualitative changes in the electric contractility of the paretic muscles, and this finding is typical in peripheral neuritis. *Neuritic paralysis is atrophic and degenerative.* In both the acute and subacute forms—i. e., in nearly all cases—atrophy soon follows paralysis. In the course of a few weeks it reaches its

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[1] See Bernhardt's article in this volume, p. 662.

acme, and, if marked, produces extremely characteristic *deformities* which aid in the diagnosis. For instance, our patient presented the typical *claw-hand*: hyperextension of the basal phalanges with marked flexion of the middle and terminal phalanges, as well as depression of the interosseous spaces and flattening of the ball of the little finger. This deformity originates in the weakness of the paralyzed muscles and the immoderate tugging of the non-paretic ones. We find anomalous and just as characteristic positions and deformities in diseases of other peripheral nerves; thus the *ape-hand* in median neuritis, and the *wrist-drop* of radial neuritis, etc. We cannot at this point discuss these conditions, neither can we now enumerate the special derangements of innervation which are expressed by paralysis of individual nerves, since these are fully considered in another part of this volume.[1]

The paralyzed muscles often show tenderness on pressure, although, certainly in localized neuritis, it is not usually prominent. When it appears early, even before the paralysis, and is marked, it suggests a coördinate substantive disease of the muscles (*neuromyositis*).

The *tonus* of the paretic muscles is invariably *decreased*; therefore the paralysis is *flaccid*, and, as is usually the case in other conditions, flaccidity of the muscles is combined with an arrest or decided *decrease of the tendon reflexes*. This decrease of the tendon reflexes is, as a rule, an early symptom, and is marked even when the paralysis is slight. Occasionally it is the only sign of a further extension of the neuritis, as might appear from the paralysis. In neuritis of the peroneal nerve the Achilles tendon reflexes alone may be absent, although the branches of the tibialis posticus are not wholly intact. The decrease or arrest of the knee-jerk may be the only positive sign of implication of the crural nerve—provided the power and electric contractility of the quadriceps show no conspicuous damage. Absence of the tendon reflexes may persist until convalescence and after the disappearance of all other neuritic symptoms. The reflex may be abolished for years and may never return, even though all other functions are restored to the norm.

But there are exceptional cases. The tendon reflexes may be preserved and even increased in neuritis. Moebius and Strümpell first reported this. These authors, as well as Sternberg and Déjérine, assume that the cause of the phenomenon is neuritic irritation of the sensory fibers of the mixed nerves. Such exceptional cases have recently been described also by de Buch,[2] Brissaud and Brouardel, and others. Of course an increase of the tendon reflexes may be due to paralysis of the antagonistic muscles. At all events a true increase of the tendon reflexes in neuritis and polyneuritis is a very unusual phenomenon, which is seen only in mild cases, and always calls for careful diagnostic consideration.

The investigation of the *electric contractility* is of the greatest diagnostic and prognostic importance. Concerning this reaction, I shall mention only a few general points, others of special significance being described later. For individual conditions I refer the reader to Bernhardt's article in this volume.

In neuritis we meet with endless variations in the disturbance of the elec-

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[1] See Bernhardt's article, "Paralysis of Peripheral Nerves," in this volume; also Bernhardt, "Die Erkrankung der peripherischen Nerven," 2. Aufl., Wien, 1902.

[2] "Polynévrite et réflexes." *Journ. de neurolog.*, 1901, p. 143.

tric contractility. In our case there were symptoms of *partial DeR*; i. e., a decrease of faradic and indirect galvanic excitability, and examination of the muscles with the galvanic current showed sluggish contractions; the quantitative contractility is not decidedly changed in this form of irritation, nor is it reduced in a sequence of contractions: cathodal closure contraction—CaCC —is greater than anodal closure contraction—AnCC. The earliest disturbances of electric contractility, as is well known, appear toward the end of the first week. They consist in a decrease of contractility on faradic and indirect galvanic irritation; in the course of two or three weeks more, in severe cases there is complete arrest of contractility to this form of irritation. In the meantime, upon direct galvanic irritation we note a *qualitative* change in the contraction, which becomes sluggish with a slow ascending and slow descending motion. The sluggishness of the contraction is the only evidence of the DeR inseparably associated with it. Occasionally the contractility in response to the direct galvanic current is *increased*; often the sequence of contractions is inverted, and AnCC is more readily evoked than CaCC. When this contraction is not sluggish but lightning-like, we cannot refer it to DeR. The form here described is *absolute DeR* (arrest of both faradic and indirect galvanic contractility and sluggish contraction with direct galvanic irritation). Retained power of contractility to the faradic and indirect galvanic currents, although decreased and sluggish with galvanic muscular irritation, we call *partial DeR*. This form was observed in our patient.

In a *diagnostic* respect, certainly so far as neuritis is concerned, the differentiation between these two forms (which present many variations) is not essential; in a *prognostic* respect, however, it is of paramount significance. The electrodiagnostic finding in this case enables us to assure our patient that his paralysis is curable, and that the duration of the disease will probably not be more than two or three months. We will subsequently see how the finding of complete DeR may modify the prognosis.

The majority of cases of neuritis are characterized by a *qualitative* change of electric contractility in the form of a partial or total reaction of degeneration, and, as a rule, the milder forms of paralysis show partial DeR, while the most severe forms of complete paralysis often present total DeR. But there are cases of neuritic paralysis in which no qualitative changes of electric contractility can be demonstrated; the derangement (diminution on all forms of irritation) is merely quantitative, and, although rarely, there may be no disturbance of electric contractility. We find this condition in the cases in which paralysis is incomplete, these cases having throughout a good prognosis. The following fact, the knowledge of which we owe to Erb, is of great diagnostic importance: if a point above the presumable interruption of the nerve is stimulated, no contraction follows, but this appears promptly if the electrode is placed over the diseased area. This is illustrated by the following case:

CASE 2.—A young man, after a severe wrenching backward of his right arm, noticed weakness in the right hand which in the next few days increased, and was accompanied by paresthesia in the area of the ulnar nerve. The paresis in the muscles of the hand innervated by the ulnar persisted but was only slight; there was neither objective sensory disturbance nor change in the electric contractility. Upon stimulating the ulnar behind the condyle a distinct effect was produced in the muscles supplied by this nerve, while no contractions could be evoked by irritation of the fibers of the ulnar nerve in the axillary cavity. This proved that an organic change had

inhibited conduction in the ulnar nerve, and, from its results and development, this change must have been neuritis due to trauma. In traumatic cases in which the opinion of an expert is desired the importance of such a proof of the interruption of conduction is obvious.

As in the non-neuritic peripheral paralyses, in neuritis there is frequently an incongruence between the functional and the electro-diagnostic findings in some stages of the affection. In the stage of regeneration, electric contractility may for some time be absent or may be pathologically changed even after the function has reappeared—a fact proven by experiment.

As a final objective symptom in our patient we demonstrated sensory disturbance, which, as in most cases of neuritis of non-traumatic origin, consisted in *reduction of sensation for all sensory qualities.* This hypesthesia may be more or less marked according to the severity of the affection; it ranges from complete anesthesia, which is seldom observed, to slightly decreased sensation. Usually all the sensory qualities are simultaneously affected. Partial sensory paralysis has been only a few times observed in neuritis. Lähr [1] compiled a number of such cases. The case published by J. B. Charcot proves that dissociated sensory paralysis may occasionally occur in neuritis, but it is certainly exceptional.

Notwithstanding the appearance of *severe, motor, paralytic phenomena, sensory symptoms of absence of function* are often either slight or wholly absent. If these phenomena were observed in true neuritis, we might assume that the sensory nerve fibers have a greater power of resistance against the causative agent of the disease. Explanations are not wanting, and such cases may now and then actually come up for consideration. It has been determined experimentally that under gradually increased pressure the conduction of motor fibers is interrupted sooner than that of the sensory (Lüderitz); an effort was made to account for this by the supposition that the sensory nerve fibers are situated in the interior of the nerve trunk, and are thus better protected from mechanical disturbance. That in many cases a different explanation must be given is taught by the fact that, even after the complete severing of mixed nerves, there is either no anesthesia or it is restricted to a much smaller area than might be expected from the anatomical relations. Amelioration of such a sensory disturbance is often very rapid and far exceeds the restoration of the motor function. It has been alleged that the sensory cutaneous innervation of such a region is performed through manifold anastomoses of the different nerve trunks in the periphery. By painstaking investigations Zander proved this for the dorsum of the hand, and he and Frohse working jointly, for the face.[2] For example, Frohse states that in the lateral portions of the face there are many areas in which the muscles innervated by the three branches of the trigeminal nerve overlap each other, and in other areas there are even branches of the large auricular nerve, a branch of the pneumogastric. Anastomoses have also been proven between the nerve trunks in the forearm. Other explanations contend that fibers of normal cutaneous areas extend into the anesthetic portions; some authors (Duvante, for example) have recently assumed a so-called au-

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[1] Lähr, "Ueber Störungen der Schmerz- und Temperaturempfindung infolge von Erkrankungen des Rückenmarks." *Arch. f. Psych.*, Bd. XXVIII, p. 773.

[2] See Fig. 189 (p. 678) in Bernhardt's article, "Paralysis of the Peripheral Nerves."

tochthonous regeneration—i. e., a regeneration of peripheral nerves which have no connection with the central organ—to be the cause of this phenomenon. No matter what the conditions, there is no doubt of the clinical importance of sensory phenomena in the majority of cases of neuritis.

Although the motor disturbances are as a rule more prominent than the sensory, yet there are exceptional cases in which the reverse is true; therefore in which the sensory symptoms prevail. We will later describe such a case. There is also a pure *sensory neuritis* in which only the *sensory* fibers of mixed nerves are affected. Finally we sometimes observe a pure sensory neuritis due to the fact that nerves entirely composed of sensory fibers are affected.

Thus, although rarely, the sensory branch of the trigeminal nerve may be the seat of a spontaneous neuritis, as I once had the opportunity of observing. Kuner [1] recently described such a case of neuritis affecting the sensory branch of the fifth nerve. The prototype of the second form is *meralgia paresthetica*, described by Bernhardt [2] and Roth, which is a neuritis of the external femoral cutaneous nerve. There are but scant reports in literature of other sensory forms of mononeuritis. Ballet described a case of neuritis of the greater saphenous nerve, Remak one of neuritis of the internal femoral cutaneous. Kutner reported two cases, both occurring in alcoholics, in each of whom the affection was symmetrical. In one case the superficial peroneal nerve, in the other the large saphenous, was affected. In none of these cases was the neuritis purely sensory, but it occurred in the larger branches of mixed nerves.

Compared with the sensory symptoms of absence of function, the peculiarities of which will be enumerated in other observations, the sensory, objective, and irritative phenomena are secondary. Only in traumatic neuritis do we often find *hyperalgesia* of such a nature that irritations not otherwise painful will produce pain, and the slightest prick of a pin will cause an indistinctly localized, but diffusely distributed and unbearable, burning sensation. This hyperalgesia may be located in an area in which sensation is decreased (*anesthesia dolorosa*). Weir Mitchell,[3] to whom we are indebted for much of our knowledge of these neuroses, has pointed out that there is no actual hyperesthesia in the sense of a true increase of cutaneous sensation.

Etiology.—In our patient the affection can be etiologically traced to influenza. The case therefore belongs to the great group of infectious and postinfectious forms of neuritis, and, since only one nerve was affected, may be designated a *post-infectious mononeuritis*. On attempting to review the subject of neuritis and polyneuritis, and to subdivide it, we are confronted with difficulties, and in spite of the utmost care it is impossible to make a satisfactory division which will be uniform from all points of view. We may discriminate between *local* and *general* neuritis (*mononeuritis* and *polyneuritis*).

The majority of cases of *mononeuritis* are of *traumatic* origin due to the *severing*, *torsion*, *contusion*, or *compression* of the nerve, and to this form belongs the pressure resulting from some occupation (*occupation neuritis*). Among the local forms of neuritis we must also include the local infection of

[1] Kuner, "Zur Klinik der sensiblen Mononeuritis." *Monatschr. für Psychiatrie und Neurol.*, 1905, p. 29.

[2] See this volume.

[3] Lectures on "Diseases of the Nervous System, Especially in Women." Second Edition, Philadelphia, 1885.

a nerve from trauma (wound neuritis), also that in which toxic products have effected an entrance and damaged the peripheral nerve (such as injections of *ether, antipyrin, hyperosmic acid,* or *corrosive sublimate*), also neuritis *propagated* from the immediate surroundings: for instance, a purulent process around the nerve may finally generate a local disease of the nerve although the nerve for a long time exerts a certain power of resistance. In addition, the much discussed *ascending neuritis* must be included in this group, as well as the local neuritis of which our first patient forms an example—local infectious or post-infectious neuritis; it must also embrace those cases in which, either after *refrigeration* (*rheumatic neuritis*) or without a recognizable cause, a local neuritis develops (*idiopathic mononeuritis*). The general infections and intoxications, whether from exogenous or endogenous poisons, occasionally produce a local neuritis. In the alcoholic we find an isolated peroneal neuritis, in the diabetic a crural neuritis. Infections as well as intoxications frequently produce a more widely distributed form of the disease (polyneuritis), which may also appear as an *infectious disease sui generis.*

While the innumerable varieties of neuritis at present prohibit classification, yet the etiology is by no means always *uniform.* Two or more causes may act together to produce the disease. Oppenheim long ago maintained this multiplicity of causes. The effects of alcohol may cause an otherwise insignificant pressure to interrupt the conducting power of a nerve; *tuberculosis,* chronic *lead poisoning,* or other intoxication combined with the damaging effect of an *occupation* may cause neuritis, the nature and distribution of which depend largely on the latter cause. Thus we may form a conception of a toxico-traumatic, toxico-professional, or toxico-traumatico-professional neuritis. *General disease of the central nervous system* from a local cause may predispose a person to neuritis, and this is not uncommon in tabes; here no other recognizable cause of neuritis appears.

Localized neuritis does not implicate merely a single peripheral nerve. If the local effect is sufficiently extensive it may attack several nerves in the same member, a nerve plexus may be more or less damaged, or infectious and post-infectious neuritis may implicate an entire plexus (of which I shall later give an example) and thus attack different nerves. Remak[1] called attention to multiple localized neuritis which he designated as mononeuritis multiplex or disseminated polyneuritis. It is evident, therefore, that the boundary line between local and general neuritis cannot always be sharply defined.

Localized or general neuritis may occur in some of the acute infectious diseases, in influenza, enteric fever, pneumonia, appendicitis, scarlatina, measles, whooping-cough, acute articular rheumatism, angina tonsillaris, and mumps; probably no infectious disease can be omitted from this list. In some it is common, in others rare; in the majority the nature and distribution of the neuritis shows no intimate relation to the preceding infection. An exception to this rule is diphtheria, which produces the characteristic pathologic picture of a definite form of polyneuritis. The following illustrations of localized neuritis occurring after infectious disease demonstrate the variations in the etiology, and furnish other points in the symptomatology of localized neuritis.

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[1] *L. c.*, p. 303.

CASE 3.—The case was that of a laborer, aged 40, and was especially interesting because of the rare localization of the neuritis. The patient was admitted to the hospital on the 14th of November, 1901, and remained until the 6th of January, 1902. A diagnosis of pneumonia was made. After the severe symptoms of the primary disease had abated, the patient complained of pain and weakness in the right arm when he attempted to flex it. When he was examined on January 31st, 1902, his condition was unchanged, and was as follows: Flexor muscles of the arm flaccid; on flexing the right forearm the supinator longus alone showed powerful contractions, while contraction in the biceps and brachialis anticus was very slight. Electric examination revealed a typical and conspicuous DeR, which, however, was not quite absolute in the biceps and brachialis. The brachial plexus was somewhat sensitive to pressure, and the patient complained of numbness and paresthesia on the external side of the forearm and external border of the metacarpal bone of the thumb. In this region there was also distinct tactile hypesthesia which did not affect the thumb. While he continued under observation the symptoms improved, and upon examining him a year later nothing abnormal was noted.

Here we find characteristic sensory and motor irritative symptoms and those denoting absence of function in a neuritis of the musculo-cutaneous nerve, this condition, according to Bernhardt,[1] having so far been observed in only 14 cases. The causal affection, pneumonia, has rarely been described as an etiologic factor in neuritis. This may be accidental, for I have seen several such cases. The favorable prognosis based on the electro-diagnostic findings was justified by the subsequent examination.

CASE 4.—A cobbler, aged 35, had suffered six months previously from appendicitis in which the temperature rose to 104° F. Even while in bed he had pain in the right arm, and he soon noted that he could not elevate it at the shoulder-joint. The greatest difficulty was experienced in moving the arm backward. Examination showed marked wasting of the right deltoid. The patient could really raise the arm, but the power to do so was greatly diminished, and on attempting it there was distinct tension in the pectoralis major and the infraspinatus. In the region of the axillary nerve there was complete DeR. The patient complained also of numbness in the upper arm on the external side of the upper third. Needle pricks were here only slightly painful. No objective sensory disturbances appeared.

Here also the diagnosis was clear. The case was one of neuritis of the axillary nerve following a severe febrile attack of perityphlitis. The course was unfavorable, inasmuch as the neuritis was followed by atrophy and insufficiency of the deltoid, which persisted. I examined the man a few years later, and found the same condition of almost complete atrophy and absence of function in the deltoid. Nevertheless, even at the first examination, the damage in function was not so decided as we might have expected, for the patient was able to raise his arm to a horizontal plane. Subsequently the power to do this so increased that he was perfectly able to work. In this case, as has several times been reported in paralysis of the deltoid,[2] compensatory muscles assumed the functions of the deltoid, these being the pectoralis major and the infraspinatus. The possibility of this occurrence, which is occasionally noted in other paralyses, must always be considered if we would avoid diagnostic errors.

Although this patient had suffered but little from the neuritis, yet it is

[1] See this volume, p. 707.

[2] Bernhardt, "Erkrankungen der peripheren Nerven." 2. Aufl., p. 404.

noteworthy that his condition continued unimproved. The prognosis in neuritis after acute disease is relatively favorable. In the majority of cases, although sometimes only after many months, recovery takes place or there is at least marked improvement. Upon what this more or less favorable course depends is very difficult to state in the individual case. Of course, the severity of the neuritic affection as well as the general constitution and the prevention of further damage must be considered in prognosis. The results of electric examination will chiefly decide the prospects of cure. In those cases in which DeR is only partial we may count upon a favorable course. When DeR is complete we must expect a long duration of the disease. If complete DeR persists for from nine months to a year, if more powerful currents are constantly necessary in order to produce contraction, the hope of complete recovery or even of great improvement is remote. I previously stated that, even when polyneuritis does not develop, post-infectious neuritis may occasionally implicate several nerves or an entire plexus. Remak [1] distinguished these two groups as *amyotrophic plexus neuritis* of adults and *disseminated amyotrophic polyneuritis* (mononeuritis multiplex).

Case 5.—The following case is an example of the first form; it is that of an apprentice, aged 17, who, three months previously, had an attack of acute articular rheumatism which lasted a few weeks. According to his report a serum injection was given him during this time. Three weeks after defervescence, he felt severe pain in the right arm, and a little later weakness. The pain ceased, but the weakness continued until he came under observation. The right shoulder was decidedly flattened, and the biceps and supinator longus wasted. In the right infraspinatus fossa there was a deep groove. Abduction of the arm was impossible, and neither the biceps, supinator longus, nor brachialis contracted. Outward rotation of the upper arm was impossible (weakness of the infraspinatus). The action of the other muscles was normal. Typical DeR was demonstrated in the region of Erb's muscles.

The plexus in the area of Erb's point was extremely painful. There was a hypalgesic zone in the area of distribution of the axillary nerve upon the outer side of the upper arm. The supinator phenomenon appeared on the left side, but was absent on the right. Therefore the case was one of typical Erb's or upper plexus paralysis, in which the sensory as well as motor phenomena of neuritis, such as painfulness of the nerve on pressure, spontaneous pain, and sensory disturbances, are very conspicuous. In addition, motor irritative phenomena in the form of fibrillary contractions were said to have been present at the onset. The post-infectious origin is clear. So far acute articular rheumatism has only exceptionally been mentioned as a cause of local or general neuritis.[2] Cases of pure mononeuritis have several times been attributed to a propagated neuritis. In cases like my own such origin is impossible; but, on the other hand, arthritic changes and neuritis may be produced by the same cause.

Such a case was described by Remak,[3] and I saw a similar one in which neuritic and arthritic phenomena developed *pari passu*. I shall later describe this case in detail. Whether in this patient the injection of serum had any etiologic significance is uncertain, but it is quite possible, for Warrington [4]

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[1] *L. c.*, page 290. [2] Remak, *l. c.*, p. 533. [3] Remak, *l. c.*, p. 275.
[4] Warrington, *The Lancet*, 26th September, 1903.

reported a case in which a prophylactic injection of diphtheria antitoxin was followed by urticaria and swelling of the right shoulder with paralysis of the deltoid and infraspinatus. We know little of the origin of these post-infectious paralyses.

It cannot be assumed that neuritis is caused by specific pathogenic agents. On the contrary, all experience leads us to the conclusion that the affection is due to abnormal products of metabolism generated by pathogenic agents. As is evident from the cases quoted, it is unlikely that the form of the original infection has any influence upon the localization or nature of the neuritis, although to this there are exceptions; as, for example, in diphtheritic neuritis and polyneuritis. Here the poison appears to have a decided affinity for special nerves. In mononeuritis and polyneuritis, we can by no means always name the infectious agent which produces the neuritis. Occasionally we must assume a cause which has fallen into general discredit; that is, *refrigeration*. In one form of localized neuritis—facial paralysis—some authors have recently again assumed cold to be an important factor. The possibility that other forms of neuritis may develop from this cause must always be borne in mind, and we must especially take this into account when any portion of the affected nerve is superficially situated. Often, however, there is no history of chilling although the patients are inclined to attribute the disease to cold.

Case 6.—I am now treating a woman who at Easter, 1904, was attacked by severe pain in the right lower extremity. The pain was chiefly confined to the posterior side of the leg, and was frequently combined with numbness and formication. I examined her for the first time on August 18th, 1904, and found a typical and severe sciatica, but neither sensory nor motor signs of absence of function. The right Achilles tendon reflex was normal. Under the ordinary remedies improvement gradually took place, and on the 24th of October the patient was discharged as cured. About the middle of November a weakness of the right foot appeared, combined with extremely severe pain in the sciatic nerve, and some in the crural. Examination revealed paralysis of the extensors of the foot and of the toes, the peroneal muscles being apparently normal. Tenderness on pressure in all the well-known pressure points of the sciatic nerve and its branches, a conspicuous sciatic phenomenon, and distinct hypalgesia in the areas of the peroneus profundus, especially at the adjoining borders of the first and second toes, were noted. Therefore we were dealing with a partial neuritis which affected chiefly the sensory and motor portions of the profundus peroneal. Such affections which implicate the peroneal profundus alone have been described as of varying etiology (traumatic, Bartel's maternal birth paralysis). While she continued under observation atrophy and DeR appeared in the paralyzed muscles. I shall describe the electric findings somewhat more minutely than in the previous cases, in order that we may correctly appreciate the individual points in the electrical relations.

Faradic Examination.

Right.	*Left.*
Peroneal nerve, 95 mm.D.* to 60 mm., no contraction in the extensors of the feet and toes.	Peroneal nerve, 105 mm.D.
M. tibial. antic., to 50 mm.D., no contraction.	M. tibial. antic., 95 mm.D.
M. extens. hall., no contraction.	M. extens. hall., 95 mm.D.
M. extens. digit. long., no contraction.	M. extens. digit. long., 100 mm.D.
Extens. digit. brev., no contraction.	Extens. digit. brev., 105 mm.D.
Peroneal muscle, 75 mm.D.	Peroneal muscle, 95 mm.D.

* D. is the distance in millimeters that the secondary coil has been removed from the primary. [J. L. S.]

GALVANIC EXAMINATION.

Right.	*Left.*
Peroneal nerve with 4 ma.* contracts only in the peroneal muscle; with 15 ma. no contraction in the extensors of the feet and toes.	Peroneal nerve, 2 ma.
M. tibial. antic., 6 ma., CaCC. = 4 ma. AnCC., contraction sluggish.	M. tibial. antic., 4 ma., CaCC., 8 ma., AnCC., contraction lightning-like.
Extensor hall. long., 5 ma., CaCC., AnCC., sluggish contraction.	Extensor hall. long., 4 ma., CaCC.
M. extens. digit. long., 7 ma., CaCC., 5 ma., AnCC., contraction sluggish.	M. extens. digit. long., 5 ma., CaCC.
Extens. digit. brev., 4 ma., CaCC. = AnCC., contraction sluggish.	Extens. digit. brev., 2 ma., CaCC.
Peroneal muscle, 7 CaCC., 5 AnCC., contraction somewhat sluggish.	Peroneal muscle, 3.5 ma., CaCC.

* ma. = milliampères. [J. L. S.]

Examination demonstrated DeR in the region of the peroneal nerve; this was complete in the course of the peroneal profundus but only partial in the superficial peroneal. Our experience led us to conclude that the partial paresis of the peroneal muscles would improve in a few weeks, but that a longer time must elapse before the other muscles could completely recover. This prognosis proved to be correct. At the end of April, 1905—therefore after seven months—the power and contractility of the peroneal muscles was normal, but the extensors were still weak and showed partial DeR.

In point of frequency, among all the causes of mononeuritis *trauma* occupies the front rank. If we endeavor to subdivide further the group of diseases known as traumatic neuritis, we at once differentiate between *neuritis from* and *without external wounds.* The first was formerly regarded as very common, and was attributed by Weir Mitchell to an injury of the nerve trunks. With the introduction of antisepsis and asepsis came a complete change of opinion, and we learned that not the traumatic lesion of the nerve in itself, but *the infection* caused by the wound, generated inflammatory disease of the nerve. Hence so-called acute neuritis from injury is only very rarely observed. The most complete description of the symptoms will be found in Weir Mitchell's book, which is chiefly based upon the numerous cases furnished by the American Civil War.[1] Besides the local symptoms of pain, paralysis, and anesthesia, due to the infectious foundation, fever and mental disturbances appeared. Motor irritative phenomena, secretory, vasomotor and trophic disturbances, which are otherwise rare have often been observed in this variety. We designate the neuritis as chronic and subacute if the symptoms at once become chronic or gradually assume this form after an acute stage.

The *second* variety of traumatic neuritis includes those cases without external wounds. It is extremely difficult to differentiate these from simple, traumatic, non-inflammatory injury to the nerve. It is clear that if a nerve —for instance, the radial—is simply severed by a saber cut, the knife of the surgeon, or in any other way, and if merely a paralysis of the muscles supplied by this nerve follows (perhaps hypesthesia in the area of its distribution), we may regard it as *traumatic nerve paralysis but not as neuritis.* Only

[1] Weir Mitchell, Morehouse, and Keen: "Gunshot Wounds and Other Injuries of Nerves."

when irritative phenomena are added to the inevitable consequences of nerve lesion do we speak of *traumatic neuritis* (Remak). This standpoint is in some respects of little consequence, and, as Remak states, is explained only by the historical development of the teaching of neuritis. We do not diagnosticate non-traumatic, post-infectious, or spontaneous neuritis solely from the presence of symptoms which indicate more than a mere interruption of conduction. In the diagnosis of traumatic neuritis we require more signs and symptoms than for the other forms of neuritis, and this demand is unjustifiable, since no anatomical differentiation between "parenchymatous inflammation" and "simple degeneration" can be made. In the following description, in conformity with early usage, the sequel of traumatic destruction in the course of the peripheral nerves will be almost excluded.

Without external wound nerves may be suddenly or gradually damaged by various kinds of force: a blow, a contusion, torsion, pressure from dislocated fragments of bone, from dislocated bones, and from callus. Maternal neuritis *ex partu* is that paralysis which follows trauma during birth. In traumatic neuritis the *etiology often varies,* for in addition to the *exciting trauma* there may be a predisposing one, such as chronic or acute intoxication, cachexia, etc.

Case 7.—The patient is a coachman, aged 45, whose appearance shows that he is addicted to alcohol, and this he admits. Six weeks ago he fell from a wagon upon the left side of his head. Whether or not he made any attempt to protect himself with his right arm he does not know; but there is no external wound, neither a fracture nor dislocation. Soon after the accident the patient felt pain in his right arm which became numb, and after two weeks gradually increasing weakness developed in the right hand, which extended to other parts of the right arm. Severe pain and numbness persisted, especially in the thumb, and index and middle fingers. On examination the movements of the head were free, no change was apparent in the vertebral column, not even with the X-rays; the vertebræ were insensitive to pressure. The muscles of the entire right arm were flaccid, the soft parts of the dorsum of the hand were greatly swollen, the volar manus looked as if upholstered, and the right hand felt warmer than the left. There was a distinct fibrillary contraction in the right deltoid. Only the following movements were possible: pronation and supination as well as adduction of the upper arm, and slight flexion of the fingers. There was distinct hypalgesia in the region of the right axillary, median, and ulnar nerves. The nerve trunks and plexus were sensitive to pressure, the tendon reflexes were abolished, in Erb's muscles electricity revealed partial DeR, in the other muscles it was almost complete. Four weeks later marked improvement occurred, and Erb's muscles acted as well as some of the muscles supplied by the median nerve.

In this case there can be no question concerning the diagnosis of *true neuritis*; a simple traumatic nerve lesion is excluded by the gradual development and the progress of the affection. We have little information concerning the accident. It is not likely that the nerves of the right arm were immediately exposed to injury, but there may have been shock combined with *torsion* of the nerve. *Alcoholism* must be regarded as a predisposing factor. The cases are not rare in which shock, alone or with torsion, is a traumatic factor, and in which the symptoms of neuritis gradually develop to their full extent. A similar case is described by Redlich.[1] Occasionally a neuritis of the peroneal nerve develops after the so-called "turning of the foot." In such cases

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[1] Redlich, "Zur Casuistik der traumatischen Neuritis." *Wiener klin. Rundschau,* 1902, Nr. 16.

we agree with Remak that by a lesion in the surrounding areas and by direct injury to the nerve itself the original trauma produces changes which subsequently lead to degenerative paralysis. Such paralysis may also be due to trauma which gradually develops and increases, as in so-called callus paralysis, also in that due to slowly growing tumors.

Case 8.—This patient met with an accident on the 23rd of May, 1902; in consequence of the sudden stoppage of a street car in the middle of which he stood and supported himself by a strap, he received a heavy blow upon his left hand. The patient felt sharp pain in the wrist-joint, which began to swell. It was at first impossible to determine whether or not there was a fracture; gradually besides the pain there was weakness and numbness in the left hand. I examined the patient a year and a half later. He then complained of severe pain in the ulnar side of the hand and a feeling of numbness passing upward, also of weakness in the left hand on executing certain movements. Examination revealed a typical ulnar paralysis: claw-hand, atrophy of the ball of the little finger and of the interossei, with corresponding weakness and partial DeR. In the sensory distribution of the ulnar nerves there was slight hypesthesia. Examination with the X-rays disclosed a fracture of the styloid process of the ulna which had united and produced an abnormal configuration of the distal end of the ulna. As the paralysis came on gradually it must be assumed that it was not due to direct injury of the nerve by trauma, but to a dislocation of the styloid process —that is, to the formation of callus and damage to the nerve by the healing of the fracture.

Here the etiology was purely traumatic—a simple, persistent compression. In such a condition it is very difficult to decide whether we are dealing with *neuritis* in a restricted sense or with simple *traumatic nerve paralysis*. The same doubt arises in the relatively common cases in which, after a dislocation, especially that of the shoulder-joint, more or less profound plexus paralysis at once develops. Here we must assume that the nervous structures are injured either by direct or indirect force and degeneration follows, although the mechanism and the effect of the trauma are by no means clear, even in these apparently simple cases. Sometimes the relations between the trauma and the damage to the nerve cannot be perceived. The trauma may have occurred years or decades previously, and the affection of the nerve may have appeared late, following a new trauma in itself insignificant, or in consequence of some other deleterious factor, such as an infectious disease or an operation. Cases have been described in which during youth there had been a luxation of the elbow-joint, and only after decades did ulnar paralysis develop. Weber [1] reports a case in which the dislocation dated back twenty-seven years. When the patient was compelled for a time to use the left forearm, a gradually increasing ulnar paralysis appeared. In a second case reported by the same author the patient had in early youth a purulent inflammation of both elbow-joints after variola; this healed with deformity of the joints, most marked in the right. Thirty years later, and without special cause, formication appeared in the right hand with a painful contraction of the fingers—the paw-hand; as the irritative symptoms receded ulnar paralysis gradually appeared. The patient suffered from migraine. Her occupation was that of sewing and washing. Here also other factors aided in the development of the neuritic phenomena.

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[1] Weber, "Zur Aetiologie peripherer Medianus- and Ulnarislähmungen." *Deutsche Zeitschr. f. Nervenheilk.*, Bd. XV, p. 181.

With increasing frequency we have recently noted that *cervical ribs* must be regarded as etiologic factors in neuritic affections of the upper extremities.[1]

CASE 9.—A girl, aged 20, reported that for 9 months she had observed weakness in the left hand, and had previously had pain in the ulnar side of the arm, radiating from the elbow to the hand. She said that the hand always felt cool. There was no history of over-use of the hand. The muscles of the ball of the thumb and little finger of the left hand, as well as the other small muscles, were emaciated. The function of these muscles was almost entirely arrested, even the flexors of the middle and terminal phalanges and the pronators failed to act properly; but the muscles supplied by the radial nerve were intact. There were no objective sensory disturbances, but the brachial plexus was extremely sensitive to pressure. The pain increased when the patient raised her arm and stretched it backward. The left hand was usually cooler than the right; in the atrophic muscle there was partial DeR. On palpation of the supraclavicular fossa an indistinct abnormal resistance was felt, which was not understood. Since all etiologic factors were absent, we were constrained to think of the cervical ribs which appeared distinctly in the X-ray picture.

As this condition is always *congenital*, another auxiliary factor must be held responsible for the later appearance of neuritic phenomena, which, as in the above-mentioned cases of late neuritis after luxation, etc., may be due to over-exertion, infection, or additional trauma. In this patient we were unable to discover any accidental cause; but slight periostitic processes originating from these abnormal formations were suggested. We must be cautious when we refer nervous irritative symptoms and those due to absence of function in the upper extremities to congenital cervical ribs; the latter, as Oppenheim maintains, are a sign of degeneration, and may appear in many general diseases of the nervous system. Oppenheim found them twice in gliosis, Levy[2] in multiple sclerosis. Therefore we must not regard cervical ribs as the cause of nervous phenomena without further investigation. This caution is especially important because the treatment of neuritic paralysis due to congenital cervical ribs is surgical; they have often been removed, although the operation is technically very difficult; when the indication is unmistakable, it is usually productive of good results. In this patient we advised operation for we believed the paralysis to be due to cervical ribs, and no signs of central disease could be demonstrated—here *syringomyelia*, in particular, is suggested.

We must also consider *anesthesia paralyses* under traumatic neuritis, and it must be emphasized that the trauma itself does not so much come into question as the other effects of anesthesia, especially the intoxication. Usually one of the upper extremities is paralyzed; at the operation the arm is held too tightly or too high, and this produces torsion of the plexus. The paralysis may not affect the entire plexus, and it may even be limited to isolated nerves. The damage may also vary mechanically; there may be pressure from the head of the humerus on the axillary nerves or pressure from the clavicle. Occasionally other factors besides traumatism and intoxication must be considered (Oppenheim[3] does not positively assert the influence of the latter, but emphasizes that the complete flaccidity of the muscles favors the development of this paralysis). I saw a case in which paresis occurred

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[1] See Oppenheim, "Lehrbuch," IV. Aufl., p. 448; also Seiffer, *Berl. klin. Wochenschr.*, 1904, p. 818; Ascher, ibid., p. 839; Borchardt, *Verhandl. d. Berliner medic. Gesellschaft*, XXXII, p. 432; and others.

[2] *Berl. klin. Wochenschr.*, 1904, p. 840.

[3] Oppenheim, "Lehrbuch," p. 443.

in the form of Erb's paralysis, and here the anesthesia and abnormal position of the hand had lasted a very short time; operation was performed on account of a severe secondary hemorrhage after appendectomy, and it is quite possible that the extreme anemia of the nerves thus produced rendered them abnormally sensitive to slight trauma. At other times chronic cachexia must be considered. Anesthesia paralysis of the crural, peroneal, and tibial nerves of the lower extremities is more rare than in the upper extremities. Bernhardt[1] described a case of the latter. An operation was performed under anesthesia for prolapse of the uterus; the legs of the patient rested upon supports, the pressure falling upon the popliteal space. Paresthesia and paralysis in the course of the posterior tibial nerve immediately followed. Paralysis after the application of the Esmarch bandage is of similar genesis, but in all of these cases the prognosis is comparatively favorable.

The cases of *maternal neuritis after parturition*[2] represent a special traumatic genesis, and the following case illustrates all of the typical peculiarities.

Case 10.—A woman, aged 24, the wife of a policeman, came under observation on the 6th of December, 1904. This patient, who was otherwise healthy and very strong, was attacked two weeks before delivery (which occurred early in November) with tearing pain in the right leg and the small of the back. She was a primapara, and labor was easy. The puerperal period was normal; there was no fever, no cachexia. Even during the first days of the puerperium and while the pain persisted the patient felt a numb sensation, especially in the posterior side of the right leg, and when she left her bed nine days later this member was very weak. Examination six weeks after delivery revealed the following: continuous pain, numbness, and a cold sensation in the right foot. The nerve trunks in the right leg were very sensitive to pressure, and there was a well-developed sciatic phenomenon. The right Achilles tendon reflex was abolished, the right patella reflex was present, and the tibialis anticus functioned freely; but the extensors of the toes were markedly paretic, the peroneal muscles did not respond, flexion of the toes was imperfect, but that of the foot was good. In the course of the right superficial peroneal nerve there was distinct hypalgesia, while upon the external side of the foot in the region of the sural nerve pin pricks were only slightly painful.

ELECTRIC EXAMINATION.

Faradic Test.

Right.	*Left.*
Peroneal nerve with 100 mm.D., action upon tibial. antic., with 60, action upon extens. digit. comm. and hall., but no effect upon peroneal muscle.	Peroneal nerve 110.
N. tibial. postic. in the popliteal space, 70 mm.D.	N. tibial. postic. 115.
N. tibial. on the internal malleolus with 60 mm.D., reaction in the flexor dig. comm., not in the flexor hall.	
Muscle tibial. antic. 85 mm.D.	Muscle tibial. antic. 100.
Extens. hall. long. 50 mm.D.	Ext. hall. long. 100 mm.D.
Extens. digit. 50 mm.D.	Ext. digit. comm. 100 mm.D.
Muscle quadric. sur. 50 mm.D.	M. quadr. suræ 110 mm.D.
Peroneal muscle, no contraction.	Peroneal muscle, 100 mm.D.

[1] Bernhardt, "Festschrift für v. Leyden," II. Bd.

[2] See Remak, *l. c.*, p. 253; also Hösslin, "Ueber periphere Schwangerschaftslähmungen." *Münchener medic. Wochenschr.*, 1905, p. 636.

GALVANIC TEST.

Right.	*Left.*
Peroneal nerve with 6 ma. action upon tib. ant.; with 15 ma. no further response.	3 ma.
N. tibial. post. in the popliteal space 9 ma.	3 ma.
M. tibial. ant. 10 ma. CaCC., lightning-like.	5 ma.
M. extens. hall. long. 12 ma. CaCC. = AnCC., sluggish contraction.	
M. extens. digit. comm. 12 ma. CaCC. = AnCC., sluggish contraction.	
M. extens. digit. brev. 6 ma. CaCC., AnCC., 8ma., sluggish contraction.	Everywhere lightning-like contraction. CaCC. greater than AnCC. Values between 6 and 10 ma.
Peroneal muscle 15 ma. CaCC., sluggish contraction.	
M. quadric. suræ 15 ma. CaCC., sluggish contraction.	
M. flexor. digit. long. 10 ma. CaCC., AnCC,. 8 ma., sluggish contraction.	

We are therefore dealing with *neuritis of the sciatic nerve*, which affects the branches somewhat irregularly. Muscles innervated by the *peroneal nerve* as well as those supplied by the *posterior tibial nerve* are affected, and sometimes the muscles of both areas are exempt. In maternal neuritis it has been believed that the distribution of the paralysis follows a certain law, intimately dependent upon the etiology of the disease. According to Hünermann, pressure paralysis of the nerves of the hip at birth is prone to occur when the pelvis is narrow. The majority of cases of *obstetric paralysis* have followed the use of forceps. As Remak[1] states, in decisive cases pelvic measurements have been lacking. Even in the case of one of my own patients I was unable to secure the necessary data; but it must be noted that here paralysis appeared after a normal delivery, which is somewhat unusual. According to Hösslin (*l. c.*) among 80 deliveries 66 were abnormal, and in 61 of these forceps were used. Among the cases in which paralysis occurred during a normal birth one is worthy of mention as having occurred in a tabetic. Many of the patients were elderly primiparæ in whom a severe labor is extremely common. In my case the peculiar localization of the paralysis did not conform to our original theory; for, according to this, the area of the peroneal nerve is the first to be wholly or partially implicated (Hünermann and others). But some investigators have described the posterior tibial as being involved. Remak believes the statement that the peroneal nerve is exclusively affected in this form of paralysis to be an exaggeration not borne out by the actual facts. Hösslin declares the implication of the peroneal alone to be extremely rare. In another case that I recently saw the type of paralysis differed from that described inasmuch as there were severe functional and electrical disturbances in the anterior tibial muscle; the other extensors and the peroneal muscles were less severely implicated, the muscles of the calves were paretic, their contractility being only quantitatively diminished, and the Achilles tendon phe-

[1] *L. c.*, page 254.

nomenon was absent. The predilection for the peroneal area is here distinct. An implication of the crural region is usually denoted by neuralgia; paralysis of the obturator has also been observed.

It would lead us too far from our subject to discuss the different hypotheses, or to explain the predisposition of the peroneal region. In several instances of non-traumatic sciatic neuritis, degenerative paralysis has specially attacked this region, so that these cases seem to follow a general law which does not depend wholly upon mechanical conditions.[1]

In my case the first symptoms were observed during *pregnancy.* These consisted of paresthesia and pain in the leg, such as are common in the later stages of pregnancy. Marked symptoms often appear during delivery (according to Hösslin 29 times during, and 35 times immediately after the birth). Even before the termination of labor profound paralysis may be noted. The prognosis is generally good, but is more favorable the more limited the paralysis.

The cases of different genesis developing *post partum* must be here included; following a phlegmasia alba dolens or an excessive parametritic exudate, paralysis may gradually develop in the course of the sciatic nerve, and Hösslin designates this as *neuritis puerperalis per contiguitatem*; the paralysis may be readily differentiated by its slow course and incompleteness. The chief thing here is to demonstrate the local findings.

From these forms of *neuritis, gravidity* or *puerperal neuritis* must be differentiated; these are usually forms of polyneuritis which will be later described. But their close etiologic relations make it necessary for us briefly to consider them. According to Moebius,[2] puerperal neuritis after a simple uncomplicated labor is most apt to implicate the terminal branches of the *median* and *ulnar* nerves. But later observations show that there is frequently another localization. Koester[3] described a case in which the pathologic process was limited to the axillary and musculo-cutaneous nerves; Nothnagel reported one in which the deltoid, serratus, and pectoral muscles were paretic; Moebius observed one in which there was paralysis of the deltoid, of the supra- and infraspinatus, and of the triceps muscles; the facial nerve alone may be affected. Those cases in which the puerperal neuritis affects only one nerve of the leg are difficult to distinguish from the maternal paralyses which have just been described. Since, in the apparently normal labor of our case, a traumatic cause could not positively be excluded, the etiology is uncertain. The fact that the first signs of neuritis appeared during labor is not proof of its nature, for the cause of puerperal neuritis is frequently traced to the period of pregnancy. According to Hösslin, of the cases which developed into *polyneuritis generalisata puerperalis et gravidarum,* 36 cases began before, and 65 after delivery.

Puerperal polyneuritis may often be due to *pregnancy,* especially if this is accompanied by *excessive vomiting.* In its distribution and course it may show all the variations which are observed in polyneuritis, and may resemble

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[1] For many interesting details of the distribution of paralysis in affections of the peroneal nerve see the article by Daus, "Zur Pathologie der Peroneuslähmungen." Berliner I. D., 1902.

[2] Moebius, "Ueber Neuritis puerperalis." *Neurolog. Beitr.*, IV, 1895, Nr. 25.

[3] Koester, "Ueber puerperale Neuritis." *Münchener med. Wochenschr.*, 1896, Nr. 28.

Landry's paralysis (as did a case of Eulenburg's) or may simulate post-diphtheritic paralysis or alcoholic polyneuritis.

In severe cases, as in generalized polyneuritis of other etiology, paralytic phenomena often develop in the lower extremities, but their localization does not conform to the type described by Moebius. When the paralysis ascends, the muscles of the trunk and of the cranial nerves may be implicated, as well as those of the arms. The facial, the fifth, and the optic nerves, the muscles of the larynx, of the pharynx, and of respiration, as well as those of the bladder and rectum, are occasionally implicated. Psychical symptoms characteristic of polyneuritis (Korsakow's syndrome) may be present. Hösslin found these symptoms present in 16 among 40 severe cases; of 50, 10 were fatal.

Etiologically an attempt has been made to differentiate two groups, those occurring in a febrile puerperal state after abortion, and those following a normal pregnancy. Since we are ignorant of the underlying cause in either form, these distinctions are valueless. But, as in the genesis of polyneuritis, the development of a *toxic product* in the *body* (*autointoxication*) appears likely. The frequent appearance of polyneuritis in the pregnant who suffer, or have suffered, from hyperemesis is remarkable. That in these cases polyneuritis must be attributed to the cachexia caused by hyperemesis is not borne out by the facts; nor is cachexia invariably present in other cases of puerperal neuritis.

Returning to *localized neuritis of traumatic genesis* I shall describe a few forms in which, besides the motor symptoms of absence of function, there are others from irritation. The pathologic picture here shows a special implication of the *sensory* fibers, which also occurs in non-traumatic neuritis, as is shown by the following brief clinical histories:

Case 11.—A laborer, aged 57, complained for five weeks of pain and paresthesia in the right hand, limited to the ulnar distribution, and increased by rotation of the hand. The patient could assign no cause for this condition, although he was a moderate user of alcohol. The right ulnar nerve was somewhat broader and thicker than that of the opposite side. The slightest pressure caused typical paresthesia in the area of distribution of the nerve, and its mechanical irritability was also increased. There was no claw position of the hand. Separation of the fingers and adduction were somewhat impeded, especially in the third and fourth fingers, but there was no decided paresis, and only a slight quantitative disturbance of the electric contractility. On the contrary, sensory disturbance was very conspicuous. It was wholly confined to the ulnar region, and affected all the *various sensations*, even that of position. In these cases, which might well be designated sensory neuritis, I would urge the greatest caution in *diagnosis*. The diagnosis of neuritis cannot be based wholly upon pain on pressure; and in differentiating sensory disturbances we must always consider whether or not there is hysterical alteration of sensation. If, as in the case just described, this alteration is limited to the area of a definite peripheral nerve there can be no doubt of the organic and neuritic nature of the affection.

Case 12.—In this instance sensory irritative phenomena were very prominent. The patient was a sailor, aged 30, who, two weeks prior to examination, suffered from an accident. He was carrying on his back a heavy sack which began to slide off. In order to throw the sack on the floor he had to lift it above his head, and this compelled him to bend far forward and to the right. Soon after he noticed a stiffness at the nape, and a little later felt pain in the inner side of the left arm, radiating downward. Gradually paresthesia appeared in the last three fingers of the left hand. Examination revealed the following: the interosseous spaces of the left hand were somewhat depressed. There was slight weakness of the interossei and lumbricales which increased

from the first to the fourth finger; the flexor carpi ulnaris showed weakness and partial DeR. The pupil and palpebral fissure on the left were somewhat wider than on the right. The patient referred his pain to the whole ulnar area of the arm as high as the shoulder. Throughout this entire extent he felt a marked sensation of cold. Brush contact was only slightly perceptible over the ball and pulp of the little finger. There was no hyperalgesia. The paresthesia and pain persisted for about two months after the accident. Paresis was slight. Nothing indicated any injury of the vertebral column.

In addition to the *prominence of the sensory symptoms* this case is interesting because we are not dealing with disease of a peripheral nerve, but of one or two *roots*: the eighth cervical and first dorsal were implicated. As injury of the vertebral column is definitely excluded, we can only assume that there was torsion of these roots, the nature of the accident making this quite possible. Torsion, in a manner previously described, gradually produced neuritis. Such cases are uncommon; often there is profound paralysis, and even more frequently we observe the *plexus paralysis* of which I have already given examples. Such a case of root neuritis, although of much greater extent, was recently described by Déjérine and Roussy.[1] Bloch[2] mentions a case of traumatic torsion neuritis which affected some of the branches of the cervical and brachial plexuses, and specially implicated the sensory fibers. But this diagnosis of root lesion[3] must be cautiously made, and we must assure ourselves that a process starting from the vertebral column or the membranes of the cord (which, of course, might readily injure a root) is not masked by the apparently substantive disease of the root.

In descriptions of neuritis little has been said of *affections of the skin and its adnexa.* Traumatic neuritis presents the greatest variety of nutritive changes in the skin. There are frequently *vasomotor* and *secretory* disturbances as well as trophic. Investigators have given much attention to a change in the composition of the skin called *glossy skin,* which was thoroughly studied by Weir Mitchell. The skin becomes intensely red, then glistening; it is tense without creases or hair, and is most sensitive to external irritation. Usually the patient complains of an acute sensation of burning and of neuralgic pain. Weir Mitchell points out that this cutaneous change is most readily produced when there is *partial* lesion of a nerve which causes continuous irritation. In this opinion many other investigators as well as I concur. After such a partial trauma glossy skin appears with intense sensory irritative symptoms; but, as Remak reports, a total cessation of the function of the nerve may follow such changes. In the most severe case of this kind that I ever saw traumatic paralysis of the ulnar and median nerves led to the complete arrest of nerve conduction. Glossy skin appeared, and, what is rare, the growth of the bones was checked; the phalanges were pointed and thin. By means of the X-rays it has recently been demonstrated that certain osseous changes (osteoporosis) which otherwise elude clinical demonstration are common in peripheral neuritis. It had been previously known that after severe injuries to the nerves in infancy (birth paralysis) there may be a retardation of growth in the affected areas of the member. According to

[1] Déjérine et Roussy, *Revue neurologique,* 1904, p. 619.
[2] Bloch, *Deutsche med. Wochenschr.,* 1902, Nr. 32.
[3] See Oppenheim, "Lehrbuch," p. 445.

Remak[1] glossy skin may possibly be due to *direct inflammatory processes* without neuritic foundation. But if we find such nutritive disturbance limited to a definite nerve region, we may feel assured that it is of neuritic nature. In addition to glossy skin we sometimes note *anomalies in the secretion of sweat.* That these are directly due to the nerves and not to the distention of the blood-vessels is proven by physiology. In neuritis we often find *hyperhidrosis,* and this is even more common in partial traumatic neuritis than in the primary idiopathic form. I shall merely mention the secretion of sweat in disease of the peripheral facial nerve (in which a pathologic condition is relatively often observed), and diseases of the cervical sympathetic in which also there are deviations from the norm.

CASE 13, the history of which I shall now detail, is an illustration of the prominence of *trophic, vasomotor,* and *secretory* disturbances in traumatic neuritis; as a rule, sensory disturbances are also markedly prominent, while the motor are less so. Four years after the death of her husband the patient, who is of decidedly neurotic temperament, attempted suicide by cutting the arteries of her left hand. Since then she has had severe but indistinctly distributed pain in the left hand, with signs of disturbance in nutrition, of sensation, and of motility which will now be described. Immediately above the wrist is a transverse red cicatrix. The third and fourth fingers are flexed at the first and second interphalangeal joints, and at the second interphalangeal joint it is impossible, even passively, to straighten the fingers. There is also a fixed contracture in the interphalangeal joint of the thumb. The second, third, and fourth fingers are bluish red, the skin over these areas is smooth and glistening but quite cool, and the nails of the first, second, and third fingers are said to have repeatedly fallen out. They are now abnormally curved, claw-like, and show longitudinal striæ. The ball of the thumb is atrophic. The cicatrix is still painful on pressure. The patient complains of numbness in the thumb and first two fingers, and on the inner side of the third. Brush contact is not distinctly perceptible on the internal surface of the first, second, and third fingers. There are radiating pains in these regions, also hypalgesia followed by a subsequent burning sensation. On opposition, the power of the thumb is diminished, but that of the small muscles of the hand is normal. Extension of the terminal phalanges, in so far as is mechanically possible, can be carried out, as well as flexion of the middle and terminal phalanges. In the ball of the thumb there is partial DeR.

The case was diagnosticated as follows: *traumatic neuritis of the left median nerve with mechanical sequels and essential sensory and trophic disturbances.* A fine discrimination of these cases may be very difficult, since in addition to the neuritic phenomena there may be mechanical sequels, as in the preceding cases. The latter may be directly due to trauma or to the succeeding inactivity which leads to fixation of the injured extremity; this fixation is frequently caused by the firmness of the bandage applied (*ischemic muscular paralysis and muscular contracture*). We shall soon revert to this. In the case just described the *strict limitation of the sensory disturbance,* the atrophy, and especially the *DeR,* to the muscles of the hand supplied by the median nerve renders the diagnosis of neuritis certain. In all similar cases these symptoms form the corner-stone of the diagnosis, and the proof of conspicuous DeR will always stamp the atrophy as neuritic, and distinguish it from muscular atrophy due to arthritis. The same diagnostic factor renders us valuable service when there are ischemic muscular changes. Among the *trophic* disturbances in this case we note the typical glossy skin, as well as

[1] *L. c.,* p. 133.

changes in the *nails* which are commonly observed in this form of the disease. The nails are thickened, are bent like a talon, often friable, and sensitive to cutting; shedding of the nails, mentioned in our case, has been several times reported. The trophic disturbances may reveal other characteristics, such as ichthyotic change in the epidermis, which has been repeatedly observed. Remak mentioned it in a case of puerperal perineuritis of the median nerve. Löwenfeld described a so-called *neuritic flat-hand,* the chief feature of which was hypertrophy of the subcutaneous cellular tissue. Edematous infiltration has also been reported as a symptom of neuritis. True edema of the dorsum of the hand may be due to the fact that in consequence of paralysis the hand always droops. But occasionally the unusual intensity of this symptom, coupled with its rapid appearance, leads us to assume that other causal and non-mechanical factors are operative. This symptom is also found in other areas which do not become permanently immobile, and are not subject to gravity; we find it in some forms of polyneuritis, particularly in beri-beri, localized in the areas of the peripheral nerves. Consequently there can be no doubt that disturbances in the innervation of the blood and lymph-vessels may, at least in part, account for the development of such edema, which must be included among the vasomotor trophic disturbances of neuritis.

Among these symptoms a few must be described and considered in detail. First among them is *herpes zoster.* The combination of herpes zoster with neuritis is seldom met with. More frequently we find herpes zoster of the neck and head associated with neuritic paralysis of the facial nerve. Eichhorst[1] has compiled statistics which show that zoster occipito-collaris appearing with pain is the most common form. In this an eruption appears in the area of the third and fourth cervical roots (the region of the occiput, ear, and anterior throat region). After two to four days, occasionally later, even after weeks, peripheral facial paralysis appears on the same side. An inverse sequence—first facial paralysis and then herpes—is exceedingly rare, and the zoster eruption is then much less extensive than in the former condition. Up to this time but few cases have been reported of herpes zoster and neuritic paralysis of the extremity; nevertheless they do appear. Remak compiled Eulenburg's cases of this kind occurring since the year 1867, as well as one of his own, also cases of Hardy's and Joffroy's. In Remak's case, simultaneously or a few days after the eruption of zoster femoralis in the region of the crural nerve, paralysis of this nerve developed, and lasted about five months longer than the accompanying zoster. Considering the relative rarity of this form of the disease, I shall report a case of my own which belongs in the same category.

Case 14.—A man, aged 63, was examined upon March 1st, 1904. He reported that on the 4th of January of that year he was attacked by an acute illness with fever and severe pain in the right shoulder. The next morning there was a profuse eruption of small vesicles filled with a transparent fluid; these were first noted on the occiput, and extended to the ear. Shortly afterward a similar rash appeared over the entire right shoulder and right arm, especially the external surface. The side of the thorax adjacent to the arm was covered with the eruption. The pain was said to have been intense. Now there is merely burning and formication. The vesicles, a few of which gradually turned black, disappeared in two weeks. While the patient previously had

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[1] See Remak, *l. c.*, p. 143.

perfect use of his right arm, on the appearance of the eruption he noticed that he could no longer move it at the shoulder- or elbow-joint. In the next 2 weeks this disability had so increased that the patient could no longer make a fist with his hand, which was formerly quite possible. There was no difficulty in micturition. Upon examination a number of lentil-sized dark brown areas were found in the supraclavicular fossa, at the nape, and extending to the third dorsal spinal process, also on the upper arm, the entire external and posterior aspect of the lower arm, and in the radial portion of the hand. Pressure on the plexus caused radiating pain, and the radial nerve also was sensitive to pressure. There was neither hypesthesia nor hyperesthesia to brush contact, but a conspicuous hyperesthesia to needle pricks, especially on the external side of the arm. The right shoulder was flattened; there were no tendon phenomena in the right arm. The response in Erb's muscles was imperfect, the deltoid being even inactive. The muscles supplied by the radial and ulnar nerves were functionally intact. On the other hand, the long flexors of the fingers, the radial flexors of the hand, and the apposition of the thumb were weak. Faradic contractility was diminished in the deltoid, less so in the supinator longus and biceps. Contraction upon galvanic stimulation was distinctly slowed in these muscles, as well as in those supplied by the median nerve.

The characteristic reports of the patient, as well as the examination, made it plain that *herpes zoster in the region of the right cervical plexus* had at once been followed by Erb's paralysis. It is noteworthy that while the herpes zoster receded in the usual way, yet even six weeks after its onset new eruptions appeared with the same paralytic phenomena; this showed the implication of another root in the plexus. I know nothing further of the case as the patient did not return for treatment.

This combination of positive neuritic paralysis with herpes zoster makes a neuritic origin seem plausible in the pathogenesis of herpes. We know that the discussion of the foundation of herpes has led to many controversies. Certain investigations of Bärensprung indicated that it might depend upon a *disease of the spinal ganglia.* This view was confirmed by researches which Head and Campbell [1] instituted upon a broad anatomico-physiologic basis. But a few other anatomical investigations have shown that, even with an intact spinal ganglion, *peripheral neuritis* may lead to herpes zoster. I cannot minutely discuss the development of herpes. Whether in a case like that just quoted herpes zoster may be regarded as a purely neurotic symptom is difficult to decide. I believe it must be admitted that the same cause may here produce *on the one hand a disease of the peripheral nerve, especially of its motor portion,* and on the other hand a disease of the sensory conduction tract, without our coming to any decision as to whether the latter is seated in the spinal ganglion or in the fibers which pass from it.

Among trophic disturbances is perforating ulcer of the foot (*mal perforant*)—an ulcer which forms on the sole of the foot and penetrates deeply in funnel shape; it has thickened edges, aften develops painlessly, and is exceedingly slow in healing. This ulcer has been repeatedly observed following injuries to nerves, especially to the sciatic, also after compression of this nerve by a tumor, while it has never been demonstrated with certainty [2] in idiopathic neuritis, whether mononeuritis or polyneuritis. An exception to

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[1] Head and Campbell, "The Pathology of Herpes Zoster and its Bearing on Sensory Localization," *Brain*, 1900, p. 351.

[2] Adrian, "Das Mal perforant." Jena, 1904, Gustav Fischer.

this rule is the formation of a perforating ulcer of the foot in leprous neuritis; the appearance of ulcer in diabetic neuritis can probably be otherwise explained. In the development of this trophic disturbance we must consider another factor than a change in the nerve influence, and this is the effect of pressure, but only upon tissue whose power of resistance is damaged by nervous or other influence. As Remak maintains, mal perforant is of decided clinical importance in neuritis and polyneuritis, but not as an exclusive symptom of clinical mononeuritis of the sciatic, and it is still rarer in the case of polyneuritis.

The same considerations apply to *bed-sores*. Changes in peripheral nerves in the area surrounding a bed-sore have often been demonstrated (Déjérine and Leloir).[1] Although the causal significance of this change is still doubtful, certainly bed-sores have not been observed as a clinical symptom of neuritis, and the same may be said of other trophic disturbances, especially *gangrene*. Many cases have been reported in literature, especially in connection with the investigations of Pitres and Vaillard, but none has proven that gangrene depends upon neuritis;[2] there is still less justification for regarding certain peculiar forms of gangrene, particularly *Raynaud's gangrene*, as symptomatic of neuritis. How far other symptom complexes which belong to vasomotor trophic neuroses, particularly *erythromelalgia*, *asphyxia*, and local syncope, depend upon neuritic changes cannot here be stated. It is certain that these disturbances play no rôle in ordinary neuritis; but it is also probable that some cases which may be grouped with erythromelalgia, and in which the symptoms are confined to certain peripheral nerve regions, are due to disease of portions of special peripheral nerve fibers.[3]

There are cases which are most obscure because, in addition to the symptoms of traumatic neuritis, there are other pathologic phenomena which can be referred neither to trauma nor to the treatment which the injured extremity has received. If the trauma was fracture of a bone, the treatment of which is usually absolute rest of the member and a firm bandage, there may have been simultaneously torsion of a nerve; if a growing callus subsequently compresses the nerve, on removing the bandage we often find not merely the signs of a neuritic affection, but also those of *ischemic muscular paralysis* and *muscular contracture*. At first there is usually severe pain, the hand which remains free becomes swollen, and the fingers are implicated; then the muscles rapidly atrophy, become *hard as wood* and rigid, and the affected parts assume the fixed position of contracture. Electric examination reveals only quantitative changes in the contractility. There are no sensory disturbances. These symptoms enable us to distinguish ischemic muscular paralysis from the neuritic form. When these forms are combined, in which case ankylosis accompanied by muscular atrophy and cicatricial adhesions of muscles and tendons may appear, the pathologic picture may be disguised. I have most frequently seen this combination in fractures of the elbow-joint in young persons.

CASE 15.—A child, 11 years old, suffered a year previously from fracture of the right arm involving the elbow-joint. After removal of the splint, severe disturbances in the motility of the right arm were noted. Only the movements of the shoulder were

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[1] Cassirer, "Die vasomotorisch-trophischen Neurosen," p. 89.
[2] Cassirer, *l. c.*, p. 312. [3] Cassirer, *l. c.*, p. 186.

both actively and passively free. Pronation and supination were mechanically limited in the elbow-joint, and the wrist-joint was almost absolutely rigid in a flexure position. The right hand and forearm were cooler than the left and cyanotic; the muscles were firm and hard. The finger-joints showed many ankyloses. That the changes were not wholly arthritic and ischemic was proven by the fact that the muscles of the ball of the thumb were free, while the joints acted imperfectly, and there was partial DeR; a disturbance of sensation limited to the region of the median nerve was also demonstrated. The circumstance that, in addition to all other lesions, some area of the nerve itself may be damaged is naturally of great significance in the treatment, but I shall discuss the therapy of neuritis later.

Another form of neuritis has often been described; but, while its occurrence is not actually doubted, it must be regarded as extremely rare; I refer to *ascending neuritis.* Ever since the time of R. Remak, we have understood by the designation "neuritis ascendens" those neuritic symptoms which proceed from a primary and distally situated disease of a nerve and which extend upward, and may be referred to perineuritis or neuritis interstitialis. This was said to be distributed, not in the path of the nerve, but irregularly, even to other nerves and to the spinal cord. Our experience with traumatic neuritis showed this to be based chiefly upon the palpable swelling of the nerve found in such cases. The term ascending neuritis was by no means limited to traumatic cases, but was given a much wider application. Later researches have thrown discredit upon this teaching, and in 1895 Moebius pronounced it traditional. But in this he seems to have gone too far. True, many of the assumptions of our earlier conception were untenable, but it has been experimentally demonstrated that in purulent neuritis it is *possible* for the process to ascend from the periphery to the center. Homén and Laitinen have proven that streptococci and their toxins introduced into the nerve sheaths reach the spinal cord in the tract of the nerve and spinal cord roots, and this coincides with the results of the early researches of Rosenbach, Treub, and Kast. Marinesco[1] reported a notable case which led him to similar conclusions. Here there was gangrene of the right leg, and just where the internal popliteal nerve was embedded in these gangrenous foci numerous streptococci were found. A marked infiltration of leukocytes was demonstrated in the central portion of the sciatic nerve, and was most distinct in the right side of the spinal cord in the posterior external vessels of the anterior gray substance. In his opinion the microbes generated in the nerve a substance which was disseminated along the nerves as far as the spinal cord, where it produced anatomical changes in the vessels and nerve cells. Therefore, this was an ascending neuritis in the true sense of the word. Recently other authors have reported cases in which a neuritis proceeding centrally was assumed to be present. Remak[2] published such a case, and this was also seen by Oppenheim. The patient had cut his index finger, and urine had been applied to the wound. Pain soon appeared, and extended to the shoulder; two weeks later Oppenheim found a paralysis of the radial nerve in the corresponding arm, and three months later when examined by Remak the biceps also was paralyzed. Kausch, Gerhardt, and Krehl have described

[1] Marinesco, "Contributions à l'étude de la névrite ascendante." *La Presse médicale,* 23. November, 1898.

[2] Remak, *l. c.,* p. 265.

similar cases. Redlich[1] also demonstrated ascending neuritis in one of his cases. Here, after a fall upon the right elbow, there was paralysis in the region of the median and ulnar nerves, while all of the muscles of the arm with the exception of Erb's muscles were subsequently attacked. In this instance one factor was absent which was present in all of the other cases—i. e., there was no open wound—while in the others the process originated in an open and, moreover, infected wound. It is certain that the diagnosis of ascending neuritis can be made only after most careful investigation and consideration. At most it is an extremely rare affection. Special diagnostic care is necessary in the cases in which sensory symptoms alone follow the trauma; as when there is merely sensitiveness of the nerve to pressure and hypesthesia. Here we must always consider whether or not the symptoms are due to traumatic hysteria, and it is astonishing how often this diagnosis will be found correct.

Another uncommon form of *traumatic neuritis* is inflammation of the nerve from the *subcutaneous injection* of ether. This is usually due to a careless or two deep injection through failing to pinch up a fold of the skin, hence the ether penetrates the fascia. In the arm it always produces paralysis of the radial nerve, while, as a rule, the supinator longus and the extensor carpi radialis longus are unimpaired. The other muscles supplied by the radial nerve are affected differently. Pain and objective sensory disturbances rarely appear. The paralysis is only moderate, there is partial DeR, and in the overwhelming majority of cases recovery takes place in one or two months. Neuritis has occasionally been observed after the injection of other substances, such as *chloroform, hyperosmic acid, corrosive sublimate, calomel, biniodid of mercury,* etc. The cases due to the last drug are caused by injections in the gluteal regions, and the paralyses affect the sciatic nerve or its branches; therefore we should be most careful when giving these injections. The pathogenesis is here readily understood; the nerve is directly altered by the injected chemical agent. In a pathogenetic sense these cases must be distinguished from the previous forms of traumatic neuritis.

Occupation Neuritis.—The cases which we designate as occupation neuritis (*professional paresis*) occupy a special position. We apply this term to *all those injuries of the peripheral nervous system which are caused by a certain occupation, and in which we can demonstrate other typical symptoms of neuritis.* Among these we first note degenerative atrophic paralysis to which are often added sensory, and occasionally also vasomotor, secretory, and trophic irritative symptoms, and those due to absence of function. Of course we cannot deny that there is a purely sensory occupation neuritis, since this finds its analogy in a sensory form of other localized forms of neuritis. But we must approach this subject with great circumspection, for its differentiation from neuralgic and *occupation neurosis* is most perplexing. We know that exhaustive labor leads to derangement of those parts which are excessively used. But the affections designated as *occupation neurosis* I shall consider in a special chapter.

In the development of occupation paresis, various and by no means simple

[1] Redlich, "Zur Kasuistik der traumatischen Neuritis." *Wiener klin. Rundschau,* 1902, Nr. 16.

factors are operative. I consider these in connection with traumatic neuritis because the pressure exerted upon a nerve trunk or its branches is frequently of pathogenetic importance. We have learned to recognize the damage wrought by continuous pressure. To these pressure paralyses or forms of neuritis due to pressure, to compression by a fragment of bone, by a callus, or by an Esmarch bandage too tightly applied, we may add others which, in their mode of development, form a transitional stage between the first mentioned forms and occupation paresis. Here I must mention the so-called *crutch paralysis* —paresis due to the pressure of a crutch in the axilla—which implicates the triceps and causes a decided radial paralysis.

Pressure upon the terminal twigs of the median and ulnar nerves sufficient to produce paralysis may be caused by an improper cane carried in the hand and used as a support. *Paralysis of the serratus,* due to the bearing of loads upon the shoulders, as in *carpenters* who carry *beams,* in *butchers* who lift *heavy meat,* and in other workers who move *sacks,* is more closely related to occupation pareses. A number of similar localized paralyses particularly involve the brachial plexus. Rieder (after Remak)[1] described as hod-carrier's paralysis a plexus paralysis particularly involving the axillary and radial nerves; this is due to the pressure of the hod which rests upon the clavicle, and is most common on the left side; in young laborers the clavicle is still yielding, and compresses the brachial plexus. Coal-heaver's paralysis, described by Osann, is of similar origin, as well as the paralysis which Curschmann[2] reported in a man who carried trees upon his shoulders. On the left side it affected Erb's muscle; on the right side only the deltoid. I recently saw in a wood-carrier paralysis which affected chiefly the left radial region. The acute, almost apoplectiform, appearance of the paralysis and its course tending toward improvement, which was very slow, were noteworthy. But in these cases, besides the mechanical factor we must often include a toxic one in the form of chronic alcoholism. In a somewhat restricted sense, *over-exertion* plays the chief rôle in professional pareses. How this is brought about, Edinger attempted to explain by his *substitution theory.* There can be no doubt that in the activity of the nervous system processes of metabolism occur in which definite substances are utilized, and these accordingly require renewal. In the form of activity designated as exercise, this renewal is normally sufficient, and the affected parts are strengthened or undergo higher development. In *over-exertion,* this substitution is insufficient, and decomposition takes place in the peripheral nerves, and affects the medullary sheaths and the axis cylinders. As confirmation of his views, Edinger mentions the fact that those persons who suffer from occupation paresis are from one cause or another under-nourished. Undoubtedly we must agree with him that, besides over-exertion, other factors are frequently operative in the development of this paralysis, some of which we have already mentioned, and which will be more clearly understood from a few illustrations. This explains why comparatively so few persons engaged in the kinds of work spoken of are attacked by occupation paresis. These examples also show us in how far other factors, besides

[1] *L. c.*, p. 282.

[2] Curschmann, "Beiträge zur Lehre von der Beschäftigungsneuritis." *Deutsche med. Wochenschr.*, 1905, pp. 570 und 634.

injurious pressure, are responsible for the development of the pareses. In regard to the localization of these affections, of course the muscles of the hand supplied by the median and ulnar nerves are most frequently affected, but the location varies, and only the cerebral nerves have so far been exempt.

CASE 16.—The following case is calculated to reveal the most important etiologic and symptomatologic phenomena of professional pareses. It is the case of a woman, aged 25 and very pale, who was employed in the manufacture of dress trimmings. She was obliged to hold a round wooden block in the hollow of the left hand, grasping it tightly with her fingers. At the same time the hand had to be flexed at the wrist-joint—that is, she had to bend the wrist frequently. She worked in this way 12 hours daily for 8 years. For 4 years she suffered from tearing pain in the left arm, and in the last 6 months there was weakness and increasing emaciation in the left hand. Five weeks ago she stopped work, but there has been no improvement. On examination degenerative paralysis was found in all of the small muscles of the hand, which were decidedly emaciated, and showed partial DeR. The plexus was not painful on pressure, but the ulnar and median nerves were sensitive to pressure at the elbow and the wrist-joint. Pressure upon the muscles of the vola manus was particularly painful, and upon passive extension this pain radiated to the hand and fingers. In the ulnar region of the hand, particularly in the little finger and its ball, there was hypesthesia for tact and pin pricks. Notwithstanding the fact that the patient had long since discontinued her work, there was no improvement in the paresis, but the pain had lessened. After about 6 months, the paralysis began to yield. The patient did not continue under observation.

We note here that *pressure* and *excessive exertion* on the part of a *poorly nourished* and *anemic* person combined to produce occupation paralysis. Paralysis is now the most prominent symptom, but *pain had for years preceded*, as has often been observed by other authors, so that it may be regarded as characteristic of these cases. In addition to the pain there were objective sensory disturbances and tenderness of the nerve trunks on pressure. The fact is also remarkable that pressure upon the vola manus was painful, and although this does not actually prove the existence of a more or less substantive myositis (neuromyositis) in addition to the neuritis, yet many experiences seem to indicate that myositic as well as neuritic symptoms appear in this disease; and when we consider the nature of the damage sustained, it appears quite plausible. There is a form of occupation paralysis, recently reported, in which the processes seem to run their course in the muscles or tendon sheaths; this is the *drummer's paralysis* described by Bruns and Zander, and usually affecting the extensor pollicis longus, more rarely the flexor pollicis longus.

A pathogenesis similar to that in the history just detailed is found in the common *paralysis of laundresses*, which manifests itself by atrophy and weakness of the muscles in the ball of the thumb, of the adductor pollicis, and of the interosseous primus. In addition to excessive use, these are attributed to pressure in grasping the laundry iron. Similar neuroses are found in many other occupations—for example, in *gold burnishers*, in *carpenters*, in *smiths*, etc. I saw a similar picture in a man, aged 38, who for 2½ years had worked 8 or 9 hours a day at stamping letters, while for 6 years previously he had sorted letters; a somewhat similar form of occupation paralysis has been described in *letter sorters*. This patient had suffered for years from pain which radiated extensively, also paresthesia and slight object sensory disturbances.

Case 17.—I shall here describe a form of paralysis limited to the distribution of the median nerve, and it is in many respects interesting. It is the case of a tailor, aged 37, who, upon the 26th of August, 1902, sustained a knife cut in the left forearm. The blood loss was insignificant. On the morning of the 17th of September, he was unable to flex the first three fingers of the *right hand*, whereas during the night, while asleep, as was his habit, he had placed the arm under his head. As a cutter he was obliged to use heavy scissors with his right hand. There was slight median paresis, with distinct objective sensory disturbances and slight electrical changes. The paralysis disappeared 3 months after stopping his work. Nine months later he was again suffering from radial paralysis which appeared suddenly, apparently from torsion. From this also he recovered.

We notice how many etiologic factors must be considered in this case, for, besides loss of blood and consequent anemia, pressure on his arm during sleep, over-exertion, and pressure from the heavy scissors were also operative. It is remarkable, too, that after a brief time another nerve became paralyzed after a slight injury; this indicates a lessened power of resistance in his peripheral nervous system.

Remak saw a case in some respects analogous. A lathe-worker, who worked at a plane, and used alcohol in moderation, had a bilateral ulnar paralysis which disappeared 4 months after he stopped his work. After a year and a half he returned with a right-sided median neuritis due to the continuous use of a brush, and from this also he recovered. Special etiologic factors were noted in the next case.

Case 18 is that of a laundress, aged 22. In her youth, this patient had suffered from a traumatic peripheral paralysis of the left ulnar nerve, and 3 weeks after beginning her present work a median neuritis appeared. The sudden onset of this hyperexertion neuritis is explained by the fact that in consequence of the preceding paresis the patient was compelled to over-exert the small muscles of the hand.

Case 19.—A somewhat unusual cause was noted in the case of a woman, aged 49, who presented bilateral atrophy in the distribution of the median nerve. She had been obliged frequently to lift heavy pots. She admitted having consumed a great deal of coffee, but denied the immoderate use of alcohol. Here the excessive use of coffee may have caused the paralysis.

It has been reiterated that chronic intoxication is a common cause of this affection, and it is proven by numerous cases. Besides *chronic alcoholism* and *nicotinism, chronic occupation intoxication*—for example, with lead in paper glazers—has been mentioned. Other predisposing factors are the following: Chronic diseases which lower the general nutrition; at other times a lessened power of resistance of the nervous system, especially the peripheral portions. Tabes has been repeatedly noted as a cause. I once treated a tabetic tailor for occupation paralysis. Remak mentions tabetic cigar-makers; Oppenheim reports the case of a patient in whom, during convalescence from influenza and when she undertook to carve wood, atrophy appeared in the small muscles of the hand. Sano[1] reported the case of a painter, who held his palette in his left hand, and who, during the course of scarlet fever, became paralyzed. The muscles most used in his work—on the right side the muscles of the shoulder-blade, particularly the serratus anticus and deltoid, and on the left the biceps—were affected. Perhaps lead poisoning, to which his work exposed

[1] *Journ. d. Neurol.*, 5. Mars, 1898.

him, was also operative. Oppenheim believes that patients who have had acute anterior poliomyelitis in childhood are specially predisposed to occupation paralysis. He treated a man suffering from poliomyelitic paralysis of the right leg who supported himself with a cane carried in the right hand, and who presented an atrophic paralysis of the muscles of this hand; under rest of the muscles and electricity it improved.

In such patients symptoms of general nervousness are often present, and occasionally even the marked stigmata of a hereditary neurotic diathesis. To this I attribute a marked kyphoscoliosis which I observed in one of my patients, a seamstress, who suffered from occupation paresis, the same deformity being presented by several other members of her family. Remak states that he has found paralysis to be very common in persons with this deformity. Possibly improper technic may be responsible for these disturbances; in young girls chlorosis and anemia are frequent accompaniments. Hence it is evident that the etiologic secondary factors in the development of occupation pareses are manifold.

Symptoms.—Following this discussion of the etiology, I return to the *symptomatology* of occupation pareses. Of course I cannot enumerate all those callings in which pareses follow the excessive use of the small muscles of the hand. In those mentioned, pressure as well as over-exertion led to the paralysis. In a case of Curschmann's, the patient worked on metal clocks, and combined with pressure and over-use of the muscles shock from continuous light blows on the surface of the hand produced paresis of the left ulnar nerve. In other cases no cause was apparent save excessive use; for instance, the pareses of *seamstresses, embroiderers, cigar-makers, and diamond-cutters.* In cigar-makers, as Coester first pointed out, paresis of the median nerve may also implicate the ulnar nerve. Pains often long precede, as well as vasomotor symptoms, especially numbness of the finger. In cigar-makers I have frequently observed this local syncope combined with acroparesthesia and pain, but without paralysis. The chief cause of the vasomotor symptoms seems to be the fact that these patients are compelled to work with wet, cold fingers. When paralytic symptoms and objective sensory disturbances peripherally distributed are lacking, it is doubtful whether we are justified in assuming a neuritic basis for the affection. But the discussion of this question would include acroparesthesia in general, upon which I cannot here touch.[1]

It is well to bear in mind that acroparesthesia is occasionally merely a preliminary stage of subsequent and true degenerative paralysis and neuritis. This has been described by Raymond and Courtellemont in a coachman,[2] and by Lévy and Wormser in a bicyclist.[3]

A localized degenerative atrophy of the interosseous primus and secundus combined with sensory disturbances, particularly in the little finger, was observed by Wertheim-Salomonson in *diamond-cutters,* these workmen being compelled to hold the diamonds with both hands. To milker's spasm, an occupation neurosis, neuritic paralysis and sensory disturbances may be added. Mild *motor irritative symptoms* are by no means rare in occupation pareses.

[1] See Cassirer, "Vasomotor Trophic Neuroses," in this volume.
[2] *Rev. neurol.,* 1904, p. 500. [3] *Rev. neurol.,* 1904, p. 1219.

A degenerative paralysis localized in the small muscles of the hand supplied by the ulnar nerve is observed in *glass-cutters, engravers, lathe-workers,* and *xylographers.* Although the point of lesion in these cases is to be sought at the elbow-joint, the nerve being here pressed against a hard, underlying surface and thus irritated, nevertheless, as Bruns asserts, only the terminal branches of the nerves in the hand are as a rule implicated. If continuous pressure causes inflammatory processes to develop in the tissues covering the nerve, the damage to the nerve is even more intense. For instance, Curschmann reported the case of a gold polisher, in whom a bursitis with induration had developed over the ulnar nerve; of course such occurrences greatly retard the disappearance of the paralysis.

Occupation pareses more rarely implicate the nerves of the upper arm than the small muscles of the hand. Bernhardt[1] reported left-sided radial paresis in a waiter. This patient, for an entire winter, had carried plates in the left hand, which was strongly flexed dorsally while his head was turned toward the right. A weaver, treated by Oppenheim, had paralysis of the right triceps due to the fact that he was compelled to extend the lower arm about 20,000 times daily. The same author saw paralysis of the deltoid and the supra- and infraspinatus muscles in a railroad guard whose business it was to depress the lever of a signal. Paralysis of the serratus from over-use in various occupations has been described by Bernhardt and Remak. Hoeflmayr[2] described cases of occupation neuritis which implicated the brachial plexus (the axillary nerve) in carpenters, tanners, and watchmakers.

Paralyses of the *lower extremity* from over-exertion are much more rare. I recently saw a case *implicating the crural nerve* which I must regard as *occupation neuritis.*

Case 20.—A smith, aged 42, and healthy, was compelled while at work to fix his left leg firmly at the knee-joint and to extend it; at the same time with considerable force and various forward and backward movements of the trunk he swung a heavy hammer high above his head. For a year he had complained of weakness in the left thigh. Walking was difficult, especially the ascent of stairs. He also felt dull, unpleasant sensations on the anterior surface of the thigh. I found the typical signs of crural neuritis—sensitiveness of the nerve to pressure, atrophy of the quadriceps, markedly decreased knee-jerk, weakness of the ilio-psoas and quadriceps, partial and mild DeR in these muscles, and a decided disturbance of sensation in the thigh along the course of the crural nerve, and over the saphenous major on the inner side of the leg and foot. Here the affection was attributed to over-exertion combined with turning and torsion of the nerve, especially since all other etiologic factors were absent.

Except under such circumstances, professional paresis in the muscles of the thigh is extremely rare. I must call attention to a case reported by Curschmann of professional paresis in a stone breaker and farmer; this extended along the course of the peroneal and tibial nerves, and implicated the crural nerve. He localized the injury as occurring in the flexor of the groin, and attributed the compression of the nerve to constant kneeling. Occupation pareses in the *nerves of the leg* are somewhat more common. I saw a tailor who, two weeks before he came under treatment, was suddenly attacked by paresis of the right thigh followed by weakness and degenerative paralysis of

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[1] Bernhardt, "Festschrift für v. Leyden," II.
[2] Hoeflmayr, *Münchener med. Wochenschr.*, 1901, 45.

the muscles of the right leg; the latter affected chiefly the posterior tibial, but there was no sensory disturbance. The patient reported that his work compelled him to flex the right leg against the thigh as much as possible, and at the same time to cross the left leg over the right. The paralysis improved after the patient suspended his work; here excessive use of the muscle combined with pressure was the cause. A similar origin for the paresis was reported by Bernhardt [1] and Jones in saddlers. Muthmann quotes a case described by Bernhardt of professional and bilateral neuritis of the peroneal nerves in a patient, aged 28, who had for years used his leg to turn a lathe; he usually stood on his left leg while he turned the machine with the right. The neuritis was most marked on the left side. I saw a woman, aged 35, the wife of a painter, and poorly nourished, who 3 months previously was suddenly attacked by weakness in the left ankle-joint and a numbness in the dorsum of the foot. I found a decided peroneal paralysis, all the muscles supplied by the peroneal nerve being uniformly implicated. The nerve was sensitive to pressure; on the dorsum of the foot there was distinct hypalgesia. There was partial DeR. The patient had previously sewed on a machine for about 15 hours daily. A similar case was described by Charcot and Meige. In some cases the genesis of this form of neuritis is not always clear. In addition to over-exertion, pressure and torsion have a deleterious effect. An origin similar to that of the peroneal paralysis from the use of the sewing-machine was reported by Raymond and Courtellemont [2] as following excessive bicycling. The most common form of occupation paresis in the lower extremity is probably observed in *potato diggers, farmers, brick-layers,* and the like —paralyses due to the kneeling posture necessary for the work of these men. This was first described by Zenker, then by Kron, Hoffmann, Remak, and Bernhardt, finally by Schultz [3] and Schleutzka and Curschmann (*l. c.*).

Case 21.—The following case is an example of this type of professional paralysis. The patient, a girl aged 20, was engaged in planting beets when in May, 1902, with symptoms which came on gradually she was taken ill. She noticed that her right foot clung to the ground, and that the dorsum of the foot was numb. On continuing her work she felt severe pain in the foot. The affected right leg was the one she was compelled to use most. I found paralysis, chiefly in the peroneal region, but also implicating the muscles of the calf. The Achilles tendon reflex was abolished; there were electric disturbances which were most marked in the extensors of the foot and toes, but also distinct in the muscles of the calf.

In these cases the peroneal nerve is the one chiefly implicated, but other investigators as well as I have known the posterior tibial nerve to be affected. The reason why the peroneal nerve is usually attacked is that in kneeling the tendon of the permanently contracted biceps presses the nerve against the head of the fibula. The posterior tibial nerve is less severely compressed by the persistent contraction of the gastrocnemius. Schultz asserts that, besides mechanical factors, such as a kneeling posture, other causes are operative; for instance, when planting beets the person usually kneels upon the cold, moist earth while he is frequently over-heated and perspiring.

[1] "Die Erkrankungen der peripheren Nerven," 2. Aufl., I, p. 514.

[2] *L. c.*

[3] Schultz, "Ueber Fusslähmung, speciell Peroneuslähmung bei Rübenarbeitern." *Deutsch. Arch. f. klin. Med.*, 1904, Bd. 80.

Diagnosis.—It is unnecessary again to discuss occupation pareses in general, and only a few differentio-diagnostic considerations are called for. Oppenheim [1] states that the condition is not infrequently confounded with spinal progressive muscular atrophy. The points of differentiation are the following: occupation pareses are usually, although not invariably, unilateral. Paresthesia, pain, and objective sensory disturbances in the affected nerve regions are common, and are often permanent. If, as sometimes happens, a differentiation is not at once possible, the *course* of the disease must be considered. In the great majority of occupation pareses the prognosis is favorable if the patient can abandon his work; if the atrophy has not existed too long, it then usually disappears. As a rule the cases chiefly due to pressure run an especially favorable course; and, in consonance with this, the changes in the electric contractility are usually slow—as is also the case in non-professional paralyses from pressure. When the pareses are due in the main to over-exertion, the course is often very prolonged, and does not become aggravated. In a case that I observed the diagnosis of occupation paresis was indicated only by the course. Here, combined with indefinite sensory irritative disturbances and degenerative atrophy, there was a weakness of the right triceps pectoralis and teres major. No objective sensory disturbances were present, nor was either muscle or nerve painful on pressure; certainly not when I first saw the patient. He had strained these muscles bilaterally by athletic exercises. It was impossible at first to decide whether the case was *chronic anterior poliomyelitis* at the height of the seventh cervical segment, whether spinal progressive muscular atrophy had gradually taken place, or whether it was neuritis from excessive use of the muscles. After being under observation for several months, the course indicated that the last view was correct. The differentiation from *syringomyelia* may be even more difficult since in this disease there are motor as well as sensory disturbances. The marked limitation of the motor as well as sensory disturbances to peripheral regions of innervation, the exemption of proximal parts of the upper extremity, the unilateral implication, the conspicuous sensitiveness of the nerve to pressure, all favor occupation paresis; oculo-pupillary symptoms, severe trophic disturbances in the bones and skin, thermo-anesthesia, and spastic symptoms in the legs favor the diagnosis of syringomyelia. In doubtful cases prolonged observation is most necessary.

Treatment.—The therapy primarily demands that the work which produced the disease must be abandoned. This should be done as soon as possible, best during the stage showing sensory irritative phenomena, which, however, is not always practicable. The treatment is the same as for other forms of neuritis. *Rest* is the first and chief remedy, whether we are dealing with spontaneous, infectious, or traumatic neuritis. It is obvious that all motion, every muscular contraction, will intensify the inflammatory process. We must give the diseased member rest, and, on the other hand, we must see that there is no pressure upon the diseased nerve and muscles. Therefore a firm bandage cannot be used, but, as Edinger proposes [2] a relative position of rest

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[1] "Lehrbuch," p. 247.

[2] Edinger, "Behandlung der Krankheiten in Bereiche der peripheren Nerven." *Penzoldt-Stintzing's Handb.*, 1. Aufl., Bd. V, p. 568.

—as in bed—for the legs, and a sling for the arm, is much better. In traumatic neuritis, in which the nerve lesion necessitates that the bone and joint be firmly bandaged, direct pressure or torsion of the nerve must be carefully avoided, and we must guard against even the slightest compression of the paralyzed muscles.

When there is a rheumatic or infectious etiology, general *diaphoresis* according to the ordinary methods is highly recommended by Oppenheim, who believes in removing the deleterious substance from the organism as soon as possible. For local relief, derivatives to the skin and occasional *blood-letting* with wet cups or, better, with leeches may be tried. We must be very cautious in the use of vesicants, particularly over anesthetic areas of the skin. Local inflammation may also be relieved by various hydriatic procedures. As a rule, cold in some form should first be employed, preferably ice-water compresses, while the application of chemical agents to produce a sensation of cold (as ethyl chlorid spray, etc.) should be reserved for the later treatment of pain. When cold increases the pain—there is no rule for this—*moist heat* in the form of packs or hot compresses, bags of sand, local hot baths, and hot air (with Tallermann's apparatus) should be resorted to. That these have any direct effect to influence regeneration favorably is doubtful. Certainly with one or the other method we may frequently lessen the severe inflammation and nerve pain. Some drugs, particularly *arsenic* and *strychnin,* have had ascribed to them a paralyzing action upon toxins. Remak quite properly doubts this, but, nevertheless, advises their occasional use in small doses. To relieve the pain, we require other remedies, among which the newer *anti-neuralgics* are especially active. Sodium salicylate has occasionally been regarded as an absolute cure for rheumatic and infectious neuritis; as belonging to this category I must also mention antipyrin in doses of 7½ to 15 grains, salipyrin, lactophenin, trigemin, and especially aspirin in the same dose; in small doses phenacetin (7½ grains), antifebrin (3 grains), and pyramidon (3 to 5 grains). The last remedy frequently has an excellent effect, but, as with all of the others, the patient must be carefully watched. Only on the rare occasions when there is severe pain in the lumbar or sacral nerves should we resort to epidural injections of cocain, as Chatélineau advised. In some cases we find it impossible to get along without an occasional injection of morphin.

Electricity is generally regarded as the agent which, above all others, is calculated to stimulate the regeneration of a diseased nerve. This is not the place at which to discuss electrotherapy theoretically. Practically, except in the mildest forms of neuritis, there is scarcely a case in which electro-therapeutic treatment is unnecessary. In the earliest stages when pain is severe, experienced authors advise the use of only the constant current. For the amelioration of the pain the stabile employment of the anode has in many cases proven effective. An anode having a diameter of 10 to 20 cubic centimeters should be applied to the most painful area, while the broader cathode is placed at an indifferent point. When a nerve of great extent is susceptible to the electric current—for instance, the sciatic—the cathode may be applied at some other point of the nerve itself, so that the nerve will be, as it were, between two electrodes. After the current has been allowed to pass through the entire extent of the nerve, by slowly moving the electrodes we may occasionally increase the anodyne effect. The current is to be gradually increased to

the necessary strength of 4 to 6 milliampères, and all marked fluctuations therein are to be avoided. In the later stages we may apply stronger currents. The duration of the sitting should at first be for about 5 minutes; this may be increased to 20 minutes or longer, and even alternating currents may be employed. During this time unpleasant sensations of numbness can be relieved by the occasional use of the faradic brush; even permanent results are attained. Electrical treatment of neuritic paralysis should begin after the severe irritative symptoms subside. Whether it facilitates the histologic regenerative process is uncertain, but experience has shown that, at the time when anatomical restitution is to be expected, suitable electrotherapy may decidedly benefit the motor function. Remak [1] maintains that a diseased nerve which has been subjected to the influence of the constant current is much more readily innervated voluntarily than prior to this treatment (investigations in so-called pressure paralysis of the radial nerve). In such cases it is best to institute *labile cathodal treatment*, perhaps combined with stabile anodal treatment. Where faradic irritability is not too markedly lessened, the faradic current also is indicated. In the treatment of motor paralysis, particularly its later stages, *massage* is often used; this is said to produce a mechanical irritation of the paretic muscles and an alteration in the circulation, also to prevent deformities from contracture of the antagonists. From the onset the development of these deformities should be counteracted by a suitable position and the bracing of the paralyzed parts; later, in addition to massage, gymnastic exercises (active and passive movements with and without apparatus) should be advised.[2] In the chronic stage thermal baths occasionally give remarkable results. At first the indifferent baths may be used, such as those of Wildbad, Gastein, Badenweiler, Teplitz, Warmbrunn, Schlangenbad, Johannisbad, Ragatz, etc.; subsequently the thermal salt springs (Baden-Baden, Wiesbaden) and the carbonate springs (Nauheim, Oeynhausen, Soden). Simple salt baths as well as peat baths may be recommended, and the local application of peat compresses (fango).

If neuritic paralysis is incurable, improvement in function may be brought about by operative treatment; especially by the transplantation of tendons which was first advised by Nicoladoni, Drobnik, Franke, and others for the deformities of infantile spinal paralysis. This has been ingeniously adapted to cases of neuritis, and in incurable neuritic radial paralysis good results have been attained. In the last few years another form of substitution has been attempted, and the peripheral end of a diseased or injured nerve has been inserted within the fresh edges of a healthy nerve. This "graffe nerveuse" has several times been performed between the facial and spinal accessory nerves, also between the facial and hypoglossal nerves. In several cases the operation has undoubtedly promoted regeneration, but the functional result is questionable on account of the constrained movements produced; further experience is awaited. Léthievant prefers to unite the nerve loops which are formed by longitudinal splitting of the end of separated nerves.

A few words may be added in regard to special procedures occasionally

[1] Remak, "Ueber die antiparalytische Wirkung der Elektratherapie bei Drucklähmungen des N. radialis." *Deutsche Zeitschr. f. Nervenheilkunde*, Bd. IV, p. 377.

[2] Further consideration of these therapeutic questions will be found in Edinger's article.

necessary in traumatic neuritis. Here I follow Oppenheim.[1] If there is an open incised wound of the nerve, it should at once be sutured, and this, as a rule, is successful if carefully performed. If we see the patient only in the later stages, the question whether there is a complete interruption of conduction (which excludes the possibility of a spontaneous reunion) is difficult to answer. If there is complete paralysis and total DeR, we should not at once operate. It is better to wait and see whether there is any chance of spontaneous recovery, which cannot take place earlier than from 9 to 12 months after the appearance of paralysis. Then we are justified in attempting a secondary nerve union, which has been successfully done even after the lapse of years. Improvement after such an operation may be very slow, and hope should not be relinquished too soon. When the nerve is compressed by foreign bodies, these should be removed. Fragments of bone must be extirpated, or removed from a cicatrix (neurolysis).

Pathology.—I have not as yet touched upon the *anatomical* foundation of localized neuritis. It was long ago shown by Sigmund Mayer that certain degenerative and regenerative processes may take place in *normal* peripheral nerves, and that here and there under normal circumstances local changes of structure may appear in the constituents of peripheral nerves. These "degenerations" affect only individual fibers or segments of fibers. The axis cylinders and medullary sheaths may be disturbed, the nuclei of Schwann's sheaths may be increased and enlarged, and apparently play the rôle of phagocytes. We must bear this in mind when considering the anatomical relations of peripheral nerves. Regenerative processes may also appear. We find thin, delicate fibers with a narrow medullary sheath, also very fine axis cylinders which must be regarded as newly formed nerve fibers.

Regarding the changes which take place in *traumatic neuritis*, we find it difficult to differentiate *nerve inflammation* due to *traumatism* from *traumatic nerve degeneration.* This point we previously discussed, and were forced to conclude that for clinical purposes a clear formulation and sharp differentiation of their modes of development was impossible. This is also true of the anatomical condition. After the aseptic section of a nerve a consequent degeneration takes place in the peripheral (centrifugal) portion of the nerve (*Waller's degeneration*). Recent experience shows that the first histologic sign of this degeneration appears in the neurofibrillæ, the conducting elements of the axis cylinders (Homén, Mönckeberg and Bethe)[2] The fibrillæ are no longer so smooth as they formerly were, they are less tense, and here and there they show granular thickening. The granulation gradually increases and contraction and granular destruction of the fibrillæ take place. The perifibrillar substance is also slowly destroyed by swelling from the taking up of water. The changes in the axis cylinder are soon followed by those of the medullary sheath. The first signs appear from 2 to 5 days after separation. This structure is probably converted by a chemical change into a substance analogous to fat, small or large globules partially surround the

[1] Oppenheim, "Lehrbuch," p. 428.

[2] Mönckeberg and Bethe. "Die Degeneration der markhaltigen Nervenfasern," etc. *Archiv f. mikrosk. Anatomie*, 1899, p. 135; also the latest investigations of Schultz, including references. *Centralbl. f. pathol. Anatomie*, 1904; of Kattwinkel and Kerschensteiner in *Lubarsch-Ostertag's Ergebnissen*, 1904, IX, p. 8.

axis cylinders (medullary ellipsoids). Figure 1 in Plate I distinctly shows these changes in a Marchi preparation. The medullary sheath undergoing destruction is indicated by black globules, clumps, and rings, while the inter medullary sheath is yellowish. The changes appear to begin at the point of trauma, and progress rapidly toward the periphery. The removal of decayed material is partly effected by their passage into the lymph tracts, partly by phagocytosis. The rôle of the phagocytes is assumed by various cells, some lymphocytes and leukocytes, some proliferated cells of Schwann's sheath, some from the endoneurium, perineurium, and the surrounding connective tissue.

The changes demonstrated in Schwann's sheaths are important for another reason. They are closely related to reparative processes in the peripheral nerves. These begin long before degeneration has run its course. But before discussing this feature, we must consider the processes in the *central* stump.

Great changes also take place in the first of the interannular segments of Ranvier: the medullary sheaths and axis cylinders are rapidly destroyed; isolated fibers degenerate far upward centrally; as a rule, the process is confined to the first six segments. In the nerve cells and in its fibers other changes appear, a knowledge of which is necessary for the full understanding of many pathologico-anatomical details. In the main these invade less deeply; the processes are less distributed, they appear later, and progress more slowly than those which cause Waller's degeneration. In the cells, particularly with Nissl's staining, we find transitory but obvious phenomena: dissolution of Nissl's bodies (chromatolysis) with a swelling of the entire cell and with nuclear deposit. It is certain that at least many of these cells are not destroyed and that they recuperate. Subsequently the cells themselves, as well as the fibers and all of their constituents, become thinner, smaller, and more delicate. Whether this *retrogressive degeneration* is histologically any different from Waller's degeneration is not yet known.

Reparative changes are early noticeable. Only a portion of the proliferated cells of Schwann's sheath assume the rôle of phagocytes. The others form spindle-celled bands, some of which are arranged within, some without, the old sheaths. As to the further process, there are two opinions which are diametrically opposed to each other, and a decision is at this time impossible. One view is that these bands are composed of newly formed nerve fibers which grow out of the central stump; the other (this is based upon Bethe's recent experiments) assumes that new nerve fibers are formed from the protoplasm of these spindle-celled bands (*autogenous regeneration*). Investigators admit the energetic growth of fibers from the central stump, and assert, what is clinically of great significance, that the reunion of the stump renders complete regeneration far more certain. According to Bethe, we can determine by experiments in young animals that autogenous regeneration is perfect, while in full grown animals, although always quite apparent, it is never complete. The great recuperative power of the peripheral nervous system in contrast with that of the central, is of the utmost importance in pathology, no matter how it may be explained.

If union of the severed ends of a nerve does not take place, a *nerve cicatrix* develops at the point where the trauma occurred. The connective tissue of the peripheral nerve, the endoneural, the perineural, and the epineural, proliferates further, becomes fibrous and poor in nuclei, and in dense, irregular

bands encircles the nerve fibers which force themselves between the individual nerve bundles. In the peripheral stump these gradually atrophy, but probably not until processes of repair begin.[1] At all events conduction from the center to the periphery is permanently and completely abolished.

Paralysis of such a nerve cicatrix is illustrated by Figure 2, Plate I. This is from a preparation of a section of the brachial plexus in a young man, who was cut by a knife in the supraclavicular fossa. To promote secondary union of the nerve this piece completely embedded in connective tissue was extirpated. We see that the entire, external, and otherwise very regular formation of the nerve is destroyed. In many areas the separate layers of connective tissue covering cannot be distinguished. The connective tissue is poor in nuclei, close, and fibrous. In many areas it presses upon the glistening, red-stained portions (*f*) in the van Gieson preparation, and also between the finer nerve bundles. Most of the retained axis cylinders are extraordinarily thin and fine. The changes are partly atrophic, partly the formation of new fibers. The medullary sheaths are very narrow, in stained preparations they are not sufficiently blackened, they do not form complete rings. This incompleteness and irregularity of the medullary sheaths is most distinctly evident in longitudinal sections. In many areas the nuclei in Schwann's sheaths appear to be increased. This increase is perhaps only apparent, and is explained by the compact arrangement of the individual constituents. The other connective tissue nuclei are diminished.

These changes may all be regarded as simple *degenerative processes* and their immediate consequences; there is no *inflammation*. Yet the alterations which we recognize as the invariable sequelæ of nerve injury, as Waller's degeneration, are considered to form the foundation of *parenchymatous neuritis*; in fact, whenever the diagnosis of neuritis *intra vitam* has been based on the previously described landmarks, these very lesions were demonstrated in the peripheral nerves.

The number of necropsies performed in cases of simple, localized mononeuritis is naturally scant; and such cases will only accidentally be found on the post mortem table. In a case of "rheumatic" facial paralysis, Minkowski demonstrated a purely degenerative process in the peripheral nerve branches, while the nerve trunk from the geniculate ganglion to the center was normal; the neurilemma was not implicated. In another case Déjérine and Theohari found degeneration in all of the branches, while the root was intact. After traumatic lesions of a peripheral motor nerve (see above) the cells of the nuclei show with Nissl's staining the same characteristic changes as in the experiment.

In a case recently reported by A. Westphal[2] of plexus neuritis in an apoplectic form without actual mononeuritis, there were severe, parenchymatous, neuritic changes which increased from the center to the periphery; slight lesions were also demonstrated in the entrance zone of the posterior roots. The broad pale fibers, marked by divisions which Gudden has described, were here noteworthy. They resembled spindle-celled bands, and like these are probably to be attributed to regenerative processes.

[1] See the article by Egger and Armand Delille (*Arch. de Neurol.*, 1903, p. 474).

[2] A. Westphal, "Ueber apoplectiforme Neuritis." *Arch. f. Psychiatrie*, Bd. XL, p. 64.

The accompanying figures 3 and 4, Plate II, show, in an osmic acid preparation after van Gieson, the prominent changes on a weak and powerful enlargement of a longitudinal section made in a case of "parenchymatous neuritis"; the medullary sheath is stained black, and its irregular destruction is especially distinct; in some areas it is still intact, in others, partly belonging to the same fiber, there is complete destruction. This reminds us of specimens furnished by *segmentary neuritis*, which was first described by Gombault, and designated *névrite ségmentaire periaxille*; he found it in guinea pigs experimentally poisoned with lead salts. Stransky[1] has recently reviewed these investigations, which had elicited but little appreciation. The most characteristic feature of the process is its *lack of continuity*, its limitation to more or less lengthy areas within the confines of an otherwise normal nerve fiber. The origin of the process is a fine, granular, drop-like decay of the myelin, while in Waller's degeneration the medullary sheath decomposes into much less massive clumps; from the onset there is said to be a conspicuous proliferation of the cells of Schwann's sheath (nuclei as well as protoplasm), so that, unlike Waller's degeneration, the entire process reveals a conspicuous parenchymatous inflammation. Some of the proliferated cells temporarily change into phagocytes. Gradually the *axis cylinder* is implicated, it becomes broader and paler, until finally it can no longer be demonstrated with the ordinary staining methods; Stransky doubts whether it is actually destroyed, because the peripheral portions of the nerve fibers do not show Waller's degeneration which might otherwise be expected. The process admits of perfect restitution, the histologic signs of which are easily recognized. Stransky believes this form of disease to be much more common than would appear from literature. The chief value of these investigations lies in the fact that, if Stransky's view is correct, it enables us to differentiate *inflammatory parenchymatous disease* of the peripheral nerve from *secondary Wallerian degeneration*, and the whole subject gains in clearness. But whether such a sharp distinction as Stransky proposes is practicable can be learned only by further investigations. It appears plausible in the mild forms of certain paralyses—for example, pressure paralysis during sleep—to assume that a periaxial neuritis in its initial stage is the anatomical foundation, since from the rapid restoration of the faculty of conduction in these cases we must conclude that the nerve element of conduction has not been greatly damaged.

Déjérine-Klumpke and Falkenheim reported a few instances of changes produced by *subcutaneous injections of ether* after the question had been experimentally investigated by Arnozan and Pitres and Vaillard. If a nerve is exposed to the action of sulphuric ether for 24 hours, changes may be demonstrated in its area, these corresponding to necrosis of a portion of the nerve; this is followed after an interval by parenchymatous degeneration of the peripheral portion. In pathologic cases nerve destruction to a varying extent has been demonstrated in the radial nerve. As to the propagation of the inflammation from the diseased vicinity of the nerve in the different forms of neuritis, we naturally often find true inflammatory changes. This is the case when suppuration from surrounding areas is conveyed to the nerve—for

[1] Stransky, Ueber discontinuirliche Zerfallsprocesse an der peripheren Nervenfaser." *Journ. f. Psychologie u. Neurologie*, 1903, H. 5 u. 6.

example, caries of the petrous portion of the temporal bone to the facial nerve (E. Flatau), suppuration of the cervical glands to the pneumogastric nerve (Lugaro). In these cases the inflammatory process in the interstitial tissue of the nerve is to some extent propagated, dilatation is produced with immoderate fulness of the vessels, hemorrhages, small-celled infiltration, and a serous exudate; besides these signs of true inflammatory interstitial changes, there are lesions of the parenchyma, swelling of the medullary sheaths and axis cylinders, and degeneration and destruction in the peripheral portion. Changes of this kind are macroscopically recognizable as swelling of the peripheral nerves; subsequently, in place of inflammatory infiltration we find a round-celled exudate and hemorrhage from the connective tissue, or new formations may produce the picture of neuritis and perineuritis nodosa, the most characteristic type of which is the neuritis or polyneuritis of leprosy, which we shall later describe. As an invariable and secondary result of neuritis which has interrupted conduction, we learn to recognize degeneration in the most peripheral portion of the peripheral nerve. With the same regularity as in animal experiment, we find changes in the *muscles* supplied by the diseased nerve. The first sign is a fatty metamorphosis in the muscular fibrillæ which Obersteiner and I demonstrated with the Marchi method in a case of hemiatrophy of the tongue after neuritis of the hypoglossal nerve. In a further progression of the process and its prolongation, this change in the muscle advances and leads to severe atrophy, a decrease in the size of the fibrillæ, the disappearance of the transverse striæ, and such a massive deposit of fat in the muscles that the change is recognizable even macroscopically. Westphal found the alterations less marked in his case. The caliber varied, and large voluminous fibers were found alongside small atrophic ones and some of medium size. Some of the fibers were rounded off; there were central nuclei and rows of nuclei, as well as spaces which had formed between them. Transverse striation was still evident. There was no fatty degeneration. Little is known of the changes in the motor end-plates in neuritis. The changes which take place in the *central* portion of the diseased sensory or motor nerve, and in the skin and its *dependent structures* from alteration of its sensory innervation, are more variable and more difficult to explain than the changes in the muscles.

The former are found as so-called *retrogressive degeneration*, such as appears in the central stump of the severed nerve as above described; in the nerve fibers a slow and tardy atrophy in all of its constituents, in the cells there is at first an alteration in the stainable substance—Nissl's bodies—which, after producing slight changes, passes away. In the subsequent course, atrophy of the cellular elements causes a reduction in the size of the individual elements or even their complete wasting, most of the finer structures remaining unchanged. These experiences (which in man are based particularly upon the findings in the spinal cord of persons who had lost a member by amputation some time before death) teach us that when in a case of peripheral neuritis severe lesions, like acute degeneration of the fiber or the cell, are found in the central portion of the affected nerve or its spinal origin, we are not dealing with phenomena immediately due to peripheral disease, but substantive ones; this is true both of localized and multiple neuritis, as well as of the spinal changes which often accompany the latter. In some cases for which another

explanation seemed obvious, as in a case of carcinomatosis of the left brachial plexus and degeneration in the left posterior column, published by Jacobsohn, this cannot be considered to apply.

A word must still be said of the dependence of the changes in the *skin and its structures* on disease of the peripheral nerves. Under the symptomatology I called attention to the most important of these trophic, vasomotor, and secretory disturbances. They are *gangrene, bed-sore, perforating ulcer,* some *eruptions of vesicles,* and *glossy skin* which represent the combination of many different symptoms, etc. The comparative rarity of these symptoms precludes the theory that they directly depend upon a peripheral nervous process—as, for instance, muscular atrophy. To some extent they are no doubt due to a coördinative and substantive disease of the skin, others are the consequences of analgesia of the skin from neuritis, which makes it more susceptible to injury; but all clinical experience seems to point to the fact—in spite of the great labor expended upon experiments, proof by this means is much less positive—that there is a direct trophic dependence of the skin and its structures upon the nervous system, although the nature of this dependence is by no means always clearly appreciated.[1]

POLYNEURITIS

The method of division which I have so far followed is, as stated, not a strictly systematic one. I have considered only the inflammation of individual nerves, *mononeuritis,* and I shall now describe *polyneuritis.* In tabulating the different forms of neuritis with which we have become acquainted, we find the following:

(1) *Traumatic mononeuritis* of any origin; (2) *Occupation neuritis*; (3) *Wound neuritis*; (4) *Injection neuritis*; (5) *Propagated neuritis*; (6) *Ascending neuritis*; (7) *Post-infectious mononeuritis*; (8) *Rheumatic and idiopathic mononeuritis.*

Groups 1 to 6 include all cases of *neuritis from local cause,* and groups 7 and 8 those due to *internal* cause. It would be perfectly justifiable to consider the two last mentioned groups with polyneuritis, since these are invariably produced by internal conditions. But if we adopt this method of grouping we shall meet with difficulties; for, as has been stated, in isolated cases *local* and *internal* causes may act jointly. On the other hand, the line of demarcation between polyneuritis and mononeuritis is not sharply defined; between these two groups are affections of several isolated nerves by a local cause, and *plexus neuritis* and *mononeuritis multiplex* must be included. *Polyneuritis,* in a restricted sense, occupies so large a field that some subdivision appears necessary. I shall follow Remak and classify it as follows:

(1) Polyneuritis from *exogenous intoxication*; primarily *alcoholic neuritis.* This is the most common form of polyneuritis, and serves as the type of the affection; other poisons to be considered are *lead, arsenic, carbon bisulphid, carbonic oxid,* and *mercury.* While it is unlikely that a single acute intoxication with alcohol ever produces polyneuritis, this is quite possible in the case of arsenic, hence we must differentiate between polyneuritis due to acute and to chronic intoxication.

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[1] See also Cassirer, "Vasomotor Trophic Neuroses," p. 88, et seq.

(2) The second group consists of polyneuritis due to *chronic*, and, more rarely, to *acute autointoxication*, such as occurs in *diabetes*, *gout*, *tuberculosis*, *syphilis*, *pregnancy* and the *puerperium*, *senility*, general *cachexia*, poisoning by *meat* and *sausage*, and in other forms of *intestinal* autointoxication.

(3) *Polyneuritis* during and after acute *infectious diseases*. If we class polyneuritis following *diphtheria* with that occurring in other infectious diseases, it is not strictly correct; for diphtheritic polyneuritis differs in type from the other infectious forms of polyneuritis.

(4) *Idiopathic polyneuritis*. This peculiar form of the affection occurs as an infectious disease, and includes certain forms of Landry's paralysis. .

(5) *Beri-beri*, which, as a rule, must also be considered as a disease *sui generis*.

(6) The *polyneuritis of leprosy*, which, because of its typical clinical and anatomical features, cannot be included in the second group.

Polyneuritis is frequently produced by the *combination* of several deleterious factors; for example, the combination of alcohol and lead, or alcohol and tuberculosis. Oppenheim calls attention to the fact that those suffering from alcoholic polyneuritis are much more susceptible than others to infectious diseases.

Symptoms.—The *symptomatology* of polyneuritis does not correspond to that of mononeuritis; in many respects it is peculiar, and demands a special description. The individual forms show many variations, but, on account of its preponderating frequency, alcoholic polyneuritis is the most important.

This occurs chiefly in *spirit* drinkers, but occasionally in those who drink beer and wine; for example, it is said to be exceedingly rare in Munich, where enormous quantities of beer are consumed. I will quote a case which will best illustrate the essential features of the disease, and also describe the symptoms.

Case 22.—The patient is a beer brewer, aged 39, who formerly drank upward of 4 liters of beer a day, and now consumes a large quantity of spirits. The patient presents a number of the signs of *chronic alcoholism*. Early in the morning there is retching and vomiting of mucus. It is difficult for him to fall asleep, and before doing so he is subject to the familiar occupation hallucinations, believing that he is still at work; he has frightful visions, sees black men, and the like. For a year past he has felt some exhaustion on walking, formication and numbness in the legs being added, and in the last few weeks all of these symptoms have become aggravated; periodically there are severe drawing pains. There is no difficulty in urination, no double vision, no girdle sensation. On examining the patient, his gait is striking. He lifts his legs immoderately high from the floor, and the feet wabble to and fro in a flaccid way. When he stands with his eyes closed and his feet close together, there is marked swaying (Romberg's symptom). Inspection of the lower extremities reveals marked atrophy of all the muscles of the leg, especially the extensors; to palpation they feel coarse, like fibrous tissue. These muscles, especially of the calf, are very sensitive to pressure, as are also the nerve trunks—particularly the peroneal and tibial nerves in the popliteal space and behind the internal malleolus. There is also moderate hypotonia. Upon tapping the patella tendon there is a feeble contraction of the quadriceps, followed by a tremor which simulates an increase of the patella reflex. The Achilles tendon phenomenon is abolished bilaterally, movability and power are good in the hip- and knee-joints, and the leg can be extended bilaterally with almost normal force. The power of extension is absent in the right foot (tibialis anticus), in the toes it is minimal; the power of abduction is diminished in the foot; the flexors of the foot and toes show loss of function. On the left side the dorsal flexors and the peroneal muscles are decidedly affected, while the flexors of the foot and toes function normally. On

the plantar surface of the toes and the anterior portion of the foot, brush contact is not felt bilaterally; in the same areas there is hypalgesia, while the sensation of position is not markedly disturbed. On the other hand, the "sensation of vibration" in the tibiæ is lost. On electric examination the right peroneal nerve and the muscles supplied by it fail to react to the faradic current, the posterior tibial and the muscle it innervates respond feebly to powerful currents; in the right peroneus longus very strong galvanic currents are necessary to produce even a minimal contraction. Stimulation of the posterior tibial nerve causes a feeble contraction in the triceps suræ. On direct irritation of the muscles of the lower leg (extensors as well as flexors) we obtain with strong currents only a sluggish AnCC. Consequently partial DeR with a marked decrease of contractility has here been reported.

In the extended hands there is an active vibrating tremor. The grasp of the hand and all other movements of the upper extremities are normal; the median and ulnar nerves are bilaterally painful to pressure, which produces radiating pains. The tendon reflexes are preserved and the tongue is tremulous, but the cranial nerves, the eye-grounds, and pupils show no pathologic change.

The patient presents the familiar *general symptoms of chronic alcoholism*, which are of diagnostic value, although the alcoholic often denies the excessive use of spirits: among these symptoms are the well-known *vomitus matutinus*, the retching and vomiting of mucus, insomnia or restless sleep, disturbed by frightful *hallucinations, dreams, and a tendency to spasmodic contraction of the muscles* (particularly of the calf which is perhaps the first indication of a neuritic or myositic change) and *numbness* of the legs, especially when these members rest upon a hard surface. Furthermore we note the *alcoholic tremor*, usually most marked in the tongue and hands, the general signs of nervous irritability which is often of characteristic type, uncontrollable *anger* which may culminate in a tendency to destruction, as well as *fear*, the maudlin state which so readily leads to tears, and *jealousy*. The toper may often be recognized at a glance from his external appearance.

The early symptoms of neuritis are not often so sharply defined as those of chronic alcoholism. Paresthesia in the leg gradually becomes more marked; more severe pain develops as well as weakness in the legs which becomes noticeable upon long standing, and the fundamental symptoms of neuritis then appear. In contrast to the cases with more or less *chronic* development, the initial stage of which may extend through several months, are those with acute onset. The disease may develop immediately after an attack of *delirium tremens*. By no means rarely it is due to a predisposing cause; therefore anything which lowers the alcoholic's power of resistance, such as an infection, influenza, pneumonia, trauma, etc., may be sufficient.

As to the frequency with which alcoholics are affected by neuritis, the statistics of the Hamburg hospitals show that it occurs in about 3 per cent. of all cases of alcoholism. Apparently *women* are relatively more frequently attacked than men (Gowers states three times as often) which is attributable to the more delicate constitution of women. According to Ross, the majority of cases occur between 30 and 50 years of age (64 out of 88); 13 of his patients were between 20 and 30, 10 between 50 and 60. Before and after these limits polyneuritis is very rare, but it has been reported in children aged 7 and 5 years. I saw a case of alcoholic polyneuritis in a child, aged 3, whose father drove a beer wagon. Every evening the child was given a glass of beer, and sometimes spirits, to drink. Three months previously, it was attacked by *severe pain* in the feet accompanied by fever. *Gradually* weakness of the legs appeared, and, seemingly, also of the hands, and sometimes it was difficult for the child to sit up. The pains and weakness in the arms and trunk disappeared, and upon examination sensation so far as we could judge was

normal, but the muscles of the calves still appeared to be painful on pressure. The knee- and heel-jerks had disappeared. There was decided paresis of the extensors of the foot and toes, as well as the abductors of the foot. The paresis was more marked on the right than on the left side, and the power of flexion in the toes was defective. Electrically the paretic muscles showed partial DeR. From the development and nature of the affection, there could be no doubt of the correctness of the diagnosis, polyneuritis; that alcohol is especially injurious to the nervous system of a child is proven by many experiences. It appears that even the *inhalation of alcohol* may produce polyneuritis. Remak quotes from Lancereaux the case of a woman, aged 35, who continually inhaled the vapor of alcohol which she had in her bed-room, and this caused polyneuritis. Several of my patients have attributed their neuritis to the fact that they were compelled to work in rooms filled with the vapor of alcohol. Under such circumstances it is extremely difficult to exclude the immoderate consumption of alcohol, yet there is no reason why the inhalation of alcohol should not likewise be injurious.

Even when there are no complications alcoholic neuritis may set in with *fever*, but fever may also be absent—especially in those cases which develop more slowly. It is *not necessarily present in idiopathic polyneuritis*, not even in severe cases. Among the general symptoms those referable to the *gastro-intestinal* tract must be especially mentioned, and it will often be doubtful in how far we may ascribe these to neuritis in a restricted sense and in how far to chronic alcoholism. Enlargement of the spleen is probably only observed in idiopathic polyneuritis, and the same may be said of the pathologic urinary findings (*urobilinuria* and *albuminuria*) as of the gastrointestinal disturbances.

As in our case the *pains* preceded the paralysis, and as is usually the case with polyneuritic pains they are often *most intense, drawing* and *tearing* in character and are situated deeply; they may readily be differentiated from the lancinating pains of tabetics by the description. As a rule they do not occur in typical attacks as do the lightning pains of the tabetic. Frequently they are combined with *cutaneous hyperesthesia,* and *hyperæsthesia plantaris* is often an exceedingly annoying symptom which may persist for a long time, and markedly affect the gait of the patient. Although the pains are often the earliest symptom of alcoholic neuritis, they frequently disappear or decidedly lessen in intensity after the appearance of the paralysis, certainly in those cases in which the paralysis is amyotrophic.

Besides the pain there are usually various *paresthesias* as well as *thermo-paresthesia,* and pain upon *pressure of the nerve.* This may be present when there are no other symptoms of neuritis. It is most prone to appear in the peroneal and posterior tibial nerves, also in the crural and other nerves of the lower extremities; but, as in one of my cases, it may likewise be observed in the upper extremities in which as a rule there are no pathologic phenomena. All manipulations which cause torsion of the diseased nerves evoke spontaneous pain or pain which radiates in these nerves (sciatic phenomenon and similar symptoms). The *muscles* are often sensitive to pressure, sometimes even more so than the nerves, yet we are unable to diagnosticate *neuromyositis* from this early sensitiveness of the muscles to pressure (Remak). Distinctly palpable *swelling* of the nerve is very rarely observed in alcoholic neuritis or

in most other forms of polyneuritis; leprous polyneuritis only being an exception. The *paralysis* gives to the pathologic picture its peculiar stamp, and its essential features are as follows: The *paralysis* is almost *symmetric,* it usually implicates the *distal ends of the extremities,* and, in alcoholic neuritis as well as most other forms of polyneuritis, chiefly the *lower extremities.* Here the peroneal muscles are preferably attacked, being more markedly implicated than those supplied by the tibial nerve. When the upper extremity is attacked —usually the radial region—this generally occurs later, and the *paralysis* is *of ascending type,* also *degenerative and atrophic,* and in its course shows the various stages of the *reaction of degeneration.*

Corresponding to the chief localization, the first symptoms appear as weakness in walking. The patient soon tires. The symptoms as described resemble those of so-called *intermittent claudication.* For example, a patient, aged 23, who a few weeks ago suffered from difficulty in walking (he could walk only ten minutes at a time), stated that he soon felt intolerable formication, a feeling of tension and sensation of cold in the skin, and weakness in the legs, like paralysis, which compelled him to sit down. After ten minutes he was able to resume walking, but the same symptoms constantly recurred. Examination revealed the early signs of an alcoholic polyneuritis affecting the muscles of the leg, especially the peroneal, but no alteration in the vascular apparatus. In these cases walking gradually becomes more difficult. If the patient is examined in a somewhat advanced stage, which is apt to be the case, the gait usually resembles the *steppage gait.* The legs are abnormally lifted at the hip- and knee-joints because, otherwise, the tip of the patient's flaccid foot would not leave the floor. Close examination generally reveals paresis, most marked in the extensors of the foot and toes and in the abductors, while the flexors, although paretic, usually show less disturbance of function. The extensors of the leg and the muscles which raise the thigh toward the pelvis are usually less implicated. The muscles supplied by the peroneal nerve are not affected to the same extent; in fact, in all forms of polyneuritis paralysis throughout the extent of a nerve is remarkably frequent, but only in exceptional cases are all the muscles of an extremity completely paralyzed. The paralysis is usually *symmetrical,* but to this there are numerous exceptions. One leg *in toto* or individual muscle groups may be much more markedly affected than others, and there are cases in which the symptoms of paralysis are almost wholly confined to one side.

In the further progress of the paresis walking becomes impossible; the patient is confined to bed. The muscles of the upper extremity and of the trunk are also weak. There are but few exceptions to the rule that the paralysis ascends, yet cases have been reported in which the affection appeared in the arms. The *paralysis of the arms is usually less profound and less extensive than that of the legs.* As a rule, the radial region is more markedly implicated, and examination shows that the muscles supplied by the nerve are not uniformly attacked. The supinator longus and the abductor pollicis longus may be exempt from the paralysis, so that a type resembling lead paralysis is produced (Oppenheim). The small muscles of the hand and the long flexors of the hands and fingers are occasionally implicated, while the muscles of the arm and shoulder are usually exempt or only temporarily affected. Finally, there are cases with absolute paraplegia cervicalis. I once saw the rare picture

of polyneuritis due to alcohol affecting the arms to a much greater extent than the legs. Here the special localization was apparently due to the occupation of the man—that of coachman.

In severe cases the *muscles of the trunk* may be implicated, so that rising from a recumbent posture, turning, and sitting become difficult or impossible. Oppenheim [1] has made explicit reports concerning the implication of the *abdominal muscles* in neuritis and polyneuritis, of which little had previously been known. This condition may be a partial phenomenon of general polyneuritis, and was so regarded in a case of probable post-typhoid polyneuritis in a young man, aged 18. This was a patient from the private practice of Professor Oppenheim, and he was under my observation. Paralysis of the abdominal muscles was bilateral, it especially distressed the patient on coughing, sneezing, and sitting up, and persisted longer than the other symptoms of polyneuritis. Ross also reports having seen loss of power in the abdominal muscles in advanced cases of polyneuritis. In other cases reported by Oppenheim paresis of the abdominal muscles was due to herpes zoster, malaria, and enteric fever; once, apparently after the chronic abuse of alcohol, he observed *isolated neuritis* of some of the intercostal nerves.

The occasional implication of the phrenic nerve in the neuritic process is of special prognostic importance. Strümpell first noted this in alcoholic polyneuritis, in which it was revealed by the cessation of *diaphragmatic respiration*; with every inspiration the epigastrium was deeply retracted. Paralysis of the diaphragm is usually an ominous symptom, but Oppenheim once saw the bilateral phrenic paralysis of alcoholic polyneuritis disappear in a month and complete recovery take place.

Among the *cerebral* motor nerves, the vagi are most often attacked; occasionally the *laryngeal* branches are the seat of such changes (implication of the recurrent laryngeal in cases reported by Minkowski, Kast, Roth, Rennert, and Sorgo). On the other hand an *acceleration of the pulse* to 120–150 beats per minute is relatively common in all forms of polyneuritis; and the relation of this disease to an affection of the pneumogastric nerve is confirmed by necropsy. I shall later describe the symptoms of diphtheritic polyneuritis which are referable to the vagus, but naturally other causes of tachycardia must also be considered. Serious as the symptom is, it has been known to disappear. Much more rare is a *decided bradycardia.*

Paralysis of the ocular muscles of neuritic genesis is much more common in alcoholic polyneuritis than in other forms. Among the latter we frequently note unilateral paralysis of the abducens, more rarely unilateral paralysis of the oculomotor nerve. That even complete paralysis of all the external ocular muscles, combined with disturbances on the part of other bulbar nerves (difficulty in deglutition), may be due to polyneuritis is taught by a case of Damron-Meyer, in which anatomical examination revealed complete degeneration of all the nerves of the ocular muscles, also of the hypoglossal, glosso-pharyngeal, facial, and some of the spinal nerves, while the brain trunk was in normal condition.

In *alcoholic polyneuritis,* paralyses of the ocular muscles are not uncom-

[1] Oppenheim, "Abdominaler Symptomencomplex bei Erkrankungen des unteren Dorsalmarks." *Deutsche Zeitschr. f. Nervenheilkunde,* XXIV, p. 330.

mon. Here we may see unilateral, complete, or partial oculomotor and abducens paralyses, but their *neuritic* character is uncertain. Some of them are due to inflammatory changes in the region of the nuclei of the ocular muscles; in alcoholics we find an inflammation which was first described by Wernicke as *polioencephalitis superior,* the chief local symptoms of which are paralysis of the ocular muscles; combined with *polyneuritis* this has been demonstrated anatomically by Jacobaeus and others. We will be particularly suspicious of a polioencephalitic basis when instead of complete or partial paralysis of one nerve, we find the so-called *glance* (Blicklähmung) paralysis, a paralysis of the muscles which draws the bulbus to one side (for instance, it may affect the right external and the left internal muscles which are innervated by different nerves, but which have a common center of innervation in the trunk of the brain). We have no positive guide for the differentiation of peripheral neuritic and central polioencephalitic paralysis of the ocular muscles. *Nystagmus* has several times been observed in polyneuritis, usually of alcoholic origin. The condition of the *internal ocular muscles* is most important in the diagnosis of polyneuritis, especially alcoholic neuritis. In polyneuritis we must first determine whether there is true *reflex pupillary rigidity* —i. e., arrested reaction to light with normal reaction to convergence—and whether it is permanent.

Among 1,000 cases of chronic alcoholism Uhthoff found reflex pupillary rigidity with retained reaction on convergence in only 10, and in these it was doubtful whether the pupillary rigidity was persistent and unchanged; therefore it must be regarded as rare. Pupillary rigidity of this form has also been observed in alcoholic polyneuritis (cases being reported by Gudden, Dreschfeld, Rennert, Nonne, Thomsen, Oppenheim, and others). But in two of Kramer's[1] cases of polyneuritis in which this symptom appeared, the reaction was normal after recovery took place. *Reflex pupillary rigidity is evidently an unusual symptom in alcoholic polyneuritis*; moreover, it disappears much more often than in tabes dorsalis which is the affection with which it must be contrasted in the differential diagnosis. *Differences in the size of the pupil,* as well as *ophthalmoplegia interna* which appear to occur in chronic alcoholism as well as in alcoholic polyneuritis, are much more common. In other forms of polyneuritis changes in the size of the pupils are seldom observed.

The other motor cranial nerves are rarely implicated in polyneuritis. Only *facial paralysis* is common, and this may be either unilateral or bilateral. Facial paralysis may even be the first symptom of the affection, but, as a rule, it appears at the acme of the disease, and in cases which, from their development, must be considered severe. According to statistics at hand, the percentage of cases of facial paralysis occurring in alcoholic polyneuritis is comparatively small. Quite exceptionally we note difficulty in mastication and deglutition; a paralysis of the soft palate is most apt to occur in diphtheritic polyneuritis. Bilateral paresis most severely affecting all of the muscles of mastication was noted in a singular case reported by Gaspero (*Monatsschrift für Psychiatrie und Neurologie*, XIV, p. 161). In some cases an impaired movement of the tongue has been observed (with corresponding neuritic

[1] Kramer, "Rückenmarksveränderungen bei Polyneuritis," Breslau, Juli, 1902.

changes in the hypoglossal nerve). Pastrovich saw isolated neuritic hypoglossal paresis in an alcoholic.

The paralysis of polyneuritis is of *degenerative* nature. It runs its course with *atrophy* and *changes in electric contractility* which are manifest as DeR; the *tendon reflexes* are abolished in the paretic muscles. All of these symptoms correspond to the conditions described in mononeuritis. Atrophy invariably follows paralysis, and depends upon its degree. *Edema,* which is not rare, occasionally masks the atrophy. The *muscles* are *flaccid* and *soft,* and extremely painful to pressure. In the later stages a distinct *hardness,* a kind of *induration,* is occasionally noted as an expression of connective tissue proliferation in the atrophic muscles. In this stage appear the *contractures* to which I shall later refer. In the earlier stages passive movements are free in so far as that they are not prevented by pain.

The *electric contractility* is here of the greatest diagnostic importance. In daily practice, especially in the acute period of the disease, we should be content with ascertaining the most important facts, as the suffering patient is often unable to endure the painful and fatiguing processes of a minute electrical examination, especially children in whom even a superficial test can be only made with the utmost difficulty. As a rule we find *DeR,* and, in accordance with the fact that paralysis in polyneuritis is usually incomplete, *partial DeR is more common than the complete.* The most marked changes are found in the distal regions, in the small muscles of the foot, the extensors of the foot and toes, the small muscles of the hand, and those supplied by the radial nerve. Besides the characteristic sluggishness of contraction in the reaction of degeneration upon direct galvanic irritation, occasionally we note mere *absence of electric contractility,* which, however, may be due to the accompanying cutaneous edema. Rare reactions of degeneration (indirect sluggishness of contraction, faradic DeR) are also observed, but they are of little diagnostic interest; also in regions which are functionally less severely implicated, there is only a *simple quantitative diminution of contractility,* but we must be cautious in utilizing this finding in cases of bilateral paralysis. The demonstration of electric changes in contractility is often easier than that of atrophy, although the former symptom invariably follows the paralysis by one and a half or two weeks.

The fact is of great interest that changes of electric contractility may occasionally be demonstrated in muscular regions in which functional damage has never been apparent. Remak[1] reported a case of general neuritis in a servant girl, in whom by electricity severe changes were demonstrated in the facial nerves which had never been paralyzed. The author assumed the anatomical foundation of this to be the periaxial neuritis of Gombault. Similar cases were previously and subsequently reported by Kahler and Pick, Hösslin, and Bernhardt. In alcoholic polyneuritis I have occasionally demonstrated changes in the electric contractility of the non-paralyzed muscles supplied by the radial nerve, but this finding is probably most common in saturnine polyneuritis (see below). As in mononeuritis electric contractility returns long before the restoration of function.

The *tendon reflexes* are usually abolished. Sometimes we find them ar-

[1] Remak, *l. c.,* p. 355.

rested or pathologically diminished in areas in which apparently neither the power furnished by the muscles nor sensation are pathologically affected. Under such circumstances, particularly in diphtheritic polyneuritis or diabetic polyneuritis, the knee-jerk may be absent; in alcoholic polyneuritis, Westphal's symptoms may be present without paralysis, DeR, disturbance of sensation or ataxia in the course of the crural nerve. This symptom, therefore, may be the only one which reveals the further distribution and generalization of the neuritic process, and for this reason such cases must be considered as on the border-line between neuritis and polyneuritis. I saw a man, aged 40, an alcoholic and presenting the ordinary signs of chronic abuse of alcohol, who showed degenerative right-sided paralysis of the peroneal nerve with corresponding disturbances of sensation, DeR, and atrophy of the extensors, yet in whom the right patella tendon reflex was abolished while both Achilles reflexes were present. The power of the quadriceps was quite normal. When the upper extremities are implicated in the paralysis, the tendon reflexes are absent. That the tendon reflexes form a fine test for very slight neuritic changes and those in the reflex arc is shown by the foregoing reports and by the fact that after complete restoration of function they may long be absent, even for years, yet finally reappear.

The *intactness of the tendon reflexes* in areas affected by polyneuritis, as well as in mononeuritis, is extremely rare, but exceptionally it has certainly been observed. I have seen such a case. Occasionally these reflexes show a pathologic change. For instance, Westphal noted a crossed abductor reflex while the patella reflex was absent, and I saw a similar coincidence in a typical case of arsenical polyneuritis.

Objective sensory disturbances are usually less marked than paralytic phenomena, both in intensity and extent. There is a slight diminution of the faculty of perception, and it can be determined only by very close examination. It is usually combined with *hypesthesia* and *hypalgesia*, which conspicuously involve the *distal* portions of the extremities, being most severe in the toes, and gradually disappearing as it passes upward through the foot and leg; in the upper extremities the fingers, especially their tips, are most markedly implicated while the hypesthesia gradually disappears centrally. Such slight disturbances of sensation in the fingers are not uncommon. They may be the only symptom of the disease in the upper extremity. Occasionally we find merely paresthesias, without objective disturbances to prove the extension of the polyneuritic process. More rare than a disturbance in motion is a disturbance of sensation which appears to affect only *one* nerve, but affects this *in toto*. Such cases are certainly exceptional.

As to the nature of the disturbance, *all forms of sensation* may be uniformly decreased. Characteristic combinations are noted, especially that of esthesia on contact with hyperalgesia. Sometimes it is impossible for the patients to find words with which to express their peculiar sensations, which are outside the realm of physiology. They speak of a "dull pain," but this is much more unpleasant than that produced by the prick of a pin (*anesthesia dolorosa*); hypalgesia combined with hypersensitiveness to contact is also noted, and hyperesthesia plays an important rôle among the sensory disturbances of the polyneuritic. In the various nerve disturbances the changes in sensation may also vary greatly so that examination reveals a manifold and ever-chang-

ing picture. The *sensation of position* as well as that of *deep sensation* may be markedly disturbed, and this usually corresponds to the change in the *sensation of vibration.* Synchronous with these is a derangement of the power of motion to which I shall refer later—ataxia.

It must still be stated that a *retardation of the conduction of sensation* is found among polyneuritic sensory disturbances. This was formerly thought to belong only to spinal processes, particularly tabes, but my experience leads me to believe—what is anatomically confirmed—that in polyneuritis there is a slowing of the conduction of sensation and also, in the absence of irritation, of contact and pain sensation (*double sensation*). I have several times observed this among my patients.

In a small number of cases we find no trace of objective disturbance of sensation, even on careful examination. On the contrary, there are some in which sensory disturbance and pain are so marked that these have been considered a purely *sensory form of polyneuritis.* I shall presently describe these cases, in which ataxia is usually prominent (acute polyneuritic ataxia). The cutaneous reflexes, as a rule, show a decrease of sensation, being either weak or abolished; in hyperesthetic regions they may be increased. When deep sensation is severely affected, *ataxia* necessarily follows, and shows all the characteristics of *sensory ataxia,* particularly a distinct increase on closing the eyes. Romberg's symptom, which is one of the typical features of well-marked polyneuritis, is the first expression of this disturbance. When ataxia is prominent, the gait also is of characteristic type; it becomes stamping, uncertain, the legs diverge widely. In milder grades of ataxia the movements of the hands and fingers are similarly impeded; the patients are clumsy in the execution of all fine movements, as in writing, picking up small objects, in dressing, in buttoning their clothes.

NEUROTABES PERIPHERICA.—Cases in which ataxia dominates the clinical picture, and in which the symptoms of paralysis are secondary deserve special mention. Such cases were first described by Déjérine in 1884 as a special form of polyneuritis which he designated neurotabes peripherica. Remak quite properly declared this designation to be improper and proposed for this form the more suitable term of ataxic polyneuritis; a strict differentiation is impossible, for there are many transitional stages between this and "amyotrophic" polyneuritis. There appear to be no special etiologic factors to account for the ataxic elements in the symptom picture. The affection is most common in alcoholics, but it is also noted in arsenical, diabetic, rheumatic, and post-infectious polyneuritis. It is generally admitted that in postdiphtheritic polyneuritis ataxia is the most marked symptom.

In this condition there is occasionally no evidence of weakness, which rarely happens in cases of other etiology. No matter how prominent the ataxia may be, careful investigation will usually disclose some defect in motion, and we should first direct our attention to the usual points of predilection of polyneuritic paralysis—the muscles supplied by the peroneal nerve. Minute electrical examination often aids us in the diagnosis, which is obscure in those cases in which paralysis and atrophy are uncertain, and in which positive changes of electric contractility denoting partial DeR are not recognizable.

When ataxia is more prominent we occasionally find *spontaneous movements,* which, in the toes and fingers, usually appear as slight flexion, exten-

sion, or abduction, and are sometimes jerking, sometimes slow. They may be painful, but are usually imperceptible to the patient. I shall not here discuss the pathogenic foundation of ataxia. The question whether or not the ataxic movements now so frequently observed in polyneuritis are of spinal origin calls for special consideration, and I shall subsequently refer to this point.

Among the *nerves of special sense*, the *optic* nerve not infrequently shows changes, especially in alcoholic neuritis, the primary ones being pallor of the temporal half of the papilla and *optic atrophy* or *optic neuritis*. These changes are the sign of a retrobulbar optic neuritis, and usually accompany the disturbances in function so characteristic of this affection; we find a marked *diminution of the central acuity of vision*, and *relative* or *central scotomata* for *white* and *colors*. What relation these changes bear to the polyneuritic process is doubtful, because they are found also in chronic alcoholism without other conspicuous neuritic change. Uhthoff found among 1,000 alcoholics no less than 139 with pallor of the temporal half of the papilla. Optic neuritis has also been observed in spontaneous, puerperal, and carcinomatous polyneuritis (Remak, Schanz, and Miura). Gaspero (*l. c.*) found choked disc in his case (previously mentioned); the head of the optic nerve showed a dark, grayish-red discoloration, it was edematous and prominent, the arteries indistinct, and the veins congested. There were no proliferative processes nor did hemorrhages ever appear. All of the phenomena ameliorated. This case must be considered an example of intense inflammatory change at the entrance of the optic nerve (papillitis), since choked disc is occasionally (although extremely rarely) found also in multiple sclerosis, while in this instance, as in polyneuritis, both optic atrophy and optic neuritis were present.

Strümpell reported a case, long considered unique, of a nervous defect in hearing which occurred in the course of polyneuritis, and which he attributed to *neuritis of the auditory nerve*. Alt[1] recently published a case of alcoholic polyneuritis combined with nervous disturbances of hearing which disappeared with the polyneuritic symptoms. At the same time he reported three other cases in which there were symptoms of intoxication of the auditory nerve due to immoderate smoking. In Meyer's case[2] deafness appeared on the third day with general symptoms of fever and, at the same time, disturbances in equilibrium. Neuritic symptoms (paresis and loss of the power of coördination) developed in the course of the fifth nerve and in the extremities. All of the symptoms disappeared except the deafness. Findings on examining with the ear speculum were negative. There was a diminution in the perception of high tones and the perception of bone conduction; subjective noises in the ear, as elsewhere, must in the main be attributed to the nervous origin of the defective hearing.

Bladder disturbances are not a feature of the ordinary picture of polyneuritis. On the contrary we hold it true that when severe bladder disturbances are persistent multiple neuritis is unlikely. On the other hand, it must be admitted that mild and transitory bladder disturbances do now and then

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1 Alt, "Ueber Erkrankungen der Hörnerven nach übermässigem Genuss von Alkohol und Nikotin." *Wiener med. Wochenschr.*, 1903, p. 210.

2 Meyer, "Ein Fall von multipler Neuritis mit besonderer Betheiligung des Acusticus und Trigeminus." *Monatsschr. f. Ohrenheilk.*, 1903, Nr. 2.

appear. We must, however, disregard those cases of delirious, unconscious, insane, or very marantic patients in whom these bladder symptoms occur immediately before death. Oppenheim saw a case of severe polyneuritis in which the catheter had to be used for the first five or six days, after which the bladder disturbance disappeared while polyneuritis developed still further, but finally terminated in recovery. In the severe cases of polyneuritis reported by Kramer there was temporary incontinence of urine and difficult micturition. v. Leyden long ago pointed out the possibility of such an occurrence because of two cases under his observation. Therefore this symptom and a certain weakness of the rectum must be regarded as rare in the clinical picture of polyneuritis. It is at present uncertain whether we can assume *neuritic* lesions to be the anatomical foundation of these disturbances or such changes in the posterior column of the *spinal cord* as have been observed in polyneuritis with increasing frequency in the last few years.

Among *vasomotor, secretory, and trophic* disturbances edema is most common. I refer to true edematous swelling in the distal portions of the extremities (back of the foot, the ankle, the back of the hand), pitting persisting for a long time after the pressure of the finger, but decreasing while at rest in bed. This can be regarded as a symptom of neuritis only after all other factors (a complicating cardiac affection, nephritis, or cachexia) have been excluded. If there are no such complications, the edema can be referred to implication of vasomotor nerves, and to this we also attribute the other vasomotor symptoms which occasionally appear, such as *cyanosis, erythema,* attacks of *syncope,* and *local asphyxia.* Trophic disturbances are sometimes combined with vasomotor symptoms, such as *glossy skin,* which is much rarer than in traumatic neuritis. In another article I have expressed myself concerning the doubtful relations of *Raynaud's disease* and *erythromelalgia* and need not here repeat what was stated. I shall merely emphasize that perforating ulcer of the foot (*mal perforant*) is not a symptom of polyneuritis which is of any importance in the differential diagnosis from tabes; on the contrary, it is a condition occurring in chronic alcoholic intoxication. Adrian[1] refers the changed composition of the blood in such cases to a neuritic genesis. In two cases of Bonhöffer's[2], extensive pemphigus-like vesicles appeared in different areas and became gangrenous, even those not subjected to pressure, and they were regarded as the direct cause of death. In a case of Oppenheim's, lupus developed after the onset of polyneuritis and persisted after the latter affection had disappeared. In a case which we shall later study as an example of incomplete recovery, Fränkel saw *multiple granulation tumors* the nature of which he could not understand, but which he did not attribute to neuritic changes. There are cases in which both vasomotor and trophic disturbances are very prominent; chief among these is a form of beri-beri regarded as polyneuritis. Similar and isolated cases have been observed when there was no *endemic polyneuritis,* and these were described as *dropsical polyneuritis.*

Secretory disturbances, particularly *hyperhidrosis,* are not rare in any form of polyneuritis, but are especially marked in *arsenical polyneuritis.* It is doubtful what position we can assign to the *arthritic swellings* which appear

[1] Adrian, "Das Mal perforant," Jena, 1904.

[2] Bonhöffer, "Die acuten Geisteskrankheiten der Gewohnheitstrinker," Jena, 1901.

in the various stages of polyneuritis. When they develop as multiple, painful arthritic effusions, accompanied with fever and preceding the neuritic symptoms, they are generally regarded as belonging to acute articular rheumatism and as etiologically important in the development of the neuritic affection. The solitary or multiple arthritic swellings occurring at the height of the affection must be regarded as symptoms of polyneuritis, but their relations are by no means clear, and their strict division into the two groups mentioned can by no means always be made. I recently saw a case of amyotrophic plexus neuritis in an elderly lady, in whom a painful effusion into the shoulder-joint had occurred and closely simulated a neuritic affection.

Mental disturbances appear in all forms of polyneuritis, but are most common in alcoholic polyneuritis. This, as I have stated, may develop from a preceding and ordinary but acute *delirium tremens,* which runs a typical course in a few days. But the critical sleep which usually terminates an attack of delirium may not supervene. The delirium persists while the hallucinations become less real, and with steady progress new psychopathological phenomena appear, a pathologic condition which was first described by Korsakow under the name of *neuritic psychosis* or *cerebropathia psychica toxæmia.* As Jolly suggested, we now usually designate this as *Korsakow's syndrome.* Bonhöffer [1] gives us a clear description of this psychosis. After the introductory stage of delirium, stupor follows, or a slow but progressive impairment of memory. *Defective memory* is characteristic of the psychosis, *inability to associate* places and scenes, and "*confabulations.*" The impairment of memory may be extreme. The time of day, the date, and all that concerns the patient himself, are forgotten in a few moments. Syllables and numerals are also forgotten in the shortest time. Not only is memory defective for occurrences during the time of the disease, which is readily explained on other grounds than by a loss of memory, but it almost invariably applies also to circumstances of previous months or years (*retroactive amnesia*). Patients may forget that they are married, or that they have children. They believe themselves to be again living in times long past, and are lost to their present surroundings. Finally loss of memory is closely related to the symptom of confabulation. The patients attempt to cover the gaps in their memory by all sorts of excuses, at the same time spontaneously relating adventurous tales which bear a close resemblance to their distorted dreams. In spite of their fantastic and extravagant descriptions the absence of emotion is remarkable. In contrast to these serious disturbances, the *power of reflection* is often well retained in these patients, and their deportment is excellent. These symptoms may remain at their acme for weeks or months. As a rule, they disappear more slowly than the neuritic symptoms. According to Bonhöffer, whose experiences relate only to the alcoholic form of this psychosis, recovery is never complete; the patients are no longer able to follow their occupations, memory is defective, and it seems impossible for them to take the initiative in anything. Other authors have reported a more favorable outcome.

This psychosis is noted also in other forms of polyneuritis which are not of alcoholic nature. It has been described after septic, post-typhoid, tuberculous, gastrointestinal and post-influenzal polyneuritis. Oppenheim saw a

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[1] Bonhöffer, *l. c.*

case in which the continued use of pyramidon was the only apparent cause. The relation between the neuritic and mental symptoms is not always marked; the former may be less prominent, or even absent.

The symptoms which have been described may be combined in a single case in manifold and varying ways. Their sequence and development may be more or less rapid, and may show various limitations, yet these may be neither so positive nor conspicuous that a classification based upon them would be of value.

Leyden divided the long course of the affection into four stages: the *initial stage*, the *progressive stage* when the paralysis is distributed, the *stage of acme*, the process being arrested at its height, and the *stage of regeneration.*

The initial stage, especially in the not too severe cases of alcoholic neuritis, may be prolonged through weeks or months. As, in the case I reported, it is often difficult to differentiate the sensory irritative phenomena and the general weakness, especially of the legs, from the more common symptoms of chronic alcoholism, and to ascribe them to a beginning polyneuritic process. In marked contrast to this, the mental phenomena of delirium tremens, or Korsakow's psychosis, may appear acutely and clearly mark the onset of the affection. Severe constitutional and gastrointestinal symptoms, fever, anorexia, vomiting, diarrhea and lassitude may mark the onset of the affection in the alcoholic as well as in other forms of the disease. A fulminant onset usually prefigures a similar course. The paralysis attacks the distal portions, usually of the lower extremity, in rapid succession, one member after another being attacked, both arms and legs soon becoming incapable of function. Both the recumbent posture and the power to turn around become impossible, and respiration is impeded. Symptoms also appear in the cerebral innervation. There is paralysis of the muscles, of the face, of deglutition, of speech, and of the eyes, and, even at the beginning of the second week, more often after a longer interval, respiratory and cardiac disturbances are the prelude to death. Some of these cases have been considered to be *Landry's paralysis*, and Eichhorst described such a case as *acute progressive neuritis.* Notwithstanding these alarming symptoms, in spite of the implication of the pneumogastric, the phrenic and the facial nerves, etc., I maintain that at any moment the affection may be arrested and recovery ensue.

The more *protracted cases* in which the paralysis is of slighter extent are more common. Here, at least in the early stages, pain and hyperesthesia are often prominent in the clinical picture. The paralysis is but slowly progressive, and halts before it decidedly involves the muscles of the trunk and the bulbar portions. Naturally its progress is not always continuous; the neuritic process may attack isolated proximal areas, both spinal and cerebral, may cause paralysis of the phrenic, pneumogastric, or facial nerves, without implicating the entire musculature of the extremities and trunk. According to Remak, its duration varies from two weeks to about two months, and during this time the prognosis is always grave. Aside from the localization of the paralysis, which in itself may be a menace to life, the prognosis depends also upon the general condition of the patient. If he is weak from a preceding infectious disease (enteric fever, sepsis, influenza, etc.), or if chronic intoxication is the foundation of the process (alcoholism, diabetes, etc.), or if, as so often happens, a complication such as tuberculosis is present, the prognosis is more serious,

and the patient perishes in this or the next stage. Bladder disturbances are of unfavorable prognostic import inasmuch as they show the extension of the process or the implication of the spinal cord. From its transitory nature, the cystitis is not very serious, nor is there much danger of the bed-sore which is so common in complete paraplegia, although to this rule there are exceptions (the cases reported by Bonhöffer).

A polyneuritis which affects the distal extremities in general, as well as other forms with less widely distributed and distinct phenomena (abortive forms, rudimentary cases with varying localization) offer a favorable prognosis. But in the majority of cases the *course* is very protracted. After the paralytic phenomena become well marked, they usually persist for a long time and until the pain disappears—often from two to three months or longer; very favorable cases terminate in three months. In the more severe cases the *deformities and contractures* of which I have spoken develop subsequently. Pes equinus, the inevitable result of the paralysis, which more frequently affects the extensors of the foot and toes than the flexors, may become chronic, for the muscles of the calf are forced to sustain permanently an excessive weight. Moreover, the fibrous degeneration of muscles may cause retraction. As in the cases above reported, we were several times enabled by palpation to demonstrate such a fibrous hardening of the muscles. Oppenheim reported an ossification of the joints and tendons. Contractures may also develop in the knee-joints (flexure contracture), in the joints of the upper extremities (drop-wrist), and an attempt must be made from the onset to prevent these by a proper position and braces, and the early use of passive movements.

The great majority of cases end in *recovery*, but in severe cases this may require years. Not only do the paralysis and atrophy slowly yield, but also the pain on pressure as well as on active and passive motion. Sometimes hyperesthesia disappears but tardily. It has been previously stated that the reappearance of the tendon reflexes and of normal electric contractility is frequently long delayed. The paralyses usually disappear in an inverse sequence from that in which they originated. When recovery is incomplete, those distal regions which were first and most severely attacked, are the ones which are the seat of residual paralyses and anesthesia; although this rule is by no means without exception. On the whole incomplete recoveries are rare, and they naturally are most common in the toxic, especially the alcoholic, forms of neuritis in which the effect of the intoxication continues. I will now quote from Fränkel (*Deutsche med. Wochenschr.*, 1896, Nr. 45) the history of a patient whose recovery was incomplete, this case being also mentioned by Oppenheim in his text-book (page 529).

CASE 23.—The patient states that, in the year 1894, he suffered from a severe polyneuritis of the arms and legs which implicated also the left half of the face. Now, after the lapse of ten years, he applies for treatment on account of weakness, continuous pain, and paresthesia in the legs. Both feet show pes equinus, the toes in the position of marked plantar flexion. The leg is emaciated, the right knee-jerk is distinct, the left feeble, and the Achilles tendon reflexes are absent. The muscles of the leg and their nerve trunks are sensitive to pressure, particularly the left peroneal nerve, which appears to be thickened. On the left side the sciatic phenomenon is present. The dorsal flexors of both feet act imperfectly, but retain some power. The region of the peroneal nerve shows a marked decrease in contractility; there is no DeR and no hypesthesia, but merely *hyperæsthesia plantaris*. The grasp of the hand

is somewhat weak. In the left facial nerve we note a conspicuous paresis and contracture, also constrained movements and a diminution of the electric contractility.

Another case, which I saw in the private practice of Professor Oppenheim, recovered, but there was paresis of the muscles of the abdomen (post-typhoid polyneuritis); paralysis of the ocular muscles may persist as well as other conditions.

The prognosis is rendered less favorable by the somewhat common relapses, and we must omit from consideration those cases in which the continued action of a deleterious factor may cause another attack (alcoholic and lead neuritis). Such relapses may occur after several months or after years; a case was reported in which this happened after 11 years. Cases are also reported in which the affection appeared annually at about the same time (Oppenheim).

The preceding description of the symptomatology applies mainly to that of alcoholic polyneuritis; I have not, however, wholly confined myself to this form, but have included the spontaneous, rheumatic, and infectious forms and have now and then described others. Concerning some of these I must be somewhat more explicit, partly because they are of practical importance, and partly—with the same fundamental symptoms—they vary so much as to merit a special description.

Arsenical Polyneuritis.—This is symptomatologically analogous to the form described. It may appear after the administration of a single large dose with suicidal or murderous intent, or by accident, but it may also develop after the prolonged effect of the poison taken medicinally or from chronic intoxication caused by living in rooms covered with arsenical wall-paper, handling artificial flowers, etc. Müller has lately asserted that this affection is relatively common among so-called arsenic eaters who were formerly supposed to be immune to arsenical paralysis. A few years ago arsenical neuritis was widely prevalent in England. It was traced to a combination of glucose with sulphuric acid containing arsenic, which was so largely used in the manufacture of beer that even moderate beer-drinkers were poisoned. Many English investigators (Reynolds, who discovered the admixture of arsenic in the beer, Ross, Buzzard, and others) reported these cases, and enriched our knowledge of this affection. Bordas reported that no less than 4,181 persons, principally in Manchester and its vicinity, were poisoned, and that at least 300 died. Some authors ascribed the intoxicating effect to the selenium present in the sulphuric acid or the beer. The quantity necessary to produce intoxication varied decidedly. Some were attacked after the consumption of only small quantities, and some infections—influenza, for instance—were markedly predisposing. It is significant that arsenical neuritis has often been observed after treatment by arsenic (usually the administration of Fowler's solution in chorea). Karplus and Kinsman saw symptoms after the administration of about 50 grams of Fowler's solution.

Case 24.—A woman, aged 40, on the 21st of November, 1903, took arsenic in unknown quantity with suicidal intent. Soon afterward severe gastrointestinal symptoms appeared, such as vomiting, diarrhea, and cardiac palpitation. After this acute gastroenteritis had run its course—i. e., after 8 days—the patient complained of sensations of cold and formication in the calves, followed by a dull sensation in the hands and feet, severe tearing pain and increasing weakness in the legs and arms. The

hands could not be used for the finer manipulations. Walking was irksome, and only possible with support on both sides. The affection reached its acme in January, and then gradually receded. There were no bladder symptoms. On the 4th of June, 1904, the following conditions were noted: there was no disturbance of the cranial nerves, and no muscular atrophy in the upper extremities; the nerves and muscles were insensitive to pressure, the tendon reflexes were preserved, the motor power was good, the sensation on the volar surface of the finger to brush contact was diminished, the left knee-jerk markedly decreased, the right abolished, as well as the Achilles tendon reflex. There was swaying on closing the eyes, and pressure upon the muscles of the calf and the peroneal and posterior tibial nerves was very painful. In both legs the muscles supplied by the peroneal nerves were weak, especially in the left, and the flexors of the feet were by no means normal. There was bilateral hypesthesia and hyperesthesia on the dorsum of the foot; no ataxia. Electric examination revealed partial DeR in the peroneal region and in the flexors of the toes. Hands and feet showed a persistent and abnormal secretion of sweat; there were no conspicuous deformities.

The patient presented the typical symptoms of *arsenical polyneuritis* in the stage of improvement. When the poisoning is acute a period of 8 to 10 days or even of a few weeks elapses before the appearance of the neuritic symptoms. Exceptionally, as in a case of Kron's,[1] after minor intestinal symptoms, pain and paralysis appear within 24 hours after the poisoning. In a case of Korzou's, severe pain set in after 3 days. The sequence of the symptoms is invariably the same: paresthesia, pain, paralysis, atrophy. The paralyses show a predilection for the *distal* portions of the extremities—the peripheral portions. The *legs* are most seriously affected, but the arms are more frequently implicated than is the case in alcoholic neuritis. In our patient the paralysis had disappeared from the arms when we saw her the first time. The small muscles of the hand are first attacked, then the extensors, perhaps also the long flexors of the fingers. In a case of arsenical polyneuritis which I recently saw, the small muscles of the hand as well as those innervated by the radial nerve were severely implicated. As in the case just reported, the extensors of the legs were chiefly affected, especially the extensors of the toes. Electric examination showed that, as in the case reported, the flexors were not exempt. The absence of the knee-jerk almost invariably points to the extension of the neuritic process. An increase of the knee-jerks at the onset has also been observed. In severe cases paralysis, atrophy, and DeR—all three of which are combined—are more extensive. Kron's patient could neither cough nor exert abdominal pressure. In this patient incontinence and weakness of the bladder appeared and were so persistent that for three months she had to be catheterized; there was also *incontinentia alvi.* The *cerebral nerves* are usually not implicated, even in severe cases, and this fact may aid us in the diagnosis of Landry's paralysis when the course appears to be a rapidly ascending one. Cardiac palpitation here, as in a few other cases, was attributed to an implication of the pneumogastric nerve. Recovery occurs according to the rule above mentioned, that the muscles last paralyzed first regain their motility. When the paralysis is persistent and recovery is complete, contractures may appear in the hands and feet. In the majority of cases there is retraction of the flexors of the feet, especially the toes, and in the hands and fingers (Erlicki and Rybalkin). Exceptionally a flexor contraction has been observed in the knee-joint.

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[1] Kron, "Ein Fall von Arseniklähmung." *Neurol. Centralbl.*, 1902, p. 930.

Sensory irritative symptoms and those due to absence of function are prominent in the clinical picture. The *pains* are often of extreme intensity, the sensitiveness of the nerves and muscles to pressure is very conspicuous and long-continued. In addition to the *hyperalgesia,* and even without this, objective sensory disturbances are reported in the majority of cases, the distal portions being most severely implicated, and the proximal less so.

In the case of a woman with arsenical poisoning a year after the appearance of the neuritis, the involvement of the hands was shown only by hypesthesia and hyperhidrosis. *Ataxia* could not be demonstrated in our patient, at least while under observation, but it appears to have been an earlier symptom. In my recent case it was conspicuous, affecting both arms and legs, a so-called *static ataxia.* On the whole it is very common, but is usually not so marked as the paralytic phenomena. Yet the inverse may be true. Ataxia may precede the paralysis, which for a long time may be but slight. The clinical condition then resembles tabes (*pseudo-tabes arsenicosa*), but there is no danger of confounding these diseases if sufficient attention be given to the development, the sensitiveness to pressure, and the absence of bladder disturbances.

The frequency of vasomotor, secretory, and trophic disturbances is remarkable. *Hyperhidrosis* is reported as extreme in many cases. In a case of Jolly's there was conspicuous *glossy skin. Herpes zoster* from the medicinal employment of the drug (arsenic) has been observed as an isolated symptom after neuritis. Erythromelalgia has been frequently reported, particularly by English authors. *Alopecia, abnormal pigmentation, changes in the nails,* erythema, and edema have all been observed. Reynolds attaches great significance to *keratoses* of the skin and to *pigmentation* which are often observed in arsenical paralysis.

Psychical disturbances are more rare than in alcoholic neuritis, but Korsakow's symptom-complex is occasionally observed, as well as impairment of memory and similar conditions. In some respects the prognosis of arsenical polyneuritis is favorable. Among 130 cases Alexander saw only 2 who had permanently lost the power of walking. A few cases were fatal (pneumonia, paralysis of the heart),[1] but the course may be greatly protracted. In Kron's severe case, walking and even standing were impossible after the lapse of two years, but electric treatment instituted at this time brought about marked improvement; a permanent cure and incomplete restoration to health with defects varying in degree have repeatedly been observed. The diagnosis is confirmed by the demonstration of arsenic in the urine, the hair, and the nails.

Lead Paralysis.—Lead paralysis deviates in many ways from the picture of ordinary polyneuritis. It is a common affection, probably always the result of *chronic* intoxication, which in the majority of cases is acquired from the occupation. It is most frequently seen in painters, typesetters, lacquerers, plumbers, workers in accumulator (storage batteries) factories, etc. In consequence of the great amount of lead used in the last mentioned occupation the danger of intoxication is said to be particularly great at first, and the disease appears with extraordinary rapidity (Guillain et Lhermite). There are many other occupations which occasionally expose people to lead poison-

[1] The cases in the English epidemic were much more unfavorable.

ing, for instance, those of workers in artificial flowers, paper glazers, and weavers among whom poisoning by lead was formerly common, but now under more favorable hygienic conditions is much less so. Intoxication has also occurred from the ingestion of water which had been conducted through lead pipes, from an admixture of lead in flour (Weber), in children, from playing with toys containing lead (Variot), from cosmetics and from the use of snuff containing lead, etc.

Lead paralysis is scarcely ever the first sign of poisoning. Among the symptoms which, in the individual case, are often valuable in diagnosis I must mention the *lead line* on the gums, *pallor of the face, tremor, muscular and arthritic pain*; also objective findings such as reports of a single attack of *lead colic* or repetitions of it; finally symptoms of general functional *nervous weakness* and irritability, and severe cerebral symptoms (lead encephalopathy). It must be borne in mind that many patients are attacked by lead paralysis without ever experiencing an attack of lead colic. In comparison with other symptoms of lead poisoning, such as colic, myopathy, and neurasthenia, lead paralysis is much less common. Oppenheim reports a case of "hereditary" lead paralysis in a child.

Lead paralysis owes its special prominence to two circumstances: First, the *absolute insignificance of sensory irritative symptoms* and those due to loss of function; second, to the conspicuously *elective character of the paralysis.* The predilection of some muscles to, and the exemption of others from, the disease is more striking than in the preceding cases of polyneuritis. Remak therefore designates it as *elective systematic neuritis or polyneuritis.*

In typical cases we find the following picture: A patient whose occupation has for a long time exposed him to the effects of lead, and who shows signs of chronic lead intoxication, reports to the physician that he has noticed a weakness in the hands, especially of the right, which has slowly increased. He has no pain or numbness. Examination shows degenerative paralysis in the extensors of the hand and fingers. The extensor digitorum communis appears to be the earliest and most severely implicated, especially the parts extending to the second and third fingers. The remainder of the extensor digitorum communis, the indicator (index finger), and the extensores pollicis are next affected. As the process advances the extensors of the hand are involved, the extensores carpi radiales usually prior to the extensor ulnaris. The characteristic "drop-wrist" appears. In grasping there is marked volar flexion of the hand, and its power is more or less decreased. Last among the muscles of this group the abductor pollicis longus is implicated. On the other hand—and this is of the greatest diagnostic importance—the supinators, especially the supinator longus, as well as the triceps, are exempt in typical cases. *Thus the paralysis reveals a distinctly selective action among the muscles supplied by the radial nerve.* In the severe cases, which are not rare, more muscles are involved in the paralysis, and the *small muscles of the hand* supplied by the median and ulnar nerves are implicated. First the interossei, particularly the first interosseous muscle, and the muscles of the ball of the thumb are affected; then, as a rule, prior to the implication of the abductor pollicis longus, another muscle, the deltoid, is paralyzed, although only partially.

CASE 25.—The patient is a painter, aged 35; he has a lead line upon the gums, has suffered from repeated attacks of lead colic, and for three months paralysis has gradually been involving both hands and arms. The extensors of the finger and thumb are completely paralyzed. The extensors of the hand show but little power, the abductor pollicis longus is somewhat stronger, but there is a distinct wrist-drop. The extensor muscles of the forearm are atrophic, only the supinator longus becoming prominent on flexion, and showing normal power. The interosseous spaces are depressed, particularly the first, and the ball of the thumb shows atrophy. The power of the corresponding muscles as well as that of the abductor pollicis longus is decreased. It is evident that he cannot fully raise his arm, and partial DeR is especially distinct in the deltoid as well as in all of the paretic muscles. The disturbance is more marked in the right arm, but shows no sensory changes.

As in our patient, lead paralysis invariably appears as a degenerative form of atrophy. DeR is occasionally noted in muscular regions in which neither paralysis nor atrophy can be demonstrated. From this type of paralysis there are, upon the whole, very few deviations. It may be that paralysis and atrophy *are more prominent in the small muscles of the hand,* developing simultaneously with the paresis of the extensors and to a similar degree. Usually a special cause is evident, primarily the over-use of the muscles in the occupation (the *toxico-professional paralysis of Oppenheim*). An affection of the muscles of the ball of the thumb has been reported by Gowers, Bernhardt, and Remak. Another uncommon *type* is that involving the *upper arm,* first reported by Remak. Then come the rare cases in which the deltoid is implicated, the other Erb's muscles, the biceps, the brachialis internus, and even the supinator longus and brevis. But these forms of paralysis are added to an already existing extensor paralysis. A saturnine, total paralysis of the plexus has been described by Ladd.

In adults the lower extremities are usually exempt. In 98 cases Remak saw these affected only thrice; the muscles supplied by the peroneal nerve are frequently, although not always, attacked while the tibialis anticus is unaffected, Koester saw a very unusual localization of the paralysis and atrophy in the interossei and the abductores hallucis so that a *claw-foot* appeared. It is remarkable that in all the lead paralyses of youth the lower extremities are implicated, and show the peroneal type just described.

The *bulbar* nerves are usually exempt. But cases have been described of paralysis of the vocal cords in which there was either unilateral or bilateral posticus paralysis, or a total paralysis of the recurrent laryngeal. Persistent acceleration of the pulse has been mentioned as a symptom referable to the vagus; paralysis of the spinal accessory as an isolated saturnine neuritis. *Retrobulbar neuritis of the optic nerves* with the same symptoms which appear in alcoholic neuritis has frequently been reported, as well as paralysis of the ocular muscles from lead. Some of the symptoms attributable to the cerebral nerves are difficult to differentiate because they occur in a so-called *encephalopathia saturnina,* a full description of which is impracticable in this article. Therefore the *neuritic nature* of these paralyses should always be demonstrated—a proof several times lacking in some facial paralyses which have been reported (Bewy, Debove). Finally, there is a *general saturnine polyneuritis,* of which but few cases are known. Some of these were marked by a febrile onset and a most acute course. Even the muscles of the trunk were implicated, and there was paralysis of the diaphragm with fatal asphyxia.

From the onset there was tearing pain, which is usually absent, but there were no objective sensory disturbances.

Typical lead paralysis yields, certainly the first attack, in the course of several months, or it improves decidedly. The extensors of the fingers, particularly in the second and third fingers of the right hand are the last to regain their power, and even in the first attack these may remain paretic. If the patient, as often happens, again exposes himself to lead intoxication there is a *relapse of the paralysis,* and in these relapses the prognosis is always unfavorable. Even without renewed exposure to the poison, relapses may occur from the effect of other deleterious agents (alcoholism, over-exertion). The therapeutic indications are obvious. Sulphur baths and potassium iodid have long been employed to promote the rapid elimination of the poison. Otherwise the treatment, especially the electric, is the same as for other forms of polyneuritis which I shall describe in detail. This is also true of the diagnostic considerations provided there is no special etiology. Here we find in the *characteristic localization an extremely valuable proof of the saturnine etiology.* The *absence of sensory disturbances* is also of great significance. It becomes necessary to distinguish this from certain forms of chronic, subacute, cervical poliomyelitis which, at least occasionally, produce a picture resembling lead paralysis, but which subsequently runs an entirely different course. Careful search must be made for the occasionally concealed source of the intoxication, and the signs of chronic lead poisoning must be considered. Of course, atypical cases can be explained only when the etiology is known.

There are other forms of *toxic polyneuritis* which, in comparison with that described, are of secondary importance. Gowers reported after the use of *silver pills* a paralysis resembling the lead form. A few cases of *copper neuritis* in copper workers have been observed, but Lewin doubts that copper exerts a toxic action. *Phosphorus,* whose toxic action is so evident in other regions, is rarely dangerous to the peripheral nervous system; but Leyden and Henschen have reported such cases. In Henschen's [1] last case there were merely symptoms of sensory irritation and slight impairment of function. Löwenfeld describes neuritic phenomena after taking *creosote containing phosphorus.* Erbslöh anatomically proved a polyneuritis after the *administration of sulphonal,* but carcinomatous cachexia and hemorrhage were also mentioned in the history.

Mercurial polyneuritis, which we shall discuss with *syphilitic polyneuritis,* is of somewhat greater interest. It is certain that working with mercury does not generally cause neuritic phenomena. Whether polyneuritis can be produced by the use of mercury administered as a drug is difficult to decide because this treatment is usually instituted in syphilitics, so that at least two etiologic factors for neuritis are present. There are, however, a few cases in which the etiologic influence of mercury cannot be doubted (cases of Ketly, Spillmann, Etienne, Spitzer,[2] and others). In these instances either there was no syphilis (corrosive sublimate was taken with suicidal intent in

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[1] "Ueber Phosphorneuritis." *Neurol. Centralbl.,* 1900, p. 555.

[2] Spitzer, "Ein Beitrag zur Kenntniss der mercuriellen Polyneuritis acuta." *Deutsche Zeitschr. f. Nervenheilk.,* Bd. XIX, p. 215.

Ketly's case, and by accident in Faworski's), or the discontinuance of the mercurial treatment, as in Spitzer's patient relieved the polyneuritis while the luetic manifestations persisted throughout the entire body and even fresh symptoms developed. Brauer considers the experimental foundation upon which Letulle and Heller attempted to base mercurial polyneuritis unreliable. *Syphilitic polyneuritis,* the differentiation of which from the mercurial form is, for reasons, above mentioned, most perplexing, is proven in a few cases (Schultze, Oppenheim, Cestan).[1] When its syphilitic nature is recognized, we can exclude all other etiology (alcohol, mercurial poisoning, autointoxication from a syphilitic, hepatic, or renal affection). In one of Cestan's cases polyneuritis appeared 4 weeks after a chancre, in the other 6 weeks afterward; mercury was beneficial. In a few other instances, mercury aggravated the patient's condition, therefore this is not an absolutely certain indication of the etiology. Polyneuritis cannot be regarded as an actually specific disease, but as due to the toxins generated by syphilis. Neither the symptoms of mercurial nor of syphilitic polyneuritis absolutely prove its etiology. In the secondary stage of syphilis we sometimes observe a facial mononeuritis which we attribute to a similar syphilo-toxic origin.

Carbon bisulphid poisoning, which has been much studied in the last few years, may in conjunction with other nervous disturbances, partly functional and partly of organic, cerebral origin, produce neuritic and polyneuritic symptoms; but the number of these cases is small and in comparison with the other phenomena of poisoning the symptoms of neuritis are secondary. Experiments (Koester)[2] have shown that the poison has little affinity for the peripheral nervous system. No neuritic phenomena can be demonstrated in the rabbit after the inhalation of carbon bisulphid; on dipping the paw of the animal into a solution made from this agent, changes are apparent in the affected part, but we are not certain of their neuritic nature.

Neuritic and polyneuritic phenomena are more common after *carbonic oxid intoxication.* This poison produces various localized pathologic phenomena in the nervous system as well as in the vessels and the skin (edema, bed-sores, abscesses, and gangrene). Remak assumes that the symptoms on the part of the peripheral nervous system are circumscribed perineuritic processes originating from the vessels, to which those of neuritis are subsequently added. The paralysis of carbonic oxid poisoning is peculiar in that it shows a predilection for the members which are exposed to pressure during the asphyxia, hence traumatic factors appear to be also operative. That there are exceptions to this rule is evident from the fact that paralyses appear in the course of nerves in which there is no possibility of pressure (for instance, in paralysis of the ocular muscles which, as in a case of Mezkowski's, may be combined with polyneuritic symptoms). It is noteworthy that this paralysis occurs immediately after the poisoning, and is apparent as soon as the person comes out of coma. It is usually confined to a nerve or plexus region. When nerve paralysis is multiple, the affected nerves of the side subjected to pressure present the signs of hemiplegia as well as of neuritis. Isolated forms of

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[1] Cestan, "La polynevrite syphilitique." *Nouv. Iconogr. de la Salpêtrière,* 1900, p. 153.

[2] Koester, "Zur Lehre von der Schwefelkohlenstoffneuritis." *Arch. f. Psych.,* Bd. XXXIII.

toxic neuritis have been noted after poisoning by *toluol* and *nitrobenzol.* I have now enumerated the most important of the *toxic forms of neuritis,* and must reiterate that the *etiology* is frequently a *mixed* one. Various external poisons, particularly lead and alcohol, may be combined. and, what is even more common, the intoxication may affect a portion of the body already diseased. Here tuberculosis has a paramount influence. Tubercular alcoholics are especially predisposed to neuritis. Inversely, as I have several times stated, alcoholic neuritis predisposes to other infections.

Diphtheritic Polyneuritis.—Of the many forms of infectious and post-infectious polyneuritis that have been considered, there are a few which, on account of the substantive position they occupy, I must describe somewhat more in detail: these are *diphtheritic polyneuritis, leprous neuritis,* and *beriberi.* While the other post-infectious forms of polyneuritis differ but little from the original infection, the conditions are different in these three forms, the most important and most common of which is diphtheritic polyneuritis.

Case 26.—A school-teacher, aged 25, had in November, 1904, an attack of diphtheria which was treated with diphtheria antitoxin. After the wound had healed the patient noticed that his sight was affected, his vision being dimmed; he had previously observed a change in his speech, which had become nasal. Deglutition was difficult, cough appeared, and fluids were regurgitated through the nose. These disturbances improved, but to some extent persisted. Early in January, 1905, he noticed formication and numbness in his legs and arms. The legs became weak, particularly the right, and he subsequently had pains in the legs; there were no bladder disturbances. The gait was uncertain. The patient was easily fatigued, and felt himself in danger of falling. He was unable to recognize small objects distinctly by the sense of touch; as, for instance, on putting his hand in his pocket to take out money. It was impossible for him to put on his stockings or to know whether the stockings were on his feet without looking. On washing his face he could not distinctly feel his hands. Examination of the strong, robust-looking young man revealed the following conditions: Speech is distinctly nasal; deglutition of fluids is very difficult, causing cough, and the regurgitation of some of the fluid through the nose. The soft palate rises imperfectly, and the palate reflex is weak but not absolutely abolished. Mastication, the movements of the lips, and of the tongue are undisturbed. Sensation is intact in the face. The pupils react promptly, the ocular movements are free, but there is a moderate paresis of accommodation. The tendon reflexes of the arms are arrested, but there is no atrophy of the muscles. The grasp of the hand is quite weak, and the apposition of the thumb is decidedly paretic on both sides. The nerve trunks are insensitive to pressure; besides the marked paresthesia of which the patient complains, there are secondary objective sensory disturbances. Pain, temperature, and tactile senses are unimpaired. The sensation of position is disturbed. The so-called *vibration sensation* is most distinctly affected, as well as the power of recognizing objects by touch, especially with the right hand. There is also a mild degree of ataxia. The gait is waddling and uncertain; Romberg's symptom is noted. The knee-jerk is markedly diminished, and can be produced only with the aid of Jendrássik's grasp. The Achilles tendon phenomena are absent. The nerve trunks and the muscles of the leg are tender on pressure. The motor power of the leg is moderately decreased, but this is not strictly localized. The movements of the leg are slightly ataxic, even when the patient is in the recumbent posture. Superficially, sensation is not impaired; but deep sensation, especially that of vibration, is wholly lost. The pulse is slow and weak. The cutaneous reflexes are preserved. Electric contractility reveals nothing.

If we had no knowledge of the etiology of this case we would at once make a diagnosis of *polyneuritis,* which differs somewhat though from the typical form of the disease. Instead of paralysis, ataxia is most prominent

(*ataxic polyneuritis*), but the paralysis implicates definite structures, which are usually uninvolved, and we find *paralysis of the palate,* of the *muscles of deglutition* and of the *pharynx,* as well as *paralysis of accommodation.* These symptoms give to diphtheritic polyneuritis its characteristic stamp. They are so typical that this form of polyneuritis can usually be attributed to diphtheria.

Post-diphtheritic paralysis attacks adults just as frequently as children. In its various forms it is by far the most common of the nervous sequels of diphtheria. Cerebral hemiplegia rarely follows diphtheria. Polyneuritis appears to occur just as often in cases treated with as without injections of serum; Slawyk noted it respectively in 5.5 and 5.3 per cent. of his cases. It must be borne in mind that, in the opinion of the great majority of physicians, many more severe cases of diphtheria are now cured than was formerly the case. Woollacott regards the greater frequency of paralysis after serum treatment as the direct consequence of the decreased mortality.

The first and most common symptom of paralysis following diphtheria is *paralysis of the palate,* which usually accompanies hypesthesia of the pharyngeal mucous membrane and the *loss of the soft palate reflex.* It may appear at the end of the first week of diphtheria, but usually not until the beginning of the second week, or even after six or eight weeks. Very exceptionally the condition is unilateral. The soft palate becomes perfectly flaccid and droops; even reflexly there is no contraction, and this accounts for the nasal tones as well as the regurgitation of fluid through the nose. If the electric contractility of the soft palate is tested, we not rarely find partial DeR. Our case proves that such paresis of the soft palate may long persist, for even after three months it was quite distinct, although improvement had begun. In rare instances a longer time elapses before the paralysis yields. Remak saw a case of paresis which had existed for 2½ years recover under galvanism. In a case which Oppenheim saw in an idiotic child, the paralysis was permanent. Paralysis of the soft palate may be the only paresis. This is probably the most common condition; but in many cases other pareses follow, primarily of the *muscles of the pharynx, the esophagus,* and of *accommodation.*

The first is shown by impairment of the power of deglutition. The contents of the mouth find their way into the larynx; this is partly due to hypesthesia of the entrance to the larynx. The disturbance in deglutition may even lead to deglutition pneumonia. Hypesthesia is caused by alterations in the superior laryngeal nerve, but the inferior laryngeal nerve also may be attacked, as is proven by the fact that *paralysis of the vocal cords* has been observed a few times. If the heart is affected, it is usually due to implication of the pneumogastric nerve, which is not rare in diphtheria. We may assume this if the *damage to the heart* does not occur during the first days of the infectious disease, but appears subsequently with the other symptoms of paralysis. Usually there is *bradycardia* and *irregularity of the pulse*; later we frequently note *tachycardia.* This implication of the heart is of the greatest importance in the prognosis, as will be shown later. *Paralysis of accommodation* is as certain an indication of diphtheria as is paralysis of the soft palate; as a rule, it occurs after the latter, and disappears sooner. It is revealed by a sudden disturbance of sight, a characteristic of which is that the pupillary

reaction is always retained, and nothing anomalous is noted in the pupils. On the other hand, although much more rarely, there may be ***paralysis of the external muscles of the eye,*** usually of the abducens, either unilateral or bilateral. Oculomotor paralyses are more seldom seen. Optic neuritis also has been observed in diphtheria (Botton).

The cases in which we observe the foregoing signs of paralysis of the soft palate and of the muscles of deglutition and accommodation, perhaps also the signs of implication of the heart, have been grouped as localized forms of diphtheritic paralysis, and they have been differentiated from those in which the paralysis affects other nerve regions, these having been regarded as generalized or polyneuritic paralyses. But there is no sharp line of demarcation between these two groups. In the cases in which the paralysis is apparently localized, the extension of the pathologic nervous phenomena is occasionally indicated by symptoms which are not subjectively apparent but can be determined only objectively. Foremost among these is the *absence of the tendon reflexes* in the lower extremities, which may appear with the paralysis, and without any other disturbance of motion or of sensation. Of course, this is not an invariable rule; in many cases disturbances appear in the legs after paralysis has fully developed, or even when the patients have begun to improve. Their gait becomes uncertain, they are soon fatigued, and this cannot be reconciled with their improved general condition. Then paresthesias appear, usually first in the legs, then in the arms, as well as the other sensory disturbances which were so characteristic in our patient.

Statistics show that the arrest of the knee-jerk is a common sign. Among 51 cases of diphtheritic paralysis, Rothe found this condition in 15—while ataxia of the legs were noted in only 9, and paralysis much less frequently. Therefore the absence of the knee-jerk may be the only sign of a general condition. Remak found Westphal's sign to be much more common, for among 78 cases of diphtheritic paralysis of the soft palate, the knee-jerk was abolished in 40. As in my patient, we often note that the knee-jerk is not absolutely lost but only greatly decreased, and that it may be evoked by Jendrássik's grasp. In the overwhelming majority of cases the disappearance of the knee-jerk follows paresis of the soft palate; but, according to Baginsky, the disappearance of the patella tendon reflex on one or both sides is the first symptom of diphtheritic paralysis. Since the *Achilles tendon reflexes* have been more accurately studied, we can demonstrate their absence in this stage of diphtheritic polyneuritis. The tendon reflexes may be abolished for a long time, and this symptom may persist months after all other signs of post-diphtheritic paralysis have yielded.

In addition to the disappearance of the tendon reflexes, in some cases other symptoms are noted. The most common is *ataxia,* which is of the usual type: the gait is uncertain and stamping, the movements lack coördination, and this is especially marked on closure of the eyes, but may even be demonstrated when in the recumbent posture. Romberg's sign becomes distinct. Uncertainty of motion may also be apparent in the upper extremities, especially in the hands; the ordinary finer movements of writing, buttoning the clothes, counting money, etc., become impossible. Sensory disturbances, either subjective or objective, may be combined with the ataxia. The first to appear are *numbness, tingling,* and *formication,* especially in the distal parts of the

legs and arms; these were described by our patient. Objective disturbance is shown less by the temperature and pain sense, than by that of contact, particularly the *sense of position,* and that of *deep sensation.* As an expression of this bathyanesthesia, there is a marked decrease or absence of vibratory sensation; this was noted in my case as well as in many similar ones. This sensory disturbance renders the patient incapable of recognizing objects by the touch (*astereognosis*) which was also true of my patient. Although in these cases the ataxia is usually regarded as sensory, yet, as Remak maintains, there is often a disproportion between the sensory disturbance and the ataxia, the former by no means always being demonstrable. A delay of pain conduction has rarely been observed. There may be no pain or it may be present, and pain has been repeatedly observed on pressure over the nerve trunk and muscles, although it is not an invariable symptom. The finer details of all of these sensory disturbances can be more clearly demonstrated in adults than in children.

The implication of the muscular apparatus becomes more and more apparent as *general weakness,* most marked in the distal portions of the extremities, and by a *circumscribed paralysis* which bears the characteristic stamp of degenerative paralysis—atrophy, flaccidity of the muscles, qualitative changes in electric contractility. Among 32 cases Remak found only 9 such pareses, and these in a decreasing ratio were noted in the peroneal muscles, the ilio-psoas, the ulnar and median region, in the deltoid and the muscles of the nape, usually without decided electrical changes. In severe cases the paralysis may reach an extreme degree, and make the patient absolutely unable to move. Then severe disturbance in the muscles of respiration implicates the thoracic muscles; paralysis of the diaphragm may be caused by the involvement of the phrenic nerve, after which the respiration becomes dyspneic. In a case of Epstein's[1] there was apnea necessitating artificial respiration for 11 hours; but in spite of repeated attacks of dyspnea the child finally recovered. An extremely severe case of post-diphtheritic paralysis is recorded in Hansemann's report of his own case. After passing through a series of infections, on the eighteenth day of an attack of diphtheria he was seized with paralysis of the soft palate, and tachycardia appeared. There was absolute motor paralysis of the arms and legs with severe sensory disturbances, also impairment of motion in the lips, the tongue, and the muscles of mastication which are usually exempt from paralysis. The ocular muscles were also involved, and sensory disturbances appeared even in the region of the fifth nerve. Smell and taste were blunted. Similar severe cases have several times been reported by Baginsky. He maintains that there is no rule concerning the electric contractility of the implicated nerve, for contractility may be intact or wholly lost without furnishing a sign of special value in either a diagnostic or prognostic respect. But we must remember that in these ill children an accurate electrical test is scarcely ever possible. In cases much less severe we have found it necessary to desist from an electric examination, or to limit ourselves to superficial and brief exploration lest we do harm to these unruly patients. As a rule, the electro-diagnostic laws otherwise operative are here also binding. The functions of the bladder and rectum are usually un-

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[1] *Deutsche med. Wochenschr.,* 1900, Nr. 41.

disturbed, but exceptions have been reported. As rare symptoms I must mention that Eeman found auditory neuritis in diphtheria, and Bolton reported a case of optic neutritis. Therefore we note that the entire nervous system may finally be implicated. Notwithstanding these exceptions we must bear in mind the regular cases which are extraordinarily characteristic.

The DURATION OF THE AFFECTION depends upon its extent. General paralysis runs as slow a course as in other forms of polyneuritis, hence the affection may persist for many months, but unless death intervenes complete restoration to health almost invariably follows. The course of post-diphtheritic ataxia is usually shorter—from a few weeks to months—and in localized paralysis it is even briefer.

The PROGNOSIS depends mainly upon the involvement of definite nerve and muscle regions and the paralysis. Deglutition paralysis may become dangerous, especially if there is marked anesthesia and suspension of the laryngeal reflex; also when food or fluid enters the respiratory passages and thereby directly produces pneumonia, which may lead to death. Patients of this kind should be most cautiously fed by means of the stomach-tube; perhaps even rectal alimentation may be necessary. Implication of the phrenic nerve is always a serious symptom, but, as Epstein's case shows, hope must not be too soon abandoned, and artificial respiration, possibly inhalations of oxygen, should be resorted to.

The state of the heart calls for special attention. Patients with cardiac disturbance, no matter whether myocarditic or neuritic (this cannot always be clinically differentiated), should be kept in bed, and in severe cases absolute rest should be enjoined. This should not be interrupted, not even for the administration of nourishment, or for the voidance of the dejecta, etc. Of course, the patient should be sedulously guarded from mental disquiet. The heart must be sustained by the ordinary stimulants, and its action kept regular and full. I cannot here discuss the other complications and dangers arising from diphtheria. The paralysis should be treated on the same general principles as other paralyses and will be described later. In localized paralysis of the soft palate and the muscles of deglutition, electricity sometimes gives immediate results (production of the galvanic deglutition reflex by direct stimulation of the soft palate by means of button-shaped electrodes); but, as has been stated, this form of paralysis is usually relieved spontaneously.

Diphtheritic paralyses occur in other than the pharyngeal forms of diphtheria; namely, in diphtheria of the intestine, of the vulva, and of the navel in the new-born. In a case of diphtheria of the umbilicus (Kussmaul) there was at first paralysis of the abdominal muscles, which was interesting in a pathogenetic sense. In a wound of the forearm infected by diphtheria, Oppenheim noted paralysis of accommodation before the general paralysis. I shall later discuss a few points relating to the pathogenesis. The question as to whether the foregoing and typical symptom-complex occurs *without a preceding* diphtheria is difficult to answer. In children who are not closely watched it sometimes happens that an affection of the throat is hardly noticed, and only the subsequent paralysis recalls the fact that the child had such an affection. This corroborates the opinion I formerly expressed that post-diphtheritic paralysis bears no relation to the severity of the original affec-

tion. Merklen and Broc [1] report a case in which there was intense stomatitis and angina tonsillaris which by bacteriologic investigation were positively proven to be of non-diphtheritic character, yet paralysis of the soft palate, tachycardia, paralysis of accommodation, and dyspnea followed. A few similar cases were previously published. Eisenlohr reports cases of infectious, multiple neuritis in which there was no diphtheria but an implication of the muscles of the palate and of deglutition which resembled diphtheritic paralysis; this is not usually observed in polyneuritis. Paralysis of the soft palate and of the muscles of the larynx has been observed even after erysipelas (Oppenheim), as well as after variola (Remak).

Beri-beri or Kakke.—In the Dutch East Indies, Japan, and Brazil, a disease occurs endemically which in other regions is usually sporadic; this is designated as beri-beri or kakke, and, owing to its main features, it has been included among the forms of neuritis multiplex. The etiology of this affection, which is still obscure, will be briefly sketched. Various theories have been suggested, some assuming the disease to be an infection, the pathogenic cause of which is a microörganism that has apparently been demonstrated. The adherents of the infectious theory claim that the deleterious agent which acts upon the nerves is the toxin furnished by these bacteria; others regard it as an alimentary (a ptomaine) intoxication. Some incline to the view that the poison is generated in spoiled rice, or that a pathogenic bacillus developed in this food. Others attribute it to the consumption of fish which is so general in Japan. Still other hypotheses have been proposed; for instance, that the disease is due to the contamination of rice by arsenic, or that it depends upon malaria. No unanimity of opinion has been attained. The question of the etiology remains unsolved; even inoculation experiments (in apes, Wright, *Brain*, 1903, p. 488) have not enabled us to come to definite conclusions. The anatomical foundation has not been determined in all cases. It is certain that neuritic or polyneuritic changes are frequently found, but in autopsies recently performed in 5 fatal cases of beri-beri, Glogner [2] found chiefly a disease of the muscles. Although he does not deny that finer changes take place in the nerves supplying the muscles, he regards these as secondary and considers the entire affection to be a *polymyositis*. Rumpf and Luce [3] declare that the affection is certainly a polyneuromyositis, since inflammatory processes are found in the nerves which supply the muscles. In the *clinical picture* the neuritic symptoms are most distinct: these are pain, paresthesia, sensitiveness of the nerve trunks to pressure, and hypesthesia. The tendon reflexes are at first increased, but later diminished or abolished. Paralysis primarily affects the peroneal region; the gait is the typical steppage gait. As the paralysis extends it reveals an ascending character. Except for the pneumogastric, the bulbar nerves are rarely attacked. The paralysis is often followed by atrophy, which is masked by edema. The electrical changes resemble those of polyneuritis. Bladder disturbances are rare. What gives to this form its special type is the *marked involvement of the heart and vascular system.* The patient soons complains of palpitation and pain in the cardiac region as well as dyspnea. Upon auscultation and percussion anom-

[1] *Arch. générales de méd.*, 1904, p. 1985.

[2] Glogner, *Virchow's Arch.*, Bd. CLXLI, p. 389.

[3] Rumpf and Luce, *Deutsche Zeitschr. f. Nervenheilk.*, XVIII, p. 63.

alous conditions are demonstrated in the heart. Edema is common, particularly over the tibia and in the face; later it may appear in all the paralyzed regions. Dropsy may be extreme. These cardio-vascular symptoms are attributable to changes in the pneumogastric and vasomotor nerves. A primary myopathic change is considered by Rumpf and Luce to be the cause of the hard swelling and subsequent rigid contraction of the muscles. The *course of the disease* varies according to the form and the severity of the affection. If of the pernicious type death may occur in a few days, or, after the lapse of a few weeks or even months, the case may terminate either favorably or unfavorably.

Leprous Neuritis.—I shall not here attempt a clinical description of leprous neuritis, the neuritic symptoms being only partial phenomena of the pathologic picture of leprosy. But the anatomical foundation of the leprous neuritis will be considered, since this represents a leading type of neuritis—namely, interstitial neuritis.

Each variety of post-infectious polyneuritis which has been described in the foregoing pages possesses its own peculiar features. The other forms which occur in infectious diseases present only general symptoms of this affection, and their further description is unnecessary. Any infectious disease may occasionally be the cause of polyneuritis, although the frequency with which this occurs varies. Polyneuritis is the most common after *enteric fever* and *influenza.* It has been observed also after *scarlatina, pneumonia, whooping-cough, acute articular rheumatism, erysipelas, mumps, gonorrhea, and sepsis*; in malarial regions even *malaria* plays an important rôle in its etiology. As has several times been shown by typical cases, localized neuritis may be the sequel of any of these infections.

When Remak differentiates polyneuritis occurring during and after infectious diseases from those based upon a *dyscrasia,* he observes that this distinction is by no means sharply defined. According to his view, among the forms which divide the two groups are those which appear in *tuberculosis,* in which it is questionable whether a toxemic-chemical virus or an autointoxication is the cause. It is certain, however, that in the course of this disease degenerative neuritic processes frequently occur, which were first minutely described by Pitres and Vaillard, then by Joffroy, Strümpell and Oppenheim, and recently by Colella.[1] The cases of so-called latent neuritis with absence of clinical symptoms, in which only at the autopsy degeneration was found in the peripheral nervous system, do not interest us here; but there are a number of cases in which neuritic symptoms develop in a pronounced form *intra vitam.* It is often doubtful in how far the appearance of pain and hyperesthesia in the pathologic picture may be ascribed to neuritis or polyneuritis. Here the diagnosis is positive only if, to the previously mentioned symptoms, there are added those due to absence of sensory or motor function. The last named forms of amyotrophic polyneuritis sufficiently correspond to the well known classical picture of the affection. The weakness begins usually in the lower extremities, affecting the muscles supplied by the peroneal nerve, at times increasing to complete paraplegia. In the upper

[1] Colella, "Studio sulla polinevrite tuberculare." *Annali della Clinica della mal. mente nervose,* Palermo, 1903, p. 1.

extremity the small muscles of the hand are most frequently attacked. Of the bulbar nerves occasionally the pneumogastric, the facial and the phrenic are implicated. Paralysis of the ocular muscles is not observed. The sphincters are, as a rule, intact. Subjective sensory disturbances are extraordinarily frequent, nor are those of an objective nature often absent (Francotte, Pal). The tendon reflexes are seldom produced. Colella expressly states that in his cases, as in other forms of polyneuritis, the patella reflex was absent or was so feeble that after 5 to 7 taps it ceased. In a few instances the patella reflexes were increased (Lesage, Décroly). Vasomotor, secretory, and trophic disturbances were frequent (cyanosis of the hands, edema, sweating, changes in the nails, etc.). The onset was usually chronic; only in very rare cases of pure tuberculous origin was it acute. This exclusive tuberculous etiology was present only in the minority of the cases; in the majority, as Remak emphasizes, there was also alcoholism. It must be remembered that with a very insidious development of tuberculosis, polyneuritis occurs as a late complication. An excellent example of polyneuritis due to autointoxication is that form arising in *diabetes*. The following is a typical case:

CASE 27.—A man, aged 50, reported that for three years he had suffered from the symptoms of well developed diabetes (glycosuria of moderate grade, polydipsia, loss of weight, etc.). For a year there has been weakness of the fingers, which are constantly used in his occupation, and this condition has increased within the last three months, accompanied by numbness and pain in the legs. The gait has become halting and uncertain, with the legs wide apart, and the feet, particularly the external borders, droop. The nerves of the lower extremity are distinctly painful to pressure. The sciatic phenomenon is well developed bilaterally; both patella reflexes are very indistinct; the Achilles tendon reflexes are absent. There is decided weakness of the muscles supplied by the peroneal nerve, more so in the peroneus than in the tibialis anticus. Partial DeR can be electrically demonstrated in the peroneal muscles. Also, there are well developed contact anesthesia, painful irritation and changes of position in the peroneal region; even in the recumbent posture a certain degree of ataxia can be determined. There is distinct swaying on closure of the eyes. The radial and ulnar nerves are somewhat sensitive to pressure, the interossei are markedly emaciated; the ball of the thumb is flattened, and there is typical claw position of the left hand. Only slight power is furnished by the small muscles of the hand. A coarser sensory disturbance in the hands cannot be demonstrated. Here also there is partial DeR. The patient formerly drank a great deal of beer but has abstained from such beverages in recent years.

This is a typical picture of *amyotrophic polyneuritis*, combined with well marked diabetes mellitus to which we may undoubtedly attach the greatest importance in the development of the affection, as the previous alcoholism does not play an immediate rôle. Besides the conditions occurring in polyneuritis there are also other pathologic pictures due to diabetes which may be referred to an affection of the peripheral nervous system: primarily, true *neuralgias*, diabetic sciatica, and far more frequently than otherwise, neuralgia of the crural and obturator nerves. Further there is diabetic mononeuritis, and even polyneuritis which may assume various forms. The signs of trophic degenerative paralysis are not always so prominent as in the case just described. Sometimes there are only sensory, irritative phenomena and those due to loss of function, perhaps also absence of the tendon reflexes. The following case is an illustration of such a form, and also shows the fully developed symptom-complex of diabetes.

CASE 28.—The patient complains of *paresthesia* in the tips of the fingers, *lancinating pain*, and a *somewhat uncertain gait*. There are no urinary complications. *The Achilles tendon reflex is absent; the knee-jerk may be imperfectly produced with Jendrássik's grasp*. The patient sways upon closure of the eyes. The nerves and muscles of the legs are not sensitive to pressure; there is no atrophy, no paralysis; but numbness and distinct analgesia are present in the distal portions of the leg. There is no anesthesia of the trunk. The pupillary reaction, the eye-ground, and all other conditions relating to the nerves of special sense are normal.

At first glance there is a conspicuous similarity of this clinical picture with that of *tabes*. The differentiation of this *pseudotabes diabetica* from true tabes is often difficult, and sometimes impossible, especially if we remember that *melituria may be a symptom of tabes*. As distinguishing factors the absence of pupillary rigidity, of bladder disturbance, of anesthesia of the trunk, and perhaps also of crises in this so-called *pseudotabes diabetica*, may be mentioned, while perforating ulcer of the foot occurs also in diabetes. Finally, there may be a combination of tabes and diabetic neuritis. Of the greatest diagnostic importance is the fact that in diabetes the knee-jerk may reappear with an improvement in the general condition, and it may also be absent without the occurrence of other neuritic symptoms. Therapeutically, anti-diabetic diet, which has frequently produced good results, is first to be considered.

I have already referred to the forms of polyneuritis developing in the course of *syphilis*, as well as to *puerperal* and *parturitional neuritis*. *Gout* probably, as a rule, gives rise only to neuralgia, rarely to mononeuritis. Remak, Epstein, and Grube observed the latter condition in the course of the brachial nerves. True polyneuritis, as emphasized by Remak, is exceedingly rare. Occasionally polyneuritis occurs as the result of *cancerous cachexia*, such cases having been reported by Oppenheim, Miura, and Francotte. A case reported by Oberthür[1] requires especial mention. Here the symptom-picture of an atypical peripheral neuritis resulted from carcinosis of the peripheral nerve terminations and muscles, and not, as is usual, from a toxicant. Oppenheim[2] first called attention to *senile polyneuritis*, a form of neuritis occurring in the aged. In a patient over 70 years of age there were gradually increasing disturbances which implicated sensation and movement of the hands and feet. Pain was absent or slight, but paresthesia was very conspicuous. The paralysis attained a high grade. The cerebral nerves were not involved. These symptoms are not observed in all cases of senile neuritis. In the cases reported by Stein, pain and hyperesthesia were most prominent; exceptionally, bladder disturbance and paralysis of the cerebral nerves have also been noted. The course may be favorable, although the danger of arteriosclerosis must always be borne in mind—a condition which is of decisive importance in the development of neuritic changes. It must also be remembered that the inverse condition may result from arteriosclerosis due to degeneration of the nerves (Lapinsky)—a point which we shall consider under pathological anatomy. On the other hand, neuritic changes may produce not only senile arteriosclerosis but also other forms of this dis-

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[1] *Revue neurolog.*, 1902, p. 485.

[2] Oppenheim, "Ueber die senile Form der multiplen Neuritis." *Berliner klin. Wochenschrift*, 1895, p. 289.

ease. In the case of Schlesinger's there was an endarteritis obliterans of the vasa nervosum in a man aged 69; in a second case, a man aged 23, endarteritis obliterans and endarteritic gangrene occurred. Similar conditions have been reported by Joffroy, Achard, Dutil, and Lamy; and periarteritis nodosa was present in two cases of Kussmaul and Maier. In these patients positive neuritic symptoms were not always present. A case of polyneuritis due to endarteritis obliterans has recently been reported by Franceschi.[1]

Pathological Anatomy.—The *pathological anatomy of multiple neuritis* has for its object the demonstration of changes common to all the forms, besides showing the deviations in the numerous varieties of this disease. The most extensive material for investigation is contributed by *alcoholic polyneuritis,* from which we shall start, using the conditions for a general description. *Macroscopic* examination of the peripheral nervous system at the autopsy reveals nothing of note. Rarely can we observe swelling or redness of individual nerves—a hyperemia—and this most readily in the acute infectious cases. In leprous neuritis there are spindle-shaped or globular swellings of the nerve which may frequently be seen with the naked eye, and may also be palpated *intra vitam. Microscopically* we observe mainly atrophy of the nerve fibers. These were first determined by Thomson and Lancereaux. Moeli, Oppenheim, Siemerling, Déjérine, and others have since increased our knowledge of these conditions. The *atrophic* processes in the nerve fiber implicate the *axis cylinder* as well as the *medullary sheath.* They are of varying intensity: from the mildest grades of a reduction in the medullary sheath they lead to almost complete atrophy and destruction of this substance and of the axis cylinder. According to the acuity and the stage of the process, alterations in the destroyed medullary sheath will best be seen by the Müller osmium method of staining, which gives a black tinge; at other times Weigert's medullary stain or van Gieson's stain will produce better results. A 1 per cent. osmic acid solution is valuable to demonstrate these alterations. The histologic conditions do not differ from those degenerative processes described in *mononeuritis,* but the division of these changes is usually characteristic. As a rule, the *peripheral muscular branches* are most severely implicated; and here again, corresponding to the clinical symptoms, the *peroneal nerve* is largely affected. While, here and in the peripheral branches of the tibial nerve, in the large saphenous, and perhaps also in the radial and ulnar, the atrophy may be very marked, we find in the nerve trunks proximally situated numerous column fibers besides those that have degenerated. These differences have been frequently demonstrated in the same nerve and have also been found in the *cutaneous nerves,* often with decided intensity. That the peripheral branches are most severely implicated is a phenomenon peculiar to all forms of polyneuritis. Various explanations have been attempted. It has been assumed that, on account of being farthest from the trophic center, their constituents suffer most quickly from any nutritive damage; also that they furnish the best point of attack for toxic, infectious, or rheumatic damage.

In contrast to the *atrophic-parenchymatous* processes the changes in the

[1] Franceschi, "Polynevrite ed arteriosclerosi del sist. nerv. centr. e perif." *Riv. d. patol. nerv. e ment.,* 1903, 5.

connective tissue are secondary, particularly in neuritis due to alcohol and similar toxic agents, and only exceptionally do we find a marked increase of the perineurium (Oppenheim and Siemerling), which is then usually limited to a few nerves. There is also an increase of the nuclei of the connective tissue, and even small hemorrhages, changes of the vascular walls, and exudates around the vessels occur (Eichhorst, Flemming). Figure 5, Plate III, from a patient who succumbed to a polyneuritis in the chronic stage, shows these atrophic processes distinctly. Nowhere are there signs of a fresh inflammation. Besides numerous normal nerve fibers—the preparation, taken from the trunk of the sciatic, was stained according to Pal with van Gieson's stain—we find the greater number of the medullary fibers severely damaged, a portion of these fibers and their axis cylinders having disappeared; of the remainder a greater magnification shows the nerve sheaths to be incomplete, thin, pale, partially destroyed, and the axis cylinders also changed, unevenly thinned and thickened. Figures 3 and 4, previously described, show these conditions distinctly. The endoneural and perineural connective tissues are moderately increased. In some transverse sections a greater richness in nuclei is simulated by a decrease in the nerve bundle due to connective tissue contraction.

Changes are frequently found in the *muscles*. They need not be described as they are identical with those observed in mononeuritis. Their distribution corresponds to that of the neuritic changes and thus conforms with clinical observation. The parallelism between clinical and neuritic or neuromyositic phenomena is not always complete: For example, if paralysis of the diaphragm has been the cause of death the phrenic nerve may be found normal, although there had been sufficient time for the development of neuritic changes.

In paralysis due to alcohol the changes in the peripheral nervous system are the most prominent, but are not always solitary. The *spinal cord* is often implicated. In 43 cases Gudden found spinal changes 15 times. With the development of the more delicate methods of investigation this ratio has been increased. Nevertheless it must be distinctly emphasized that occasionally *spinal lesions cannot be demonstrated* even with the employment of Marchi's and Nissl's methods. In different cases very dissimilar changes may be determined. The *intramedullary anterior and posterior roots* are frequently degenerated, usually to a slight degree, this being best determined by the Marchi method, but sometimes atrophy is so pronounced that Weigert's medullary sheath staining is sufficient to demonstrate the change. On the other hand, the *extramedullary* roots often escape degeneration,[1] even when the intramedullary roots are damaged. The *posterior columns* of the spinal cord often show more or less severe degeneration, usually analogous to the changes of the posterior roots, while, less frequently, circumscribed inflammatory foci can be demonstrated. The remaining white substance is only exceptionally the seat of these changes (the cases of Cole).[2] The alterations which are found mainly in the anterior horn cells are usually slight, as a rule, showing changes of Nissl's bodies, or, more rarely, vacuole formation in the cells.

[1] Heilbronner, "Rückenmarksveränderungen bei der multiplen Neuritis der Trinker." *Monatsschr. f. Psych.*, IV, pp. 1 and 81 and others.

[2] Cole, "Central Changes in Alcohol Neuritis." *Brain*, 1902, p. 326.

The cells of the spinal ganglion (Cole, *l. c.*, Philippe and Eide),[1] and those of the corresponding cerebral ganglia (Burr)[2] have also been found changed. These are not all of the lesions found in the central nervous system. Bonhoeffer found them in the radiating fibers which enter the cortex of the cerebrum; Cole in the fronto-thalamic fibers, in Betz's giant pyramids, etc.; inflammatory lesions have been demonstrated in the brain trunk (*superior hemorrhagic polioencephalitis*). In the clinical description we observed that symptoms may appear which extend beyond the picture of neuritis (psychotic phenomena, paralysis of sight). A portion of the central changes may explain these signs, but it now appears certain that the majority of symptoms owe their origin mainly to alterations in the peripheral nervous system, which are the only constant ones; all others vary in intensity and extent and may be absent.

The pathologico-anatomical foundation of other forms of polyneuritis due to chronic poisoning from metals is less definite and clear than that of alcoholic neuritis. In *arsenical poisoning,* investigations conducted singly (Henschen, Erlicki, and Rybalkin) have shown degeneration in the radial and peroneal nerves, and also in the anterior horn cells and in Goll's columns. In *poisoning with mercury* there is no anatomical basis. Experimental investigations (Létulle and Heller), which have, however, been contradicted, would appear to confirm the clinical phenomena, in that they assume a neuritic foundation. CO_2 and *carbon bisulphid poisoning* associated with paralysis have not been satisfactorily explained by the pathological lesions. In *lead poisoning,* however, the lesions are better understood. Here the investigations at least permit a positive opinion in points of the greatest importance. As is clinically confirmed the changes in the *peripheral nerves* are usually most marked in the radial, but the previously mentioned atrophic changes are also found in other peripheral nerves. The peripheral branches are most severely injured but proximally the lesions rapidly decrease. Gombault and others have demonstrated, particularly in lead paralysis, a *segmental periaxial neuritis,* which has also been marked in other toxic and infectious forms. The *spinal cord,* as a rule, is intact but Oppenheim found changes in the anterior horns. Here the anterior horn cells as well as the fiber-work were atrophic together with interstitial changes, enlargement of the vessels and the appearance of spindle cells. Lesions of the anterior cells have also been determined by experimental researches (Stieglitz, Vulpian). In a case recently reported by Philippe and Gothard[3] which was anatomically investigated, the authors found a *subacute anterior poliomyelitis* as the cause of the extensive suturnine paralysis, in comparison with which the changes in the peripheral nerves were only secondary. In such cases which are, however, exceptional the lesions in the anterior horns cannot be regarded as secondary to those of the peripheral nerves, as was assumed by Warrington in his case. Here they evidently have a substantive importance and it appears that lead may seriously damage the peripheral, as well as the cerebral nervous system; this is further confirmed by our knowledge of encephalopathia saturnina. This

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[1] *Neurol. Centralbl.*, 1903, p. 92.
[2] *Revue neurolog.*, 1902, p. 507. ("Veränderungen im Ganglion Gasseri.")
[3] *Rev. neurolog.*, 1903, p. 117.

view *that various poisons do not constantly implicate a definite nervous area* may be generally accepted and particularly for the conditions observed in alcoholic polyneuritis, in which there have been spinal and cerebral changes. These may, therefore, be coördinate to the peripheral lesions. That in one or the other case they may be regarded partially as the *consequence of peripheral changes* (as a retrogressive degeneration, etc.) must be admitted; but this view is not generally accepted. Even the alteration observed in the *muscles* may not at once be regarded as dependent upon neuritic changes, for clinically (myopathia saturnina, neuromyositis) as well as anatomically they show a certain substantive position.

These views are applicable not only to toxic but also to other forms of infectious neuritis. Of the latter the *diphtheritic* variety has been most closely studied, and here the *changes in the peripheral nerves* are the most constant. These conform to the clinical symptoms and are earliest and most distinctly present in the nerves immediately adjoining the infectious focus: therefore in the nerves of the palate. The local effect of the poison may be noted in Kussmaul's case previously mentioned, in which after diphtheria of the umbilical cord there was first paralysis of the abdominal muscles. Trousseau also found that in cutaneous diphtheria, paralysis first appeared in the extremities. Naturally there are exceptions to this rule. Thus in Griesinger's case paresis of accommodation appeared after intestinal diphtheria, and in a patient of Oppenheim's, following cutaneous diphtheria, the same sequel was noted. Besides the neuritic symptoms occurring *in continuo* others appear in many spinal, and especially also in the cerebral, nerves which may naturally be referred to the general effect of a circulating poison.

The lesions may be very severe. The *terminal muscular* and *cutaneous branches* are sorely involved and the destruction of the parenchyma may be complete. Proximally the degeneration decreases in intensity. The *connective tissue* apparently more often takes part in the changes than in toxic neuritis. This is often macroscopically demonstrated by hyperemia and swelling of the nerves (Stewart), and even by decided nodular formation (in the nerves of the abdominal wall, the phrenic nerve, more rarely in those of the extremities). Microscopically there are swelling and proliferation of the fibers and especially of the nuclei of the connective tissue; besides an inflammatory exudate and the migration of red and white corpuscles. The signs of *true inflammation* are therefore distinct. Streptococci are found occasionally in the nerve sheaths (Moos, Buhl). In numerous cases lesions could also be demonstrated in the *central organs*, the importance of which is not easy to estimate. Katz refers to an accumulation of black granules in the ganglion cells (investigated with Marchi's method) but no pathologic importance is to be attributed to this finding. Déjérine found marked cellular alteration in the anterior horns as well as in the anterior roots. Other authors have confirmed this and further observed degeneration in the posterior roots and posterior columns. Myelitic foci have been present, also hemorrhages in the spinal cord and spinal ganglia, meningitic changes, and lesions in the cardiac ganglia. Severe alterations have also been observed in the *muscles*, exceptionally even as the only finding. Occasionally no lesions have been demonstrated.

The foregoing explanation serves to explain the different lesions. The

toxin of diphtheria finally implicates and damages all parts of the nervous system; those mediate as well as immediate (by fever, cachexia, secondary infections, sepsis, etc.) *but the peripheral nervous system apparently is first and most severely involved.* The experimental investigations (Babonneix [1] is the latest observer in this direction) lead to different results, showing a variability in the point of attack, but prove that the peripheral nervous system is the one most seriously damaged. In other forms of infectious neuritis there is by no means so great a collection of anatomical material. Some few cases of *post-typhoid polyneuritis* show the severest changes in the nerves of the lumbar plexus. Typhoid bacilli have been found in the nerve sheaths. In *puerperal polyneuritis* (Korsakow and Serbski, Pribytkow, Mader)[2] there is primarily parenchymatous degeneration and in part also periaxial neuritis. *Malarial polyneuritis* has been anatomically investigated in a few instances (Luzzato [3] and others); in a case of Eichhorst's, which must be included, there was principally an interstitial neuritis, likewise severe parenchymatous degeneration, which could also be partially demonstrated in the central organ. In a case of polyneuritis after *follicular tonsillitis* Kast found widely distributed parenchymatous and interstitial changes.

Two forms of polyneuritis of well defined anatomical type require special description, although as I previously emphasized, with us they are rare affections. The first is *beri-beri.* In this disease we find *severe parenchymatous* and *interstitial lesions,* of which the latter, especially in chronic cases, may be very decided. There may be enormous proliferation, and thickening of the epineurium and perineurium; the individual fiber bundles then being separated from one another by broad connective tissue bands. The endoneural tissue is not markedly increased and a nuclear proliferation of the interstitial tissue can be demonstrated only in different areas to a limited extent. In a case reported by Rumpf and Luce, there was in particular a distinct increase of the epineural fatty tissue, so that these authors refer directly to a *chronic interstitial lipomatous neuritis.* Besides, there is quite a decided disappearance of medullary fibers. In this variety of polyneuritis, as well as elsewhere, peculiar structures known as Renaut's bodies have been seen. These consist of connective tissue bands, sometimes resembling onion peels, layered one above another, containing numerous large nuclei. Centrally in most of these bodies, there is a more or less well retained nerve fiber, usually with a destroyed medullary sheath. These structures apparently originate from the epineurium. Some writers believe that they come from the vessels. They do not appear to characterize any special form of the disease (Oppenheim and Siemerling, Pick, Nawratzki, Küstermann, Okada, Westphal). While in *beri-beri* the *spinal cord* usually shows only insignificant lesions, which are found in other conditions, there are almost always severe changes in the *muscles,* which may also be demonstrated clinically. In cases of acute onset there is a parenchymatous, fatty, occasionally colloid, degeneration of the muscle fibers; atrophic and hypertrophic fibers are found, the muscle nuclei are increased; in the chronic forms besides the atrophy of the fibers there is a profuse interfibrillary development of connective tissue.

[1] Babonneix, *Arch. général. d. méd.*, 1903, p. 1201.
[2] See Lugaro, "Handb. d. pathol. Anat. d. Nervensystems," II, p. 1140.
[3] Luzzato, *Berlin. klin. Wochenschr.*, 1902.

The lesions in *leprous polyneuritis* have much in common with those of beri-beri, but others again deviate greatly. Here alterations of an inflammatory nature occur alongside of those which must be regarded as proliferation, and it is not easy to differentiate between these varieties of pathologic change. As may readily be seen (compare Figures 6–8, plates III and IV) the interstitial tissue is here also markedly implicated; in part there is a formation of small nodules which may be macroscopically recognized and palpated and which are of well known diagnostic importance. The interstitial changes involve especially the *epineurium* and *perineurium*, the endoneurium being less affected. Besides the *proliferation of the connective tissue* there is *small cell infiltration* which I believe does not always indicate the preliminary stage of connective tissue hyperplasia. On the contrary it may completely characterize a *small cell granulation tumor*. The small cell infiltrations may reach an extraordinary degree, as is seen by the illustration. The cell infiltrate is situated partly external to the nerve fiber bundle, and in the connective tissue structures between the perineurium and epineurium; such a condition is seen in Fig. 6, marked *a*. Further infiltrates force themselves between the dense or fine strands of the nerve fiber bundle, following the fibers of the endoneurium. In Fig. 7, showing greater magnification, which like Fig. 6, represents a preparation stained according to van Gieson, these finer relations are shown distinctly. Besides these changes and the frequently observed *thickening of the vessels*, there are also *alterations of the nerve fibers* themselves which lead to their destruction in the manner frequently mentioned. How great this may be is seen in Fig. 8, a preparation stained according to van Gieson, showing the scant nerve fibers which are still retained (Nf). It is here to be noted that the interstitial connective tissue increase of the epineurium and perineurium is somewhat less marked, although the previously described distinct small cell infiltrations are clearly recognizable at different points (*a*). In Fig. 7, the condition of the nerve fibers can be somewhat more closely studied, in contrast with the small cell infiltration and connective tissue increase. The specimen was from a nerve with moderately severe lesion in which some of the nerve fibers (Nf) are yet intact. In leprous polyneuritis Renaut's bodies are also found.

A phenomenon, having general importance in the pathology of neuritis, observed by earlier investigators, may be distinctly demonstrated in these preparations. This is the *discontinuity of the lesions*. If I had examined above and below the points of greatest change, from which my illustrations were taken, but very slight alteration would be found and only at distant points of the same nerves are renewed severe changes of the same character demonstrated. For example, between two diseased foci of the right ulnar nerve at the elbow and wrist a large portion of the nerve showed but very few changes and peripherally from the wrist-joint better retained portions could be found. Thus Fig. 7 was taken from a peripheral branch of the right ulnar nerve in the hand, while Fig. 6 shows a portion of the same nerve from immediately above the wrist-joint. Nonne[1] demonstrated similar conditions. It is almost always possible to distinguish the *bacilli of leprosy* in the peripheral nerve trunks, although their number is usually small. In my case

[1] Nonne, "Ueber Lepra tuberosa." *Neurol. Centralbl.*, 1892, p. 454.

I was able to find bacilli in those portions shown in illustrations 6 and 7. They have been demonstrated even in the ganglion cells of the spinal ganglia and in the anterior horns of the spinal cord.

The changes of the *central organs* are usually not very marked. They have been found in the posterior roots and posterior columns; but all spinal alterations may be absent, as in my case.

Of the forms of polyneuritis developing from *dyscrasias* those of a *tubercular* origin have been most frequently investigated. Here interstitial and parenchymatous changes have been noted (Oppenheim and Siemerling). Pryce and Lapinsky found in diabetic paralysis degeneration of the nerve fibers. The same is true of paralysis due to carcinomatosis, inanition, etc. In cachectic individuals degenerative and atrophic changes have been found in the nerve fibers, often without previous clinical symptoms. These conditions were regarded as latent polyneuritis. The knowledge of this fact is important; it cautions us not to develop relations between clinical and anatomical findings too rapidly; it forces us to appreciate a phenomenon with which we constantly meet in clinical descriptions; namely, that in such individuals an insignificant damage, a brief pressure or a mild infection, is sufficient to produce a neuritis. The lesions in senile polyneuritis, particularly as regards the peripheral nerves, do not deviate from those that have been described. Alongside of these we find signs of *arteriosclerosis* in the vessels. The question how vascular changes are to be regarded in polyneuritis can, however, not always be satisfactorily answered. While in cases of senile polyneuritis, and also in those originating from an endarteritis obliterans, there can be no doubt of the causal connection of the vascular changes, in some instances at least these appear to be conjoined with neuritic ones. In other cases the question may well be discussed whether the vascular changes have not arisen secondarily in consequence of neuritis (neurotrophic changes of the vessels).

It was previously stated that *bacilli* have been discovered in the diseased nerves. Apart from leprous neuritis observations of this nature are very scant, but Glogner, Wochenius, Centanni, Eisenlohr, Moos and Bunl have reported such findings. In these cases the microorganisms were by no means identical, for the most dissimilar varieties were observed (staphylococcus pyogenes aureus, staphylococcus pyogenes albus and a special bacillus by Centanni). From this it may be concluded that not the bacilli themselves but their *products of metabolism* are responsible for the damage to the nerves. The negative finding in numerous other cases of polyneuritis as well as the fact that many other poisons introduced into or present in the body cause similar phenomena, is operative in the same sense. Thus the view that *neuritic changes are due to toxemia* is very prevalent. That the toxins which are produced may give rise to such diverse clinical pictures, these being so characteristic in their individual factors, as is the case for instance with diphtheria-toxin, is readily understood on account of the *specific affinity of the poison,* a fact that has been demonstrated by numerous observations. Experimental investigation, to a great extent, is calculated to favor this view.

In a comprehensive review of the anatomical lesions in polyneuritis as the primary and special *seat of the changes, the peripheral portions of mixed nerves* must be recognized. We have seen that in general the further we ad-

vance from the peripheral region of distribution toward the central organ, the less marked the changes become, without, however, the decrease in the process being uniform. On the contrary, it appears that special central, spinal or bulbar, areas are implicated with a certain degree of regularity. These are the intramedullary anterior and posterior roots, the anterior horn cells and the continuation of the posterior roots, the posterior columns. We must not, in order to advance a particular theory, regard the entire peripheral sensory and motor "neuron" as uniformly diseased; the assumption of such a "neuronitis"[1] by the enthusiastic adherents of the neuron theory does not find sufficient support in the anatomical lesions.

The motor nerve divisions, from the cell in the anterior horn to the terminal distribution in the muscle and in fact even this structure itself, on the one hand is most affected—and on the other hand, the sensory nerve portions from the periphery to the spinal ganglion cell and thence to the spinal cord. From this localization it follows, necessarily, that the differentiation of *polyneuritis* from two diseases, *poliomyelitis* and *tabes*, must be exceedingly difficult. The former is a malady which especially involves the first named motor, the latter a disease which most severely damages the last named sensory, division. The differential factors show, in the first place, that in polyneuritis motor and sensory nerves are simultaneously attacked (we have seen that there are exceptions to this rule) and secondly the differential points may be deduced from the varying nature of the disease.

The following points *differentiate* the affection from *poliomyelitis*: Polyneuritis develops in immediate connection with an infectious disease, or the affection in its first stages bears such a character, while in poliomyelitis these general phenomena are usually slight, and even when they are more prominent they are of brief duration and do not play an essential part. That polyneuritis is more frequent in adults is true, but as acute poliomyelitis also occurs in adults this aids but little in the decision. The development of nervous symptoms in polyneuritis is mostly gradual. As a rule sensory irritative symptoms precede and the paralysis follows in the manner previously described. In the spinal affection paralysis develops rapidly, showing its maximum extent within a few days and gradually ameliorates. Nor do relapses occur in the further course as is sometimes the case in polyneuritis. There is, however, one form of neuritis in which all symptoms, particularly the paralysis, appear suddenly. This is a malady designated *neuritis apoplectica* and described by Déjérine, Remak, Westphal and others. It is usually not a true polyneuritis but a plexus neuritis. I have seen two cases of this kind, in both of which there was partial plexus paralysis. The differentiation from polyneuritis may give rise to great difficulty as the differential factor soon to be described, particularly in these cases, often leaves us in the lurch. In my cases the sensory, irritative and paralytic symptoms, were the ones which made the diagnosis of neuritis possible. The *nature of the paralysis*, as well as the localization, quite apart from the difference in development just mentioned, is not the same. In polyneuritis the distal portions are most severely damaged. Only very rarely does complete paralysis implicate extensive muscle areas. In the early stages of poliomyelitis total paralysis may in-

[1] Teitelbaum refers directly to neuronites motrices inférieures.

volve both legs and even all four extremities. If in the further course the paralysis is limited to a circumscribed region the arrangement corresponds to the *spinal type*. It is often *asymmetric* and does not so exclusively affect the distal muscle groups. The involvement of *cerebral* nerves, which is not rare in severe polyneuritis, is exceptional in poliomyelitis; perhaps most often disease of the facial nerve takes place, due to implication of a poliomyelitis bulbi. On the other hand paralyses of the ocular muscles only rarely occur in acute poliomyelitis but they are sometimes seen in the rare malady known as polioencephalomyelitis. Upon minute examination we also discern differences in the *electric contractility*. There exists here between the degree of paralysis and the degree of alteration in the electric contractility by no means such a parallelism as in poliomyelitis. Severe paralyses may only present slight DeR or even only quantitative diminution. In areas that are only slightly paretic, conspicuous alterations of electric contractility may be present.[1] The same is true of muscular atrophy; here also differences between function and nutrition are the rule in polyneuritis while they are absent in poliomyelitis.

Fibrillary contractions are mentioned as differential factors, as they are said only to occur in poliomyelitis. According to my experience, however, in the most common form of poliomyelitis, acute infantile paralysis, they play no part, while in the subacute forms of adults they are more common and they do occur in polyneuritis.

Among the non-motor disturbances the most important diagnostically in polyneuritis are the *sensory irritative symptoms and those due to absence of function*. *Pain* is not necessarily absent in the first stage of poliomyelitis. In a case reported by Strümpell and Bartelmes, there were severe sacral pains for three weeks. In my experience this is not even rare in children, at least this symptom has been reported frequently enough.[2] But upon the whole the pain is of a different character in polyneuritis; it radiates to the distal regions often following the course of the nerves, while in poliomyelitis the pain is mostly localized around the focus of the disease. Marked *pain upon pressure* of the nerves and muscles, is the case in polyneuritis, in which there may also be only *paresthesia* and finally *objective sensory disturbances*. [The principal differences in regard to the sensory symptoms as between poliomyelitis and polyneuritis seem to be that in poliomyelitis there is diffuse tenderness over the limbs and along the spine. The pains are rarely neuralgic but commonly steady and of medium intensity. The skin is sometimes superficially sensitive. There is no particular sensitiveness of palpable nerve trunks. The pain and tenderness come on immediately with the onset of other symptoms, and usually subside within a week. In polyneuritis the sensory disturbances are almost invariably initiated by paresthesiæ, such as creeping, crawling numbness, tingling, burning, etc., and almost invariably in the terminal distribution of the nerves and in symmetrical areas. These hyperesthesias are not transient but are characterized by their constancy, and such pains as occur are distinctly neuralgic. Tenderness of muscle masses and

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[1] See Raymond, "Polynévrite et Poliomyélite. Nouv. Iconogr. de la Salpêtrière," 1899, p. 1.

[2] See Oppenheim, "Lehrbuch," p. 215.

of nerve trunks is pronounced, and when these sensory disturbances are once established they tend to persist for weeks, undergoing modifications that parallel the condition of degeneration or regeneration in the nerves that are affected.—ED.] Some symptoms are entirely missing in poliomyelitis. Among these is the *ataxia* which we have learned to recognize as a very prominent symptom in polyneuritis. Nor does optic atrophy occur in poliomyelitis nor is there a symptom-picture which corresponds to the "polyneuritic psychosis." The trophic signs, in general, are somewhat different in polyneuritis than in poliomyelitis. Glossy skin particularly is not seen in poliomyelitis, in which there is extensive atrophy, cyanosis and coldness, rarely true hyperplasia of the fatty tissue, or much edema. The differentiation of these two diseases, to which so much labor has been devoted, is very important as the *prognosis* in each is very different. Finally if doubt still exists, we are able to form an opinion from the termination of the affection. *Poliomyelitis never recovers entirely.* Improvement, which regularly occurs at the onset, finally halts; a stationary pronounced limited paralysis remains, a condition which is exceedingly rare in polyneuritis. In polyneuritis improvement takes place later, it is slower, but finally leads to almost complete recovery; and even where paresis remains there is not such complete absence of function of the affected muscles and such high-grade atrophy as in poliomyelitis. While we may assume that after 9–12 months there is a stationary condition in poliomyelitis, improvement may even occur in polyneuritis after this period.

For the classical form of polyneuritis these points of support will mostly suffice but some atypical cases may give rise to considerable difficulty. *Neuritis apoplectica* has already been mentioned. Further there are cases with a deviating type of localization as well as with marked decrease of sensory symptoms. Thus in the following case the diagnosis was in doubt for some time.

CASE 29.—A laborer's wife, aged 56, does not use alcohol and has never been seriously ill. Early in October she was attacked with very severe pain which had its main seat in the region of the left shoulder, radiating to the back and arms. With this there was formication and numbness in the left arm and hand. Fever was the only constitutional symptom mentioned by the patient and even in regard to this her reports were meagre.

Examination middle of November, 1904, reveals passive limitation in movement of the left shoulder joint, marked sensitiveness on pressure of the brachial plexus, of the ulnar, median, and radial nerves. The pressure produces radiating pain. The triceps reflex is absent but the supinator reflex is marked. Of the muscles of the left upper extremity Erb's muscles are normal in the main, only the deltoid is implicated in its activity by disease of the shoulder joint. All other muscles are more or less paretic, the debility increasing distally, being therefore most marked in the small muscles of the hand.

Electric contractility in the paretic and atrophic muscles shows at times severe at other times mild disturbance. A positive, objective sensory lesion was not present; the continuous paresthesias were mainly referred to the ulnar region. There were no oculo-pupillary symptoms, none on the part of the legs, bladder, or cerebral nerves. Continued observation of the case showed constant improvement.

At first the pain as well as the sensitiveness to pressure disappeared. The movements in the shoulder joint, passively, soon became free. The paralysis of the extensors and flexors of the hands recovered completely but in the long flexors of the fingers there was still (March, 1905) a slight degree of weakness; severe disturbance and

marked atrophy are still present in the small muscles of the hand. Here there is also great change in electric contractility. Decided coolness or cyanosis is not present; the tendon reflexes have reappeared.

In some respects this case differs from ordinary polyneuritis. From its localization, a spinal process involving the 7th and 8th cervical and first dorsal segments might be considered. The complete absence of objective, sensory symptoms, at least when the patient was first examined, is remarkable. The points, however, which favored a polyneuritis were the *early, marked, sensory, irritative symptoms, their prolongation, the pain on pressure over the nerves and muscles* and finally the course of the disease which tended toward complete recovery. We, therefore, regard the case as an *amyotrophic plexus neuritis,* the elucidation of which we owe to Remak.

Similar cases have lately been reported by different authors. A position midway between polyneuritis and poliomyelitis has been assigned to them, the affection being regarded as a neuron disease (Barnes,[1] Williamson,[2] Brissaud et Bauer).[3] That polyneuritis may, however, occur without sensory symptoms and without minutely corresponding to the localization and distribution of the peripheral nerves, is shown by *lead paralysis,* so that for the first mentioned variety there is no decisive reason for separating it from polyneuritis; at all events before a final classification is attempted sufficient anatomical evidence must be produced.

Tabes is the second disease which must be considered in a differentiodiagnostic respect. The decisive factor here is the development of the malady, for recent investigations have shown that no single symptom is pathognomonic. Tabes is a *chronic,* polyneuritis an *acute,* disease. Naturally there are exceptions from both points of view: tabes with acute development of the main symptoms; polyneuritis with slow progression. The *etiology* deserves consideration. Polyneuritis is favored by the demonstration of a toxic or infectious foundation; but naturally a person saturated with lead or alcohol may acquire tabes.

In cases of multiple neuritis in which *ataxia* and *sensory disturbances* are very prominent and in contrast the paralytic phenomena are less marked, the diagnosis is very difficult. These are the cases of *neurotabes peripherica* (*alcoholica, diabetica, diphtherica,* etc.). They have already been referred to and their differentiation from tabes has been described. Marked sensitiveness to pressure of the nerves and muscles favors polyneuritis or at least a polyneuritic component. Bladder symptoms, in polyneuritis, are slight, less intense and less continuous.

Reflex pupillary rigidity in the form of arrested reaction to light with retained convergence may be regarded the most important sign, especially if it be permanent, in which case tabes is extremely likely. Transitory alteration of the pupillary reflexes, especially a combination of disturbance of the light reflex with that of accommodation and convergence, are not of equal importance. Crises do not appear to occur in polyneuritis, nor do arthropathies

[1] Barnes, "Toxic Degeneration of the Lower Neurons simulating Peripheral Neuritis." *Brain,* 1902, page 479.

[2] Williamson, *Brain,* 1903, page 206.

[3] Brissaud et Bauer, "Poliomyélite antér. subaigue ou polynévrite motrice." *Rev. neurol.,* 1904, p. 1226.

and other trophic changes. Perforating ulcer of the foot has been observed in alcoholic polyneuritis. Korsakow's psychosis does not belong to tabes but in tabo-paresis similar psychopathic conditions may be seen. Steady progression naturally favors a spinal disease.

There is a form of tabes designated *acute spinal ataxia* which develops from disseminated myeloencephalitis. The acute development of ataxia, as well as the intimate association with an infectious disease, may closely simulate polyneuritic ataxia; but all other neuritic symptoms, pain upon pressure of the nerve trunks, sensory symptoms, and paralyses of peripheral character, are absent. For the most part cerebral and cerebellar symptoms are present, so that the differential diagnosis does not actually cause serious perplexity.

In the symptomatology I referred to a malady called by Senator, *neuromyositis*. Its main features are the early and marked prominence of myopathic symptoms (great pain on pressure of the muscles, edema of the muscles and perhaps of the skin, myopathic paralysis). The differentiation of neuromyositis from neuritis, as is obvious, is not always possible, nor has this any great scientific or practical value.

Landry's Paralysis.—It still remains to discuss the relation of polyneuritis to *Landry's paralysis* and to describe the differential factors. Here we meet with difficulties as the uniformity and substantive character of Landry's paralysis have, by no means, been solved. Landry gives the following description of the symptom-complex: In persons previously healthy there develops, after mild prodromes, subjective phenomena such as general malaise, paresthesia in the legs, etc., a flaccid paralysis of the legs, usually first in one, but rapidly, in the course of a few hours or a day, also in the other leg. After complete paralysis of the legs, the muscles of the trunk are involved and in a few days also those of the arms. Following this, the muscles of deglutition, articulation, and respiration are implicated, and with symptoms of suffocation, in a few days or in from one to two weeks, the lethal termination occurs. In only a very few cases does improvement take place in the manner that the muscles last paralyzed first regain their function. Mild sensory symptoms occur but muscular atrophy and alterations of electric contractility are absent. Autopsy findings have so far been negative. The sequence of the paralytic involvement is reversed under rare circumstances, so that bulbar symptoms are the first, followed by an implication of the arms and legs.[1]

Recent experience has familiarized us with a number of cases which, in the main, correspond to the clinical picture just sketched and thus permit of a somewhat accurate *clinical* diagnosis. On the other hand cases have been reported which deviate in one point or another and these changes concern symptoms which were supposed at first to be absolutely necessary to a diagnosis.

In fact, finally, but one characteristic remained: the *rapid* ascending, or more rarely descending, paralysis involving the entire muscular system, appearing to be the consequence of an infectious or toxic cause. That uncertainty was bound to appear in a diagnosis which seemed to be founded upon the acuteness or the intensity of the pathologic process is obvious, so that

[1] Oppenheim, "Lehrbuch," p. 553.

subdivisions and differentiations were often attempted without, however, clearing the situation.

The *course* of the later reported cases revealed that they were usually *afebrile*. The paralysis began in the toes and proceeded upward rapidly, so that in the course of a few days there was total muscular paralysis. The bulbar muscles were attacked usually even before complete paralysis of the extremities. Difficulty in deglutition, as well as in coughing, in speech, and in respiration appeared. Attacks of suffocation, due to paresis of the diaphragm, were the cause of death. The entire disease may last 2 or 3 days but often this was somewhat longer up to 2 or 3 weeks; with a bulbar onset the lethal termination has been reported after 29 hours. Electric examination in some of the classic cases reveals normal conditions. If the course of the disease is very rapid the explanation may be that even with the seat of the lesion in the peripheral nervous system there has been no time for the occurrence of electric changes (as well as of atrophy). But there are cases in which this explanation is not sufficient, in which, in spite of a prolonged duration of the malady, the electric reactions remain normal. On the other hand there are quite typical cases in which quantitative and qualitative electric changes are observed. In how far these differences are connected with the localization of the process will soon occupy our attention.

Pain is absent or at least not very pronounced, and then appears only upon pressure or passive movements; but there are also cases in which pain is not so insignificant. Among the objective, sensory signs slight hypesthesia is not rare; slowing of sensory conduction is also noted. Severe bladder symptoms are absent, although there are exceptions to this rule. While bulbar symptoms are frequent, paralysis of the ocular muscles is very exceptional. The mind remains clear and only under the influence of high, septic fever may unconsciousness occur.

These cases give us the impression that we are concerned with a *severe infection or intoxication.* Enlargement of the spleen and tumid glands often point to such an explanation. Accurate investigation has shown that the disease may follow in the course of all possible infections and intoxications: diphtheria, enteric fever, variola, influenza, tuberculosis, sepsis, anthrax and many others. One point is certain namely, that there is no *one* definite virus which produces the disease; this is also in consonance with the fact that in a number of cases bacilli have been found in the blood, in the peripheral nervous system, and that these microorganisms were by no means of a uniform character.

The pathologico-anatomical lesions are just as varied as the *etiologic* factors. There are cases in which nothing essential has been found although the investigations have been conducted with the most improved methods. Buzzard[1] only recently found insignificant changes. There are cases in which *spinal* and *bulbar* lesions, in the form of disseminated foci, have been demonstrated, and finally there is another group in which lesions were found exclusively, or at least principally, in the peripheral nervous system, closely resembling those of severe polyneuritis.

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[1] Buzzard, "On the Pathology and Bacteriology of Landry's Paralysis." *Brain*, 1904, page 94.

Thus it is seen how difficult it is to gain a firm foot-hold upon such an unsteady foundation. The cases in which there are severe pain, marked objective, sensory disturbance, decided changes of electric contractility, closely resemble the severest types of polyneuritis, and I do not believe it possible to differentiate these diseases from the rapidity of their course. The acuteness of a process is not a sufficient differential factor in pathology. Nor does the impression of a severe intoxication, which certainly exists, serve to characterize the disease more definitely. On the other hand, it must be noted that by no means in all cases of Landry's paralysis is it correct to include them under the group of polyneuritis; on the contrary there are cases which might more properly be included as *acute disseminated myeloencephalitis.* Among and between these there are again cases in which in one respect or the other the clinical picture is not sufficiently defined, and finally again such in which the intoxication appears to overwhelm and the nervous symptoms appear unimportant as may well be the case in any other intoxication, such as scarlatina, etc. It is, therefore, as yet impossible to systematize this group of cases. Common to all are the severity of the infection or intoxication and the rapidity of the resulting course, but the special etiology is as varying as the special pathology, and even the symptomatology, although apparently well defined and producing the appearance of clinical unity, is by no means always conclusive.

Treatment.—The management of polyneuritis, in the main, is identical with that of neuritis and I may, therefore, refer to that division of the subject. The *prophylaxis* consists in the avoidance and prevention of the many infections and intoxications on account of their intimate connection with polyneuritis. In this respect the advice of the physician is but little heeded. The prevention of occupation poisoning, especially of lead, depends upon general and individual measures and scrupulous cleanliness is the first consideration.

As *alcoholic* polyneuritis is mainly due to chronic and immoderate abuse of spirits, while beer is much less harmful, the avoidance of alcoholic beverages will decidedly limit this form of polyneuritis. To prevent the arsenical form, among other precautions the physician must observe care in the medical employment of arsenic.

We are hardly in a position to maintain a proper prophylaxis of the *infectious* varieties of polyneuritis. We should see to it that the patient does not over-exert himself and that he have a sufficiently long period of rest after an infectious disease, and prevent inanition and cachexia. In cachectic, dyscrasic polyneuritis the prophylaxis is the same as the treatment. In the rare form of syphilitic polyneuritis mercury, as previously stated, is sometimes contra-indicated; in malarial neuritis quinin is of great benefit.

After the disease has become manifest *absolute rest in bed* is imperative especially for the severe cases, so soon as the neuritic symptoms advance beyond the lower extremities. A bed-pan must be used by the patient; he should be fed and not permitted to alter his position without aid. This absolute rest may only be modified when there is an obvious improvement in the paralysis. To avoid *deformities* certain measures should be instituted at the onset: foot-drop is hindered by properly placed sandbags, and the pressure of the bed clothes can be removed by a wire hoop.

EXPLANATION OF THE COLORED PLATES

FIG. 1.—Sciatic nerve of a rabbit, peripheral portion; 10 days after severing the trunk. Marchi preparation.

FIG. 2.—Nerve cicatrix from the brachial plexus after an incised wound. Van Gieson-alum-hematoxylin preparation. Zeiss ocular 2, obj. AA.

FIG. 3.—Ulnar nerve in parenchymatous neuritis. Osmic acid preparation stained after van Gieson. Ocular 2, obj. AA.

FIG. 4.—The same. Ocular 2, obj. DD.

FIG. 5.—Sciatic nerve in acute polyneuritis. Weigert-Pal preparation stained after van Gieson. Ocular 2, obj. AA.

FIG. 6.—Ulnar nerve iu polyneuritis leprosa. Van Gieson-alum-hematoxylin preparation. Ocular 2, obj. AA.

FIG. 7.—Ulnar nerve in polyneuritis leprosa; different view than Fig. 6. Same stain. Ocular 2, obj. DD.

FIG. 8.—Ulnar nerve in polyneuritis leprosa; the same field as Fig. 6. Weigert-Pal preparation with van Gieson stain. Ocular 2, obj. AA.

PLATE II

Fig. 1

Fig. 2

PLATE III

Fig. 3

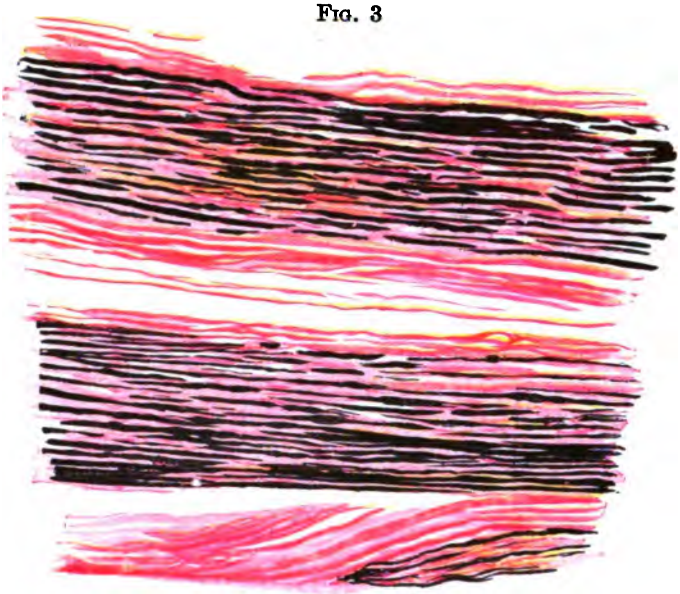

Fig. 4

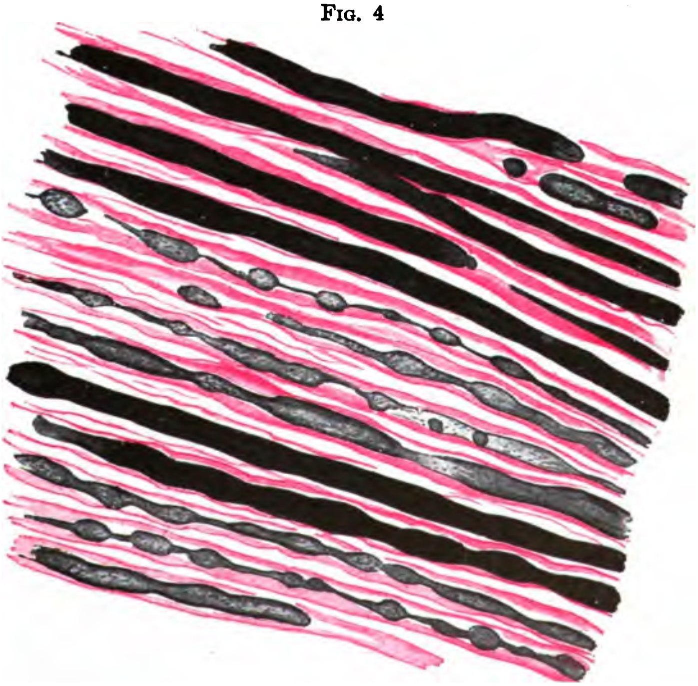

PLATE IV

Fig. 5

Fig. 6

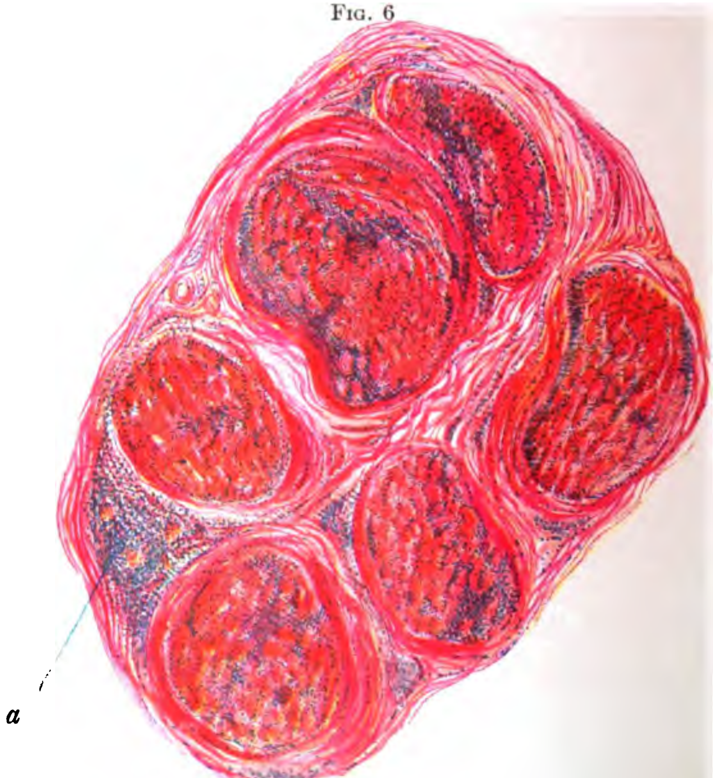

PLATE V

FIG. 7

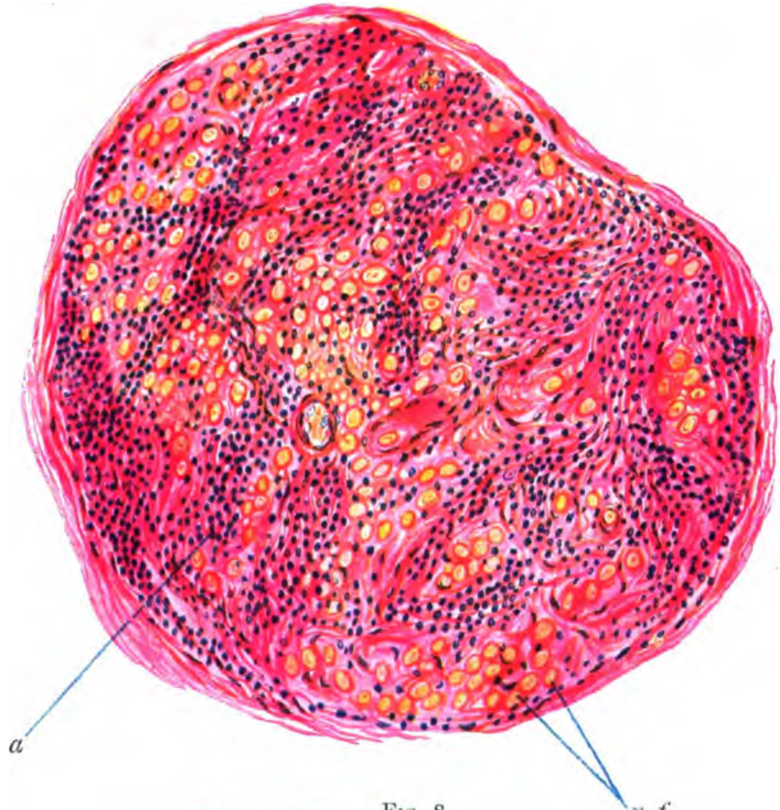

FIG. 8

The *diet* is very important. When at all possible it should be plentiful and very nutritious, which in the early stages, especially of infectious polyneuritis is not always possible. Milk is advised by Edinger,[1] as well as butter, bacon, lipanin, in fact a diet rich in fat. Sometimes nourishment by means of the stomach tube may be necessary as in diphtheritic deglutition-paralysis. In the diabetic form, a diet to combat the underlying malady often gives marvelous results.

Oppenheim also advises *diaphoresis* in polyneuritis the same as in neuritis. In robust patients hot baths should be used; in others hot packs, or heated air with hot drinks, and perhaps by the administration of diphoretics such as aspirin. Such diaphoresis may be employed daily for from 1–2 hours. Even in severe and chronic cases Oppenheim has obtained brilliant results by these means. Naturally watchfulness, in this method of treatment is necessary. It is supposed to remove, by excretion, the toxic products formed or introduced into the organism, in a speedy manner. We possess no specific for the disease but there is sufficient opportunity for the physician in the relief of the special symptoms.

The *heart* often requires attention but alcohol had better not be used; when stimulants become necessary, camphor or caffein may be administered; also sinapisms and faradization of the cardiac region, etc. *Respiratory difficulties*, which may directly endanger life, require artificial respiration, faradization of the phrenic nerve and cutaneous irritants. Raymond and Leyden have particularly advised strychnin for this purpose. *Difficulty in deglutition* may necessitate artificial nutrition.

Pain may be relieved by some of the newer analgesics, and on account of their great variety it will be possible to dispense with morphin. Hot compresses or Priessnitz's packs are useful.

Paralysis should not be treated during the early course of the disease. Only after the irritative signs have disappeared should electricity be used, as well as mechanical therapy. In regard to the special indications I may refer to what was stated under mononeuritis. In the later stages besides electricity and massage, exercises are important, but overexertion must always be prevented. Spa treatment may be advised with advantage. The sodium chlorid springs and peat baths (Baden-Baden, Wiesbaden, Nauheim, Wildbad, Ragaz, Teplitz, Marienbad, etc.) are useful.

Convalescence, which is prolonged, is often hastened by climatic treatment; a warm climate, perhaps even the sea air is strongly advised by Edinger. But here caution is necessary, the patient must avoid all rheumatic influences as the danger of relapse is not remote.

In cases in which complete cure does not occur, orthopedic or surgical treatment must be used, the necessity for which has been previously detailed.

The treatment of polyneuritis is one of the most gratifying in the entire realm of neurology; with a careful and attentive choice of remedies it brings about good results even in instances in which the affection is a very serious one.

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[1] "Behandlung der Polyneuritis." *Penzoldt-Stintzing's Handbuch*, Bd. V, p. 679.

NEURALGIA

By H. EICHHORST, Zürich

In the description of neuralgia, we shall utilize the clinical history of four patients in whom a diagnosis of neuralgia has been made. If a layman were to draw from this the conclusion that the subject of our present consideration (neuralgia) presents a uniform pathological picture (since the name certainly implies that all were suffering from the same malady), undoubtedly we should object strongly to so erroneous a conclusion. In fact, each of these four patients presented a group of highly interesting and peculiar symptoms. In the course of our description we will find abundant opportunity to revert to this.

In the great majority of cases the *recognition of neuralgia* causes the physician no great perplexity, and even a tyro in medicine can usually diagnosticate it without difficulty. Let us now consider what manifestations of the disease were particularly prominent in our four cases, so that we may be absolutely convinced of the correctness of the diagnosis.

SYMPTOMS

The chief symptom—of this there is obviously no doubt—was PAIN, but pain of a very peculiar character. Although it is true that the word neuralgia in itself means merely nerve pain, and since we can scarcely imagine pain which does not involve the nerves, every painful sensation might properly be called neuralgia; but the word has a definite, *clinical* meaning, and is applied only to pain of a certain kind. In the first place, pain which is to be designated neuralgia must be confined to the *anatomical distribution of a sensory nerve tract.* Besides this, however, a *paroxysmal* character is typical of neuralgic pain. It must be borne in mind that all of our four patients presented these symptoms which are decisive in a diagnosis of neuralgia.

Considering the importance of the subject, a closer consideration of the nature of neuralgic pain is quite justifiable. In many cases the patient is able to give such accurate and reliable information as to the seat of his pain that no doubt can exist as to the exact nerve tract which is affected. For example, our first patient asserted positively that he suffered from severe pain in the left leg, which began at the back of the thigh and thence passed through the leg to the sole of the foot and the toes. At our request he traced with his finger the area of distribution of the pain, and we are quite certain that no one could better or more accurately outline the course of the sciatic nerve in the living body. We cannot for a moment doubt that in this patient

the left sciatic nerve was the seat of the neuralgia, and that this case revealed the diseased condition known as *sciatica.*

In the second patient also it was easy to determine which nerve tract was the seat of neuralgia. Here the patient was a man of 62; he was attacked nearly six months ago by *herpes zoster* in the area of distribution of the left fifth and sixth intercostal nerves. Although we did not see the patient at that time, we cannot doubt his account, because we still observe upon his skin five, oval, brown spots, speckled with white, and almost as large as the palm of the hand, which follow the course of the fifth and sixth intercostal spaces from the left side of the spine to the sternum, and which form indubitable proof for any one of experience that zoster must have existed at those points only a short time previously. We learned from the patient that the development of herpes was preceded for several days by violent pain in those areas of the skin in which subsequently groups of herpetic vesicles appeared. The pain did not absolutely cease with the development of the vesicles but ameliorated considerably, and led the patient to hope that with the disappearance of the eruption the pain also would be arrested. Unfortunately he was doomed to disappointment. In the course of the second week the herpetic vesicles dried up and the crusts thus produced desquamated; but this had hardly occurred when the pain became more violent than ever, and for six months persisted with undiminished intensity. In our examination we were convinced that the patient indicated the left fifth and sixth intercostal spaces as the seat of pain for, in order to point it out, he drew the thumb of his left hand along the fifth and then the sixth intercostal space from the vertebral column to the left border of the sternum. After such unequivocal sign-language, we could not for a moment hesitate to assume *intercostal neuralgia* of the fifth and sixth intercostal nerves on the left side.

The localization of the pain is not always so clearly indicated as in these two patients. In our third case, a woman, 43 years of age, we made a diagnosis of *neuralgia of the second branch of the trigeminal nerve* (fifth nerve). This patient asserted positively that the paroxysmal pain did not, as a rule, spread longitudinally, but was generally limited to a deep circumscribed point. From time to time, however, she suffered from attacks characterized by radiating pain.

Moreover, we must remember that not only the peripheral sensory nerves are affected by neuralgia, but also occasionally the sensory nerves of the internal organs, in which case the picture of *visceral neuralgia* is produced.. We are forced to admit that but little is known of this form of the disease, and that, for example, many reports of *neuralgia of the liver,* of *the kidneys,* and of *the stomach* admit of a different clinical interpretation. It is well known that the *joints* may be the seat of neuralgia; and some physicians even include migraine and hemicrania among the neuralgias. In all instances of visceral neuralgia the patient feels paroxysmal pain in the affected organs, but, as a rule, is unable to locate it exactly.

We have pointed out as characteristic of neuralgia not only its seat and distribution, but also its *paroxysmal appearance.* All our patients agreed that the pain was unbearable only at times. Three of them were absolutely free from pain in the intervals between the paroxysms; the man with sciatica, however, asserted (and this agreed perfectly with other experiences) that he was

never entirely free from pain. He felt constantly a dull but endurable pain along the diseased nerve, and at intervals there were paroxysms of short duration that were almost unbearable. It is customary, therefore, to distinguish between *intermittent* and *remittent neuralgia.* Cases like the one just described belong, of course, to the remittent form.

The *number* and the *duration* of the *paroxysms* reveal astonishing variations. In some cases they follow each other at intervals of a few minutes, even a few seconds; in others they may appear only a few times in the course of a day. At times there is a lapse of several days between the distinct attacks, or there may be many weeks. Even in the same patient there are variations, and it may be stated as a general rule that at the beginning and the end of the illness the paroxysms are usually less frequent than at its height.

The *duration of a paroxysm* varies from a few seconds to several minutes. When we hear of a paroxysm lasting an hour or even several hours, closer questioning will usually reveal the fact that there was previously a series of paroxysms separated from each other by such brief intervals of freedom from pain that the patient considered them to be one continuous paroxysm.

It is often necessary to discriminate between *fully developed and abortive attacks.* The latter generally appear as lightning flashes of pain which disappear as quickly as they come, and often visibly startle the patient and cause him to shudder.

The *character of the pain* is very differently described by different patients. Most often it is designated as burning, piercing, or boring. Some patients say that a paroxysm produces a sensation which can only be compared to that of the glowing iron being bored into them; others, that the affected nerve seems to be slowly pulled with tongs. Moreover, the character of the pain varies in different attacks, and it sometimes happens that a paroxysm may begin with a boring pain and subsequently change to a sensation of violent burning.

At times the pain is confined to a certain point, but in the majority of cases it radiates through large areas. In many cases the patients assert positively that the pain shoots from the center to the periphery; whereas, in my experience, a distribution of the pain in the opposite direction is more rare. This has resulted in the frequent distinction between *neuralgia ascendens* and *neuralgia descendens,* but I do not ascribe much importance to this differentiation since, in many patients, ascending and descending pains may alternate, or the pain may simultaneously shoot from a certain point both to the center and to the periphery.

Although the peculiar pain above described is sufficient for the recognition of neuralgia, in many cases there are still other manifestations of the disease which may be grouped together under the generic title of incidental neuralgic findings. Sometimes these are *local changes in the nerves,* sometimes *reflex disturbances.*

Among these local secondary findings, the **circumscribed pressure or pain points** must be given due importance. They are also called Valleix's pressure points (points douloureaux) because Valleix,[1] in 1841, first described them

[1] Valleix, "Traité des neuralgies," etc., Paris, 1841.

in detail. If we trace with the finger the accessible peripheral course of a nerve affected by neuralgia, we will find in many cases that the entire course of the diseased nerve is more or less sensitive to moderate pressure. This is especially noticeable when we outline in a similar manner the corresponding nerves of the well side of the body. In many cases, moreover, we will find the nerves particularly sensitive to pressure at different points which, in fact, represent the painful points of Valleix. By pressing with a single finger-tip we can readily ascertain that the extent of a single area is very limited.

Brenner [1] called attention to the fact that it is occasionally possible by means of the galvanic current to discover painful points in a nerve affected by neuralgia when pressure with the finger does not reveal them. For this purpose we use a galvanic current sufficiently strong to produce sensation in the skin. The anode (positive or copper pole) is placed upon any part of the body as the indifferent electrode, and the cathode (negative or zinc pole) is slowly passed along the course of the nerve. Painful points, formerly obscure, are indicated by the fact that the patient feels pain at circumscribed points when the cathode passes over them.

The *number of painful points* and their *clinical significance* has undoubtedly been overestimated by Valleix; nevertheless they are of the greatest diagnostic and therapeutic importance.

Each nerve has its specially painful points. Our patient with trigeminal neuralgia exhibited an easily recognizable, painful point directly below the infraorbital margin, corresponding to the position of the infraorbital foramen, from which the second branch of the trigeminal nerve finds its exit. In the patient with intercostal neuralgia, three painful points were revealed: One near the spinous processes of the vertebral column, a second about in the midaxillary line, and a third just behind the left margin of the sternum. In the two patients with sciatica the locations varied. The first showed only two pressure points of which the first was situated just below the lower margin of the gluteus maximus, the second, in the popliteal space. In the other case we discerned six pressure points as follows: One near the anterior superior spine of the ilium; one on the lower margin of the gluteus maximus; one in the center of the posterior surface of the thigh; one in the popliteal space; one in the middle calf of the leg; and one under the head of the fibula. We note, therefore, from our clinical observations, that the painful points in any nerve may vary in number, and that in neuralgia confined to one nerve all of the areas known to be painful points are not necessarily involved. Continued observation of patients suffering from neuralgia would probably soon convince us that there are certain susceptible points in every nerve.

The seat of pain in a nerve is usually to be sought just below the surface, or where the nerve emerges from bony canals, muscles, or fasciæ, where it is superficial over bones, and where it gives off lateral branches. To refer once more to our patients, we see that the pressure point in infraorbital neuralgia is an instance of an affected nerve emerging from a bony canal and being superficial, situated upon an osseous surface. The three pressure points mentioned in the case of intercostal neuralgia are of varying origin. The inter-

[1] Brenner, "Ueber die Auffindung von Schmerzpunkten längs der Wirbelsäule und der Nervenstämme." *Berliner klin. Wochenschrift,* 1880, Nr. 21.

costal nerve issues from the spinal canal just as it leaves the intervertebral space close to the vertebral column. The perforans lateralis branches from it on the side toward the thorax, and the perforans anterior emerges from under the skin close to the margin of the sternum. From these two examples it is easy to understand the anatomical distribution of painful points in other neuralgias, as in our two cases of sciatica.

There are no absolutely reliable data concerning the *origin of painful points.* The hypothesis that they can be explained in a purely mechanical way, in that the diseased nerve from its superficial position is susceptible to pressure at these points, must be discarded as incorrect; since, in the same anatomical positions, only circumscribed nerve areas give any indication of being painful points, while adjacent areas, with the same anatomical situation, do not. Whether, however, these painful points bear any relation to the still very little known nervi nervorum, or to the so-called nervi recurrentes, or to peculiar vascular distributions, is a question which has not yet been answered.

Trousseau deserves credit for having added materially to our knowledge of *painful points* by drawing attention to the occurrence of distant painful points in neuralgia, or, as he called them, *points douloureaux apophysaires.* In many cases of neuralgia it appears that not only portions of the diseased nerves but also certain spinous processes of the vertebral column are extraordinarily sensitive; these correspond to the points apophysaires of Trousseau. In the case of neuralgia of the nerves of the trunk or of the extremities, we frequently recognize that only the spinous processes of those vertebræ are sensitive to pressure between which the branches of the roots of the affected nerves leave the vertebral canal. But such points apophysaires may also be found with relative frequency in neuralgia of the cranial nerves; that is, in trigeminal neuralgia in the region of the middle cervical vertebræ.

In exceptional cases, painful points may also be found in distant areas of the extremities; for instance, at the head of the radius or the ulna, and it is this circumstance which leads me to prefer the title, *distant pressure points,* to that chosen by Trousseau.

A very limited experience makes it apparent that the painful points in neuralgia may vary greatly. For instance, in some patients painful points can be recognized only at the time of the paroxysm, while in others they exist also at other times and merely become more painful during the paroxysm. In another respect also a noteworthy difference appears; in one case *pressure on the painful points* may produce an *attack of pain*; in another it causes the disappearance of the paroxysm then present. At times the effect may be lessened or intensified according to whether the pressure upon the painful point is light or heavy. The former is likely to produce a paroxysm, while the latter may cause its disappearance.

Speedy and astonishingly favorable results have been obtained from the *treatment of painful points* by means of the galvanic current. The use of the positive electrode (anode) has rapidly cured cases of neuralgia of long standing. This is true of the far distant as well as of the local painful points; stubborn trigeminal neuralgia (fifth nerve), for example, has often been speedily relieved by treating the painful vertebral points with the anode after all other remedial measures were unavailing.

Nervous disturbances, produced *reflexly,* affect sometimes sensory, sometimes trophic, secretory, or vasomotor nerve tracts.

DISTURBANCES OF TACTILE SENSE

In the majority of cases we find **abnormality of sensation.** Frequently this is at first indicated by *hyperalgesia* of the skin, only at the time of the paroxysm—mere contact with the point of a needle causing excessive pain—while at the same time there may be hyperesthesia—a blunt object, a camel's hair brush, for instance, may be passed over the skin without producing any sensation. In neuralgia of long standing, hyperalgesia and hyperesthesia are often most persistent. After a while hyperalgesia may disappear and be followed by a steadily increasing loss of sensation. In neuralgia of long standing, hyperalgesia and hyperesthesia are often, though by no means invariably, found in the distribution of the affected nerve. On the other hand, complete *anesthesia* or *analgesia* is very rare.

In many cases these sensory disturbances are of purely peripheral origin, especially when a peripheral nerve trunk shows marked change. If, for instance, a nerve trunk is being pressed upon by a tumor, a complete interruption of the conduction of the nerves may readily occur, and the area of skin appertaining to it will lose all sensibility. On the other hand, irritation of the central pressure point may cause violent paroxysms, which the patient, in accordance with the law of eccentric conduction, will refer to the anesthetic region of the skin. This condition, called *anesthesia dolorosa,* is often associated with cancer of the spine, if the cancer presses upon and irritates the sensory nerves issuing from the intervertebral spaces.

It would scarcely be in conformity with the actual conditions if we sought to trace all sensory disturbances which appear in the course of neuralgia to an impairment of conduction within the peripheral nerve tract. Undoubtedly, in many cases an obstruction or a dissolution of continuity in the conduction of sensory impulses in the nerve centers is caused reflexly through a neuralgic irritative condition, especially in the nerve centers of the spinal cord. This condition is indicated whenever the sensory disturbances extend beyond the area of distribution of the affected nerve. As long as they are limited to the diseased nerve, and unless there are changes in the peripheral nerve trunk which point directly to a peripheral stoppage of conduction, it is sometimes impossible to decide whether the cause is peripheral or central.

The same conditions prevail in other disturbances yet to be mentioned. These may be either of peripheral or central-reflex origin, and must be considered individually from the standpoint just discussed.

Vasomotor disturbances appear frequently in trigeminal neuralgia whereas they are rare in other forms; for instance, in sciatica. Our patient with trigeminal neuralgia displayed these so markedly during attacks of pain that one could deduce the onset of a paroxysm merely from the facial appearance. In one case in which I had the opportunity of witnessing the beginning of a paroxysm, my attention was attracted by the sudden pallor of the affected side of the face, this being succeeded in a short time by a still more conspicuous flushing. Obviously the pain had first produced a contraction of the vasomotor nerves which suddenly changed to a paralytic condition of the vessels

supplying the muscles. In neuralgia which has existed for a long time this spasmodic action of the vessels does not appear, and from the beginning there is intense flushing caused by their paralyzed condition. In the case cited I believe there can be no doubt that a reflex change had taken place in the activity of the vasomotors, since evidently only the second branch of the fifth nerve was affected by neuralgia, whereas the vascular change was apparent over the entire side of the face. Moreover, turgidity and increased pulsation of the temporal artery on the affected side of the head were observed during the period of flushing. As was to be expected, the flushed side of the face was abnormally warm to the touch.

Secretory and Trophic Disturbances.—But our patient demonstrated to us that besides vasomotor disturbances there may also be *secretory and trophic changes* in neuralgia. We noticed that as the flush deepened upon the affected side of her face the skin became glossy, and numerous tiny drops of perspiration soon appeared. At the same time the conjunctival sac filled with tears which overflowed upon the cheek of the involved side. Trigeminal neuralgia is characterized by the relative frequency with which secretory disturbances appear, and these are not always limited to increased sweating and the secretion of tears. Some patients show profuse nasal secretion or salivation on the affected side, and the nasal secretion occasionally contains blood.

Occasionally there are reflex, secretory disturbances in remote organs. For example, some patients pass large quantities of pale urine during a paroxysm; and these are not hysterical patients, with whom such occurrences are common even though they do not suffer from neuralgia.

To return to our case of trigeminal neuralgia: I wish to call attention to the presence of *trophic disturbances* as well as those which are vasomotor and secretory. A trained eye will readily perceive that the skin on the affected side of the face is thicker than on the other; and if we pinch up a fold of the skin between two fingers we are thoroughly convinced that the skin is thicker and coarser over the entire affected side. Moreover, experience has demonstrated that at the time of the paroxysm there is swelling of the side implicated, especially when the paroxysm is of unusual violence and duration. Naturally I look upon this not as a trophic, but as a vasomotor disturbance, in fact as a transitory cutaneous edema.

In a few cases of trigeminal neuralgia the exact opposite of this condition has been observed; namely, a unilateral facial atrophy. It has also been reported that the hair on the affected side of the head becomes gray, rough or coarse, or falls out. Occasionally we observe the so-called ringed hair (leukotrichia), in which condition pigmented and non-pigmented areas alternate in a single hair and cause these points to appear as white spots.

In neuralgia affecting various nerves it is not unusual for trophic lesions to appear *upon the skin*. As such we may cite thickening of the epidermis, an excessively active desquamation, conspicuously profuse growth of the hair, and erythematous and vesicular eruptions which either form large, single vesicles known as pemphigus or coalescent groups of pin-head vesicles called herpes.

Trophic changes in the bones and joints occur less frequently. They usually represent thickening, and must not be confounded with inflammatory changes which are often not the result of neuralgia, but its cause. Painful lesions would, in any case, contraindicate a purely trophic origin.

Atrophy of the muscles in an area affected by neuralgia is not rare, neither is it by any means invariable. It would, however, be incorrect always to interpret such muscular lesions as purely trophic. Often they are a natural result of the fact that, on account of the pain, the muscles were used too little and therefore gradually decreased in size. Such *inactivity atrophy* would seem to be indicated when the atrophy of a muscle progresses slowly but continuously for many weeks, yet in spite of this does not become extreme. A pure, trophic, *muscular atrophy* develops rapidly, is often distinctly noticeable in the second week, frequently becomes extensive, and disappears but slowly after the neuralgia is cured. Our two patients with sciatica presented instructive illustrations of both these forms of muscular atrophy; one of them suffered from sciatica only four weeks, yet the muscles of the thigh and leg shrunk considerably. This was apparent on inspection, but measurements showed that the circumference of the middle of the thigh and leg of the affected right side was 4 cm. less than that of the healthy left side. On the other hand the second patient was ill for nearly three months. In his case also the muscles of the affected leg were shrunken, but, in contrast to the first patient, he himself had not noticed this, and measurement revealed a difference of not quite one cm. It appears justifiable, therefore, to consider the first case purely trophic, the atrophy in the second as due to inactivity of the muscles. Reflex causes may be active in the case of muscular lesions also. For instance, neuralgic changes in the sensory area of the spinal cord may produce an inhibition of the trophic cells, possibly also of the large ganglion cells in the anterior horns of the spinal cord. Since, however, such muscular atrophy is usually unaccompanied by paralysis but merely produces weakness in the muscles due to their diminution, it appears more logical to seek the cause of purely trophic muscular atrophy in disturbances of the peripheral nerve tract; in fact, we are warranted in supposing that it is caused by neuritic lesions, and is indicative of these.

DIAGNOSIS

All objectively demonstrable nervous disturbances are of the greatest importance in the *diagnosis of neuralgia,* since obviously they can never be feigned by patients. We are often called upon in our Clinic to treat patients who appear to be suffering from sciatica. The affection is traced sometimes to a fall, sometimes to a blow, sometimes to the lifting of a heavy weight while at work, and accident insurance is demanded as compensation for the ostensible injury. Often the patients go so far as to bring a suit for damages, and demand a medical opinion. If, besides the pain, there are nervous derangements, in which case sciatica would soon produce muscular atrophy, our course is clear and we need not hestitate to testify to the existence of neuralgia. Under some circumstances, however, grave diagnostic perplexities arise. The alleged patients may simulate with wonderful accuracy all the subjective pain of which actual neuralgic patients are wont to complain. I have frequently convicted presumable sciatic patients of malingering by having them watched as they got out of bed, when they appeared to move in a perfectly normal manner, whereas, in my presence, they had limped markedly with the affected leg, and asserted that they could not bear their weight upon it.

DIFFERENTIAL DIAGNOSIS

In a *differential diagnosis of neuralgia,* painful muscular lesions often come under consideration. We must bear in mind that an affected muscle is likely to be sensitive to pressure, that it occasionally shows swelling and thickening, and that the pain never extends beyond the limits of the muscle implicated.

Inflammation of the bones or of the periosteum will often cause diagnostic error. Here we should especially seek to find sensitive swelling and local lesions in the tissues indicated.

At times an *inflammation of the joints* is mistaken for neuralgia. We all know that it is sometimes impossible at the first glance to state with accuracy whether we are dealing with sciatica or hip-joint disease. In the latter there is a typical position of the leg with severe pain when the patella is moved suddenly against the other bones of the knee, and especially on rotating the thigh. It is hardly necessary to say that we must try to locate exactly the lesions of the joints.

ETIOLOGY

Up to this point we have been devoting our attention to neuralgia only as a fully developed disease. If we now consider the *development of neuralgia,* we will find that it seldom appears in the form which it ultimately assumes. Often the onset of an indubitable neuralgic attack is preceded for several hours or days by *paresthesia*; the patients complain of sensations of stiffness, numbness, cold, itching and the like in those areas of the skin which are subsequently affected by neuralgia. Sometimes neuralgia announces itself by lightning-like pain which passes away rapidly and only gradually becomes of longer duration, perhaps also more severe, and develops into unmistakable neuralgic paroxysms.

It is sometimes impossible to determine the cause of neuralgic attacks. They may appear during periods of profound tranquillity, both physical and mental. Patients may be aroused from deep sleep by their onset without having previously been troubled by bad dreams. At other times the paroxysms may follow excitement or irritation of the most trivial kind. Lightly blowing on the skin, touching it, or dropping a scrap of paper upon it, may be sufficient to evoke a spasm. Many patients suffer intense pain during mastication or drinking, especially if partaking of anything very cold, very hot, or very hard; or if they happen to bring the teeth together with unusual force. In some cases the stimulus for an attack originates in the organs of special sense; a glaring light or sharp, shrill tone may produce them. Mental excitement also is frequently a cause, as, for instance, laughter, weeping, the arrival of the physician, fear of an impending paroxysm, or something similar.

Since the appearance of neuralgic attacks depends partly upon extraneous circumstances, in many cases they naturally follow no rule. In others it frequently happens that the paroxysms appear at regular and definite intervals, and continue for approximately the same length of time. One patient will be attacked by a spasm at exactly the same hour day after day, while for

another there may be intervals of two, three, or even five days. This form of the disease is called *intermittent neuralgia*, and is usually the result of *malarial infection*. But it is by no means correct to assume this cause in all such cases, for neuralgia may show a distinct tendency to intermittence even when it has no dependence upon any form of malaria.

The onset of a paroxysm is often revealed by the general behavior of the patients without vasomotor or secretory manifestations. Often the pain forces them to stop whatever they are engaged in doing. They moan and groan, and grasp the affected region with the hands. Sometimes they make the strongest possible pressure upon it, even pressing against some stationary object in order to deaden the pain. They may apply to themselves objects so hot that they receive large burns and scars, especially conspicuous and disfiguring in the face, and these constitute indelible evidence of the disease even after recovery. In some cases the paroxysms are accompanied by clonic muscular contractions—seldom by tonic—of the facial and masticatory muscles on the affected side of the face. While the facial muscles present clonic spasms the masticatory muscles, especially the masseter, show tonic muscular contraction. Trigeminal neuralgia is known to be frequently accompanied by muscular contractions. Trousseau,[1] in his Clinique Médicale, applied to this form of neuralgia the distinctive name of *epileptiform neuralgia*, because the sudden appearance of muscular spasms reminded him of a similar condition in epilepsy. The name can hardly be considered well chosen since this condition bears no actual relation to epilepsy, and this probably accounts for its having so little vogue. Besides, Trousseau considered the disease to be incurable. It must be admitted that it often resists all therapeutic endeavors; nevertheless, complete recovery has been reported.

Obviously the clonic muscular contractions which accompany neuralgia must be of reflex origin, since they often appear in the region of motor nerve tracts. At times distant motor paths are also reflexly affected, so that, for instance, in trigeminal neuralgia, there may be muscular contractions not only on the well side of the face as well as on the diseased, but also clonic spasms in one or both of the upper extremities; occasionally even general clonic muscular contractions.

In a few cases *slowing of the heart's action* is observed during an attack of pain, which shows that the pneumogastric nerve is reflexly affected.

The *constitutional condition*, as a rule, is but slightly disturbed. The temperature remains unaffected; a few reports of its rise after an attack still need confirmation. If the attacks preponderate during the night, sleep is obviously more or less disturbed, and besides suffering from physical and mental weariness the patient is pale and looks ill. Many persons afflicted with stubborn neuralgia find it necessary to lead a solitary life since the violence of the pain makes association with others impossible; moreover, excitement which cannot well be avoided in general contact is likely to increase the number and heighten the intensity of the attacks. Thus the unfortunate patients are often regarded as unsocial and misanthropic. The pain, often unbearable, does not conduce to a calm and equable demeanor; indeed, signs of moodiness and irritation are prone to appear. In a few cases stupor and delirium

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[1] A. Trousseau, "Clinique médicale," Paris, 1862, T. II, p. 44.

have been observed during excessively violent attacks. It is known to medical experience that people with incurable neuralgia have finally been driven to suicide, but this, fortunately, is a much rarer occurrence than statements in certain text-books might lead us to suppose.

In trigeminal neuralgia there is often decided loss of strength which progresses with astonishing rapidity because, in addition to want of sleep, reluctance to eat or drink for fear of precipitating a neuralgic attack tends in large measure to impair the nutrition.

We must bear in mind that *disturbances of metabolism* are said to be frequently associated with neuralgia. In some cases of sciatica the excretion of sugar in the urine, *glycosuria,* has been observed. Here we must guard against error, and must remember that *diabetes mellitus is often a cause of neuralgia, especially of sciatica.*

In many cases of neuralgia the pain is limited throughout to a single nerve branch, in others it gradually involves other branches, even far distant ones. It is not unusual, for example, for trigeminal neuralgia to begin in one trigeminal branch, and gradually to extend to other branches of the same trigeminal trunk. There are two reasons for this. It might be possible for a morbid process, e. g., neuritis of a nerve branch, to involve another nerve, but in many cases there are also so-called *radiation phenomena.* We assume these to be produced by a condition of irritability within the central nervous system which gradually implicates adjacent sensory areas. Neuralgia appears first in a given nerve, and after a time involves the corresponding nerve on the other side of the body. This is often followed by a distinct amelioration or even entire cessation of the pain in the originally affected nerve. In some cases there is a constant alternation of the neuralgia between the two sides of the body. Occasionally we find it distributed to distant nerve paths. Thus, trigeminal neuralgia is often accompanied by neuralgia of the occipital nerves or the nerves of the arm.

No fixed rules can be established for the **duration of neuralgia.** Sometimes the disease ends after a few paroxysms, perhaps even after one, while in other cases it may last for weeks, months, years, or even a whole lifetime. If the causes are such that they cannot be removed, it is impossible to count on the cure of the disease. The termination may either come so suddenly that the patient is abruptly relieved at the end of a violent attack, or else it is gradual, the attacks becoming weaker and shorter, and the intervals between them longer. For some time after the attacks have ceased there is likely to be disturbance of sensibility in the affected region of the skin.

Neuralgia shows a distinct *tendency to relapse.* A new lesion will often affect the same nerve, but in some cases other nerves are affected by the relapse. A nerve that has once been attacked by neuralgia often remains so sensitive that the slightest deleterious influence is sufficient to call forth another outbreak of the disease.

Upon closer consideration of the **causes of neuralgia,** an *inherited tendency* to the affection is of the utmost significance. In families in which neuralgia is frequent, the different members are affected by the malady. We often find in these families persons who suffer, or have previously suffered, from central *neuroses*, such as psychopathy, hysteria, neurasthenia, or epilepsy. Under such curcumstances there may be an *inherited neuralgic predisposition.*

In other cases this *neuralgic predisposition* is acquired. Persons whose nervous systems have become deranged through physical or mental excesses, such as overwork, excessive use of alcohol, tobacco, tea, sexual irregularities, or the like are especially prone to neuralgia. The same causes often lead to hysteria and neurasthenia. Therefore, we do not wonder that neuralgia is often associated with these two last named conditions.

Great loss of fluids and blood often cause neuralgia, especially in women after a difficult labor or too long continued suckling.

Chlorosis undoubtedly favors the development of neuralgia.

As predisposing causes, *arteriosclerosis* and *advancing age* as well as *pregnancy* must be mentioned.

The causes above enumerated are sufficient in themselves to produce neuralgia, but obviously the disease develops still more readily if, besides these general conditions, the nerves are affected by special lesions. In other cases these lesions alone suffice to produce neuralgia, for the neuralgic predisposition is by no means essential for the appearance of neuralgia.

Neuralgia is no doubt often caused by *cold—refrigeratory or* **rheumatic** *neuralgia.* We are less likely to doubt this because neuralgia frequently appears shortly after taking cold. In all probability the cold produces changes in the caliber of the blood-vessels, and these irritate the nerves. It would not be consistent with impartial observation to deny absolutely the possibility of neuralgia being caused by cold. On the other hand, it must be confessed that here, as in similar fields, many manifestations of disease have been attributed to cold whereas the cause was something very different—often *infection.*

Indeed recent literature forces us to the conclusion that direct *infection* of the nerves plays an important part in the etiology of neuralgia. This point is brought out prominently by the fact that, like other infectious diseases, cases of neuralgia appear in great numbers within a short period of time and in a limited region. Nine years ago Benedikt[1] pointed out that during the transition from winter to spring he had seen numerous cases of supraorbital neuralgia in Vienna, and he could explain these only as the result of infection; and only a year ago Wille[2] described the epidemic appearance of neuralgia, chiefly intercostal neuralgia, in Oberdorf in the Bavarian Allgau where, among 1,000 inhabitants, he had seen in three months 108 cases. Reilly[3] also a short time ago described an epidemic of intercostal neuralgia which was occasionally accompanied by herpes zoster. Absolutely nothing was known of the source of infection.

Traumatic neuralgia is of frequent occurrence and is important. The modes in which a nerve may be injured are so exceedingly numerous that it is impossible here to enumerate even the majority of them. Among the most common are falls, blows, contusions, wounds either incised or punctured, foreign bodies, inflammation of the bones and the periosteum, fractures

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[1] M. Benedikt, "Eine endemische Neuralgie in Wien." *Wiener med. Wochenschrift,* 1891, Nr. 11.

[2] V. Wille, "Neuralgia epidemica (localis)." *Münchener med. Wochenschrift,* 1900, Nr. 33–35.)

[3] Thomas F. Reilly, "An Epidemic of Intercostal Neuralgia." *Med. Record,* Nov. 25, 1899.

of bones, dislocations, and the formation of callus and tumors. Weir Mitchell correctly states that bullet wounds of the nerves often cause the most violent form of neuralgia, for which he has introduced a special name, causalgia. At times very stubborn and excruciating neuralgia appears after the amputation of limbs, when amputation neuroma, so called, develops at the ends of the severed nerves. This form of neuralgia was thoroughly described a few years ago by Witzel.[1] Witzel emphasized the fact that all cases of amputation neuroma are not followed by neuralgia, but only those in which there are adhesions to the adjacent parts so that movements of the limb cause stretching or tension of the nerve in question. Hence it is not the amputation neuroma, *per se*, but its adhesion to adjacent parts which is the causative agent of neuralgia. Witzel maintains, not without justification, that like circumstances may often exist in other cases in which it has been the custom to consider pressure upon the nerves to be the cause of neuralgia; for instance, in neuralgia produced by inflammation of the bones, the periosteum, etc.

Secondary infectious diseases, toxic and **autotoxic forms** *of neuralgia*, may all be grouped together, for in the final analysis of these cases it is the toxic material which injuriously affects a sensory nerve.

Under *secondary infectious neuralgia* we may class those forms which develop in the course of any infectious disease. It is well known that neuralgia not infrequently appears in the course of enteric fever and syphilis, also in malaria and in acute articular rheumatism. In gonorrhea I have often seen violent and stubborn sciatica, and, in a few cases, crural neuralgia. Herpes zoster, which must be counted among the infectious diseases, is a not infrequent cause of neuralgia. The epidemics of influenza of the last decade have demonstrated that this infectious disease also forms a very fertile source of neuralgia.

In many cases such forms of neuralgia assume the characteristics of a substantive disease which we call **masked neuralgia.** The best known of these is neuralgia intermittens larvata, which occurs in regions where malarial fever is prevalent, obviously as the result and, indeed, as the only result of an infection by the malarial plasmodium. The regular recurrence of neuralgic attacks, occasional slight enlargement of the spleen, and the quick disappearance of the paroxysms under treatment by quinin are important aids in the recognition of this form of neuralgia. In the future when making examinations of the blood, special stress should be laid on the plasmodium malariæ, the more so since there can be no doubt that neuralgia has a strong tendency to be intermittent even without the influence of malaria, and readily yields to the action of quinin. Seeligmüller,[2] for instance, has shown that intermittent supraorbital neuralgia not infrequently appears in connection with catarrh of the frontal sinus when there is a temporary retention of secretion which causes pressure on the nerves. I would say parenthetically that the use of the nasal douche often effects a rapid cure. The late clinician, Immermann [3] of Basel, deserves credit for having been the first to emphasize

[1] O. Witzel, "Ueber die Entstehung und Verhütung der Neuralgie in operativen Theilen, besonders an Amputationsstümpfen." *Centralbl. f. Chirurgie*, 1894, Nr. 23.

[2] Seeligmüller, "Neuralgia supraorbitalis intermittens." *Centralbl. f. Nervenheilkunde u. gerichtl. Psychopathologie*, 1880, Nr. 11.

[3] Immermann, "Ueber larvirten Gelenkrheumatismus." *Verhandl. d. Congresses f. innere Medicin*, 1885, p. 109.

that numerous cases of neuralgia appear at periods in which acute articular rheumatism also prevails either endemically or epidemically, yet the patients do not suffer from swelling of or pain in the joints. The astonishingly rapid results which often follow the use of salicylic acid or sodium salicylate suggest that neuralgia of this kind owes its origin to the same noxious influences as acute articular rheumatism, therefore we must differentiate a second form of masked neuralgia.

In all probability this field of masked neuralgia will be expanded in the future. As regards influenza, I maintain to-day that it occasionally manifests itself, just as malaria and articular rheumatism, in attacks of stubborn and very severe neuralgia.

In all of the above cases the cause was probably *bacterial toxins* which affected the sensory nerves and produced neuralgia. Since it is a fact, however, that neuralgia appears with varying frequency in many infectious diseases, we must conclude that all bacterial toxins have not an equally deleterious effect upon the nerves. Moreover certain definite nerves, especially the supraorbital, are uncommonly often subjected to infectious influences, which indicates that bacterial poisons have a special predilection for certain nerves, and perhaps also that certain nerves possess a very slight power of resistance to some bacterial poisons. It is, of course, superfluous to say that all this is purely hypothetical.

In *toxic neuralgia* we find exactly the same conditions. Alcoholism is not seldom the cause of very severe and protracted neuralgia, especially sciatica. But lead, mercury, copper, phosphorus, and arsenic may have the same effect.

By *autotoxic neuralgia* we mean such forms as develop from impaired metabolism; for example, in diabetes, in gout, and in uremic conditions. I have frequently been consulted in cases in which neuralgia appeared during acute or chronic gastrointestinal catarrh which soon disappeared under proper treatment of the digestive disturbance. Even though we have reason to believe that abnormal products of metabolism have affected the diseased nerves, nothing is at present known of their chemical nature. From this point of view we may explain the fact that neuralgia is alleviated when we relieve constipation; this Gussenbauer [1] noticed in trigeminal neuralgia.

Neuralgia is frequently caused by **disease of the nerves** themselves. I mentioned above that it often appears as a sort of central neurosis, particularly in hysteria and neurasthenia. Anatomically demonstrable diseases of the brain, especially tumors and suppuration, are frequent causes of neuralgia. Often it is the consequence of disease of the spinal cord. It is well known that sciatica appears as one of the earliest symptoms in tabes dorsalis; and it is an important point in diagnosis to infer either locomotor ataxia or diabetes in an obstinate case of bilateral sciatica. Under certain circumstances other diseases of the spinal cord, for instance, multiple sclerosis, may also produce neuralgia.

Besides the diseases of the peripheral nerves already referred to, we must mention *neuritis* which is often followed by neuralgia.

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[1] C. Gussenbauer, "Ueber die Behandlung der Trigeminusneuralgie." *Wiener medic. Blätter*, 1886, Nr. 32.

Notwithstanding its manifold causes the origin of neuralgia may now and then be obscure. Some cases have been interpreted as reflex neuralgia, and their occurrence has been attributed to the fact that lesions in other organs transmitted by way of the central nervous system produce neuralgic changes in nerve tracts that are sometimes quite remote. Diseases of the female sexual organs are considered to be especially common causes of neuralgia.

Considering the frequency of the disease, little is known concerning the anatomical changes in neuralgia, but this is explained by the fact that neuralgia is rarely the immediate cause of death. . Many investigations and reports must be regarded as inadequate because they are limited to the study of the affected nerves, without considering the central nervous system. It is not improbable that when the peripheral nerve course is unaffected neuralgia may sometimes be explained by anatomical changes in the spinal cord. But this could be verified only by the use of the most delicate methods at present known.

I possess a series of microscopic preparations an account of which may be interesting. Under three different microscopes may be seen transverse sections of both sciatic nerves and the spinal cord of a man who suffered for more than a year from a very severe left sided sciatica brought on by trench digging, by working in the damp earth. He died of an intercurrent and, therefore, very extensive pulmonary inflammation, which afforded us an opportunity to examine the nerves microscopically. Even a very careful scrutiny of the specimens showed nothing abnormal in the nerves or in the spinal cord, much less an actual pathologic lesion; this was especially evident on transverse section, for the neuralgic and normal sciatic nerves appeared to be no wise different. Other specimens from the same patient revealed no lesions. This led to the conclusion that nerves may be the seat of neuralgia for a long time without revealing the slightest anatomical change by the most minute methods of examination. Neuralgia of this kind, according to clinical usage, should be classed with functional nervous diseases or *neuroses*, and it may aptly be designated neurotic neuralgia.

An entirely different state of things is present in the second series of microscopic specimens which I wish to describe. These were taken from an alcoholic who suffered for many months from bilateral sciatica, the left side being somewhat the more markedly affected. He succumbed from exhaustion. I placed transverse sections of the spinal cord and both sciatic nerves under the microscopes. In the case of the spinal cord I was unable to detect any change. But even an untrained investigator could hardly fail to discover in the peripheral nerves the signs of extensive disease. One of the sections of the sciatic nerve was stained and hardened according to Marchi's method in a mixture of hyperosmic acid and potassium bichromate, whereas the other has the characteristic appearance produced by Weigert's process—hematoxylin, copper sulphate, and ferrocyanid of potassium. The preparations resemble each other in the fact that many of the nerve fibers have been destroyed, that others are beginning to disintegrate, that advanced changes are lacking in the interstitial connective tissue; in short, they reveal an extensive degenerative atrophy of the nerves which some might call neuritis, but in my opinion it is impossible to assume in these changes an inflammatory proc-

ess. In any case it is apparent from our own observations that, anatomically, there may be a **degenerative** as well as a neurotic form of neuralgia.

Undoubtedly there is still another kind of neuralgia, the *neuritic form* proper. There is an example of this also in my collection. It is a transverse section of a sciatic nerve which was for a long time the seat of severe neuralgia in a patient affected with diabetes. In this preparation the atrophied condition of the nerve fibers may be easily recognized, but more decided still is the distribution of the connective tissue between the individual nerve fibers—therefore, the endoneurium—above all, its unusual richness in cells. Under such circumstances we do not hesitate to make a diagnosis of inflammation of the nerves; in fact, of interstitial neuritis. Moreover, the spinal cord, as may be perceived through the microscope, presents no demonstrable change. Hence, from a purely anatomical standpoint, we may speak of a neuritic neuralgia as well as of neurotic and degenerative forms.

When the experiences of other physicians are recounted, we find that relatively often parts of nerves affected by neuralgia and excised from the course of the nerve trunk as a curative measure have formed the material for investigation. Lasalle[1] reports a case of stubborn supramaxillary neuralgia in which Ranvier, a universally recognized authority in the field of histology, had undertaken the examination of the extirpated nerve. He found thickening of the connective tissue and degenerative changes in the nerve fibers; in other words, inflammatory lesions. Putnam[2] had occasion to examine ten nerves from which portions had been excised on account of neuralgia. He noticed accumulations of round cells about the vessels, thickening of the vascular intima, here and there plugging of the vessels, proliferation of the connective tissue, necrosis and atrophy of the nerve fibers, and very slender nerve fibers which he was inclined to consider as newly formed ones. At all events, inflammatory changes appeared by far most frequently in the nerves affected by neuralgia. In a case of trigeminal neuralgia which Gerhardt[3] has described there was extravasation of blood into the Gasserian ganglion. Besides this, active pigmentation of the ganglionic cells was noticed, although this was hardly considered to indicate disease. These anatomical reports make it apparent to every unprejudiced mind that neuritic, and we may also add, degenerative neuralgia is by no means so rare as it is to-day considered to be. The relative frequency with which neurotic neuralgia occurs in comparison with the two other forms cannot be determined with any accuracy because these different forms are not always sharply characterized by typical symptoms during the life of the patient. Many physicians consider rapid muscular atrophy to be a noteworthy sign of neuritic and degenerative neuralgia; but this is not diagnostically accurate, and no one can deny that in neuralgia the sensory tract as well as the trophic nerve fibers may be altered in a purely functional manner.

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[1] Lasalle, "Etude sur le traitement de la névralgie rebelle du nerf maxillaire supérieur par la résection du nerf sous-orbitaire dans la cavité de l'orbite." *Thèse de Paris*, 1877.

[2] J. J. Putnam, "Personal Observations on the Pathology and Treatment of Neuralgias of the Fifth Pair." *Boston Med. Journal*, 1891, Apr. 13.

[3] Gerhardt, "Zur Therapie der Erkrankung des fünften Hirnnerven." *Deutsches Arch. f. klin. Med.*, 1880, XXVI.

Certain local lesions might form a basis for the supposition of neuritic neuralgia. In affected nerves which are easily accessible to the fingers, excessive pain in the nerve trunk, or palpable hardening, would indicate inflammatory changes in the nerves. Naturally, neuralgia points to inflammatory processes as a cause; that is, they are usually present when a nerve is injured. To be sure, a certain amount of caution is necessary in diagnosticating the anatomical nature of neuralgia. This is especially true of those cases of neuralgia which develop from disorders of metabolism, and may equally well be either neuritic or degenerative in character. When neuralgia appears in mixed nerves, an inflammatory or degenerative condition would lead us to expect a change in the electric irritability of the affected nerves and of the muscles which they supply, as well as muscular atrophy. Undoubtedly this happens very rarely, and from my own observations I must emphasize the fact that a normal condition of electric contractility by no means implies the absence of neuritic or degenerative neuralgia. A few years ago, when Eulenberg pointed out the occurrence of changes in electric contractility in certain cases of sciatica, his assertions were met with general disbelief. But Nonne[1] has shown that this is by no means erroneous, and in a few cases I have observed the same changes.

All of our present knowledge of the anatomical changes in neuralgia points to the conclusion that these, if found at all, will appear in the affected peripheral nerve paths themselves. May we deduce from this that the seat of neuralgia is always to be sought in the peripheral nerves, or is the affection of the peripheral nerve path merely the occasion for the development of a neuralgic condition in the central nervous system which may continue to exist after the peripheral cause has been removed? That neuralgia of this form does occur I am firmly convinced, for experience proves that neuralgia may remain after the peripheral cause has been removed, no matter by what means. On the other hand, it seems to me to be assuming too much to attribute to this origin all cases of neuralgia. Abundant observations have demonstrated that, after removal of a peripheral center of disease, for instance, by operation, neuralgia of the one nerve ceased instantaneously and permanently. This scarcely seems possible in a neuralgic condition of the spinal cord, and the opinion of Anstie, who regards neuralgia as invariably caused by abnormal processes in the posterior roots of the spinal cord, can hardly be considered more than a conjecture without real foundation.

We are still absolutely ignorant of the reason why neuralgia appears paroxysmally; neither do we quite understand why certain nerves are so much more frequently affected by neuralgia than others. Of course no one will deny that nerves which are superficially situated, which have an extended course, and which run through long, bony canals, are especially liable to be damaged, and we therefore understand why neuralgia is most common in the region of the sciatic and the trigeminal nerves. But this circumstance does not explain why the supraorbital nerve is so often affected in malaria. We know only, for instance, when we recall a case of lead paralysis, that certain poisons possess a special, although so far unexplained, predilection for certain

[1] Nonne, "Entartungsreaction bei primärer Ischias." *Berl. klin. Wochenschr.*, 1887, Nr. 45.

nerves; lead for the radial nerve, and the malarial poison for the supraorbital.

Some clinicians have suggested that we apply the name neuralgia only to those affections in which anatomical changes in the nerves can be demonstrated. The form which we call *neurotic neuralgia* has also been known as *true or idiopathic neuralgia.* In my opinion this nomenclature is incorrect. Neuralgia under any conditions is merely a symptom, and therefore the diagnosis of neuralgia is incomplete. On the contrary, we should ascertain by minute investigation from what causes and what anatomic changes it springs. Rather than lay stress on distinctions between *true* and *false neuralgia,* physicians should be careful to apply the term neuralgia to only such pain as corresponds exactly with our above description; namely, *pain which is limited to the area of distribution of a nerve and which appears paroxysmally.* We must therefore search for the origin in each individual case because this will determine the treatment; moreover, it sometimes happens that the appearance of neuralgia reveals the existence of certain definite diseases. Stubborn intercostal neuralgia is often the first tangible sign of aneurysms which have developed insidiously. An obstinate case of bilateral sciatica may be the first symptom of diabetes or tabes dorsalis.

PROGNOSIS.—The *prognosis* of neuralgia is so far favorable that the paroxysms, *per se,* are not fatal; but it is unfortunately true that many cases respond but slightly or not at all to treatment, and in these the patients suffer unbearable agony. The neurotic forms of neuralgia show the greatest tenacity, especially when they appear in one in whom the predisposition already exists. Even when the cause is perfectly obvious, it is sometimes impossible to effect a cure.

TREATMENT

In conclusion we shall discuss the *treatment of neuralgia*; I must state in advance that I shall make no attempt to give a complete summary of all the methods recommended, but shall consider my task accomplished when I have suggested certain fundamental measures.

The first question to be asked in treating neuralgia concerns the cause, for upon this our therapy will largely depend. And here we must assign due weight to what has been called the *etiological indication.* Malarial neuralgia, for instance, is, as a rule, quickly and permanently cured by *quinin* which has been known for centuries as a trustworthy specific for malaria. The dose should not be too small, but about 2 grams should be given four hours before the expected paroxysm; in the next three days 1.5—1.0—0.5 grams should be administered in order to guard against a relapse. It can be most conveniently taken in capsule, and should be given every quarter of an hour in doses of 0.5 until the desired amount has been taken. Of late there has been an attempt to substitute for quinin euchinin, an ethylcarbonic ester of quinin, as its less bitter taste makes it less objectionable. When patients have a tendency to vomit quinin, it can be given in the form of *an enema.* For this purpose it should be mixed with 50 of tepid water, 5.0 starch flour, and 10 drops of tincture of opium, and slowly injected into the rectum. It often happens, however, that after giving quinin enemata for several successive

days, there are signs of irritation of the rectal mucous membrane, such as pain, tenesmus, and inability to retain an enema. One can still inject the quinin hypodermically, using a warmed solution in glycerin; for instance:

℞ Chinini hydrochlorici,
Glycerini,
Aq. destill. } .... āā 5.0

M. D. S.: Heat the solution in warm water, and inject daily ½–1 Pravaz syringeful in the vicinity of the affected nerve.

If syphilis is the cause of neuralgia, *mercury* and *iodin preparations* are indicated. To be sure, Ehrmann's [1] experience warns us to be cautious in the use of iodin, since this may sometimes cause an attack of neuralgia. In neuralgia polyarthritica larvata Immermann saw excellent results from salicylic acid and sodium salicylate (1.0, one powder every two hours until tinnitus aurium appears). Neuralgia in diabetes is greatly relieved or even entirely cured when we diminish, by a carefully regulated diet, the amount of sugar in the urine. I have treated diabetic patients who could tell exactly from the violence of their neuralgia whether their urine at that time contained much or little sugar, and who regulated their dietary accordingly. At times *surgical interference* is necessary; that is, the removal of tumors causing pressure and the breaking up of perineuritic adhesions. With anemic, weak and nervous patients we must necessarily prescribe preparations of *iron*, *arsenic* or nervines (for example, the *bromids*), strengthening diet, and a stay in the country or in the mountains. Altitudes of over 1,500 meters should, however, be avoided since many people are attacked by neuralgia when at great heights. The same may be said of the seashore, where sudden changes of temperature and of the weather readily produce neuralgia.

Under all circumstances people with neuralgia should partake of a nutritious and easily digested diet, and should avoid mental excitement. The use of *alcohol* cannot be recommended, as it is usually followed by more frequent and more severe attacks. The same is true of strong coffee, strong tea, pungent spices and acids, and the excessive use of tobacco. I have previously pointed out how important it is that the bowels should act regularly; if necessary, mild aperients may be used.

In the case of neuralgia of the trunk or of the nerves of the extremities, I lay the greatest stress on *prolonged rest in bed*. One advantage of this is that the body is kept continuously at a comparatively equable temperature, and it affords the best means of resting the affected nerve. For instance, I consider the treatment of sciatica or of intercostal neuralgia with rest in bed far more valuable than the use of medicines, and I am convinced that many of my patients owe their recovery to this rather than to incidental remedies. In neuralgia of the brachial nerve it is advisable to carry the arm in a sling. Great relief can also be obtained by the use of a suitable invalid's chair.

Besides the few examples above cited (malaria, syphilis, articular rheumatism) but few *specifics* for neuralgia are known, although there is no lack

[1] S. Ehrmann, "Ueber Trigeminus neuralgien bei acutem Jodismus." *Wiener med. Bl.*, 1890, Nr. 44.

of suggested remedies. I have seen no results from methylene blue (0.3, one powder 3 times a day) as advocated by Ehrlich,[1] from turpentine (20 drops three times daily in milk), from potassium iodid (5.0 in 200, 15 c.c. three times daily) or from other supposed specifics.

Therefore, the *treatment* of neuralgia must be purely *symptomatic.* According to my observations the most beneficial effect must undoubtedly be ascribed to the *narcotics,* and among these to *morphin.* If neuralgia resists continued rest in bed and a suitable morphin treatment hope of a quick recovery can be no longer entertained. A dread of morphin, which is frequently found both among laymen and physicians, is quite groundless, provided, of course, that it be used with discretion. Above all the patient must never for a moment have morphin or the morphin syringe in his possession, for he may easily acquire the morphin habit which is exceedingly difficult to conquer, and continue the use of morphin long after his neuralgia has disappeared. I wish to say most emphatically that even physicians who suffer from neuralgia should not give themselves morphin injections. *Unfortunately morphinism is not uncommon among physicians.* I have come in contact with no small number of colleagues in whom morphinism developed by self-treatment with morphin for neuralgia.

In treating neuralgia by morphin we must make a distinction between the attempt to relieve the patient from one specific attack, and the radical cure of the disease. Not infrequently the pain is so unbearable that we are forced to relieve it as speedily as possible by a hypodermatic injection of morphin. This can, however, be accomplished only if one injects a sufficiently large dose, at least 0.01 gram.

℞ Morphini hydrochlorici 0.3
Glycerini, } .... āā 5.0
Aq. destill. }
M. D. S.: $\frac{1}{3}$–$\frac{1}{2}$ a Pravaz syringeful hypodermatically.

In neuralgia of recent origin a single injection of morphin will sometimes effect a lasting cure. After a time, as a rule, there are subsequent attacks, but the intervals between these becomes decidedly longer, and the attacks themselves lighter and shorter. Daily injections of morphin should produce a more or less rapid cure. I recommend strongly the method of choosing each day a more distally located point of the nerve course for the seat of injection. In this way I have in many cases affected a complete cure, especially of protracted sciatica, in a relatively short time.

Far be it from me to recommend morphin as a remedy to which all cases of neuralgia will yield. But, on the contrary, when morphin has failed I have never known a result to be produced by other narcotics, such as chloral hydrate, cocain, or butylchloral as in the following formula:

℞ Butylchlorali hydrati 5.0
Aq. destill. 130.0
Glycerini 30.0
M. D. S.: 15 c.c. every 5 or 10 minutes until the pain is relieved.

[1] P. Ehrlich and A. Leppmann, "Ueber schmerzstillende Wirkung des Methylenblau." *Deutsche med. Wochenschrift,* 1890, Nr. 23.

I wish to add particularly that the use of sulphate of atropin (atropini sulfurici 0.01, glycerin and aq. destill. āā 5.0, $\frac{1}{4}$–$\frac{1}{2}$ of a syringeful hypodermatically injected) which has been so greatly praised, has failed in all of my patients. Although one often hears the statement made that neuralgic patients bear unusually large doses of narcotics astonishingly well, we must not place too much confidence in this; for, as a rule, the patients are able to take large doses of remedies to relieve pain without immediate injury simply because they have become accustomed to them during their illness.

Occasionally good results are obtained by *antipyretics* and *antirheumatics*. Quinin, for example, often relieves neuralgia even when a malarial cause is out of the question. However, in such cases it is necessary to prescribe large doses, at least 2.0 grams or even 5 grams or more. Some patients are greatly relieved or even cured by the use of salicylic acid (1.0 every two hours), sodium salicylate (1.0 every two hours), aspirin (1.0 three times daily), phenacetin (1.0 three times daily), kryofin (0.5 three times daily), lactophenin (0.5 three times daily), antipyrin (1.0 three times daily), analgin (1.0 three times daily), exalgin (0.3 three times daily), tincture of gelsemium (15 drops three times daily), tincture of aconite or tincture of colchicum (10 drops three times daily).

Among the *nerve tonics* from which we may most readily expect good results, I must mention the bromids, and, after these, arsenic. Other nerve remedies have also been tried and praised; for instance, nitrate of strychnin, extract of ergot, phosphorus, zinc oxid, zinc valerianate, nitrite of strychnin. Among the bromids I prefer sodium bromid, potassium bromid and ammonium bromid. For instance:

℞ Sol. natrii bromati.............................. 10.0:200
Add kalii bromati et ammonii bromati...........āā 5.0
M. D. S.: 1.0 c.c. three times daily.

In cases of anemia, arsenic should be combined with iron. For example:

℞ Tincturæ ferri chlorati æthereæ...................... 40.0
Liquor kalii arsenicosi.............................. 10.0
M. D. S.: 25 drops three times daily.

Or, ferri lactici, natrii bromati āā 10.0; acidi arsenicosi 0.1, pulv. rad. altheæ q.s. ut f. pil. No. 100. D. S.: 2 pills three times daily, a half-hour after meals.

Among external remedies the *derivants* are much used. To these belong mustard paste, mustard leaf, cantharidal plaster, friction with spirits of camphor, mustard oil, formic ether, chloroform (linimenti ammoniati, 40, or chloroformi 10.0, olei terebinthinæ, 40.0) or friction with pure turpentine and painting with tincture of iodin. Stimulating ointments are not infrequently used, for example, veratrini, 0.5, adipis lanæ or adipis suilli āā 5.0, rubbed into the painful parts morning and evening. In neuralgia of the nerves of the extremities, dry and wet cupping over the affected nerve trunk are often resorted to. Cauterization is at times effective. Paquelin's cautery is now preferred to the old actual cautery; moxas and heated points are also used. In neuralgia of recent origin we should be very cautious in the use of derivants, for these are occasionally succeeded by more prolonged

and more violent attacks. Fontanels and setons also belong to the category of derivants, but are now seldom employed.

The *electric current* is also liable to increase the pain, especially in a fresh case. As a rule, I prefer the *galvanic current*; but at first it should not be too strong nor used for too long a time. Treatment with the anode is best adapted to affected nerves, and if there are painful points these should be especially subjected to its influence. Stubborn neuralgia with painful points along the spinal column has frequently been known to disappear under galvanic treatment. Cases have been reported in which very weak but long continued galvanic currents produced admirable results; these were brought about by connecting copper and zinc plates with a metallic conductor and placing them over the affected nerves.

The *faradic current* is often used in the form of the faradic brush or the faradic moxa. The faradic brush is usually applied to the skin over the entire course of the nerve, while the faradic moxa is used only over definite areas, especially the painful points.

Franklinization is a method of electrical treatment not yet generally adopted into practice, but is used by a few specialists. Its superiority to the galvanic or faradic current has not yet been demonstrated.

Electric baths are less used for neuralgia than for general nervousness, but, as a rule, they fail to give the results originally hoped for.

Electric light baths have recently been tried, but in this the electricity is probably of less benefit than the warmth.

Many patients are relieved by the *application of extreme cold* or of *considerable heat* to the part. Pieces of ice, or an ice-bag, are applied to the skin over the affected nerve, or it is sprayed with ether, methyl chlorid, or ethyl chlorid. Vapor baths, Irish-Roman baths, sweat boxes, sand and mud (Fango) baths have also been employed. For sciatica I often lay the affected leg on bags filled with hot sand or cooking salt. Astonishingly good results are sometimes produced by the Scotch douche, which consists of alternating streams of hot and warm water.

In chronic neuralgia bath cures are sometimes beneficial, especially the indifferent springs (Pfäffers, Ragatz, Teplitz, Wildbad, Wildbad-Gastein), sulphur, salt and mud baths. I have known the malady to be relieved by treatment in a *cold water sanatorium* after everything else had been tried in vain, even Kneipp's treatment. In most of these cases, however, the pain subsequently increased considerably.

Mechano-therapy, cautiously employed, is often effective, especially in chronic neuralgia.

Massage is considered by its enthusiastic devotees as an infallible remedy, but I happen to know of not a few cases in which massage, although performed by experienced persons, had to be stopped because it aggravated the neuralgic pain. Recently the field of mechano-therapy has been greatly extended by the introduction of vibratory instruments, and Naegeli has proposed certain manipulations for combating neuralgia.

Notwithstanding the fact that even the great Königsberg philosopher, Immanuel Kant, became interested in the cure of painful sensations, *hypnosis and suggestion*, in other words so-called *psychotherapeutics*, yield only insignificant results. Anatomical changes cannot be removed by suggestion. It

is always wise to encourage the patient to bear the attacks as quietly and patiently as possible, for anxiety, restlessness and excitement tend to increase their violence and frequency.

Surgical treatment of neuralgia is generally a last resource, provided anatomical changes in the affected nerve and its surroundings cannot be demonstrated. For one not a specialist in surgery the simplest procedure is to employ *subcutaneous injections* near the affected nerve with substances which will destroy the nerve fibers. Water, ether, carbolic acid, and hyperosmic acid have been used for this purpose. But the method does not insure lasting recovery, for neuralgia frequently returns as soon as the destroyed nerve fibers have reformed. If the seat is in a mixed nerve, permanent paralysis may be the result. *Bloodless nerve stretching and compression of the large arteries* are therefore preferable. In stubborn cases of sciatica I have seen admirable results from bloodless nerve stretching. By this method the leg is stretched at the knee-joint and then pressed as firmly as possible against the abdomen. Gerhardt describes a case of trigeminal neuralgia in which compression of the carotids relieved the pain. It is a much more serious operation if we lay bare the affected nerve with the knife, and then stretch it by means of a forceps (a procedure known as *bloody nerve stretching*) or if we *ligate the large arteries*. Often the affected nerve is severed (neurotomy) or, in order to prevent as long as possible a new formation, larger portions of the nerve are excised (resection, neurectomy) or, as Thiersch suggested, pieces of the nerves are extracted from the bony canals by means of forceps (nerve extraction). Operations are not always crowned with permanent success. If the point of exit in the course of the peripheral nerve is not removed there is no relief, and if nerve conduction is reestablished by the new growth of nerves the cure will be but temporary. Very extensive operations have been undertaken in the hope of removing the cause of neuralgia; for example, the trigeminal nerve itself has been traced into the cranial cavity in order to cut through the nerve toward the center from the Gasserian ganglion and to remove it.

Abbe,[1] Chipault[2] and others cut with the knife as far as the sac of the spinal dura mater in order to sever the posterior spinal roots within it. As regards the formation of new nerves, it should be borne in mind that this may take place even when a portion of the nerve trunk several centimeters in length has been excised. Operation is unsuccessful if the neuralgic conditions have become so deeply seated in the central nervous system that section of the peripheral nerve tract has no effect. It is wise, therefore, to be very conservative and not to prognosticate complete cure after an operation. In mixed nerves, motor paralysis will be the inevitable result of direct section of the nerve trunk; therefore one should consider seriously whether the ensuing motor paralysis will not be a more serious disability than the neuralgia. In any case, we could to-day scarcely find a surgeon who would *amputate a limb* because of neuralgia as the earlier physicians have occasionally advised.

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[1] R. Abbe, "Intradural Section of the Spinal Nerve for Neuralgia." *Boston Medical and Surgical Journal*, 1896, No. 14.

[2] A. Chipault, "Du traitement de certaines neuralgies rébelles." *Gaz. des hôp.*, 1898, No. 8.

HEADACHE AND MIGRAINE

By L. EDINGER, Frankfort-on-the-Main

When the young physician who has finished his clinical studies begins to practise medicine he is confronted by a number of conditions for which his previous instruction has not sufficiently prepared him. Among well-to-do patients many cases differ entirely in type from those which he had previously seen as a student of medicine, and which had been impressed upon his mind at the bedside—patients who had been carefully nursed, and prepared by much preliminary examination, etc., to be utilized in clinical instruction. Although he has regularly attended the Out-Patient Department, a number of mild disturbances are almost unknown to him, because patients who resort to the Clinic do not seek professional aid for these minor ailments. What, however, collegiate instruction has not given him, and cannot confer, is a correct estimate of the relative frequency of individual affections. Only in practice or from the nature of his work in the district in which he practises does he learn their range. Every experienced physician utilizes this knowledge consciously or unconsciously, and this involuntary conclusion as to what is likely to occur often gives him a great practical advantage over the less experienced beginner. Many a physician will, like myself, recall with mortification some of the experiences of his first years of practice, when he did not recognize what was most obvious. The obvious conditions, however, are the most frequent ones. Even the most intelligent of our young physicians are apt to take entirely too serious a view of the condition of their patients during the first years of practice. They have seen so few trivial cases that it is not at all remarkable that they are inclined to make serious diagnoses or prognoses.

FIRST EXAMINATION. DIFFERENTIATION OF TRUE HEADACHE FROM ALLIED AFFECTIONS

These considerations occur to me whenever, in consultation with a younger colleague, I see a case of long continued headache which does not conform to the ordinary type of migraine. The more he has studied, the more likely is he to behold in each case a number of grave affections. True, we should never forget how serious a symptom headache may be under some circumstances. *The knowledge, however, is more important, that most cases of headache are not caused by severe general affections.* If they are actually partial phenomena of such, with some care this can almost always be recognized; it is certainly true of the pain in the initial stage of febrile infectious dis-

eases, true, also, of the pain of nephritis; in most cases headache which is accompanied by delirium represents a symptom of disease in limited areas of the skull; above all, that form which accompanies the varieties of leptomeningitis, and which may readily be recognized; but to these I shall revert later. First, I wish to describe some affections which, causing pain in the head, are often regarded as true headache.

The various **forms of neuralgia** in the head form a special obstacle to diagnosis which, in my experience, is rarely passed with safety. Only recently I saw a young man who was treated for months with numerous remedies because of an exceedingly severe "headache," yet found not the slightest relief. An examination, which was easily made, and was based upon his report that the pains occurred only on the left side, particularly in the frontal bone, revealed that the condition was one of supraorbital neuralgia. The point of exit of the supraorbital nerve upon the left side was much more sensitive to pressure than that upon the corresponding right side; in the entire region of the painful nerve the touch of a cold key was felt to be much colder than in other parts of the head. A single application of ethyl chlorid upon the point of exit of the nerve caused absolute cessation of pain, and this permanently ceased after the same region had been repeatedly subjected to this treatment within the next few days. During the last decade which was so rich in cases of influenza, I more or less rapidly cured quite a number of such patients. Nearly all of them came to me with the diagnosis of "headache." Not always is supraorbital neuralgia so rapidly cured, else a diagnosis *ex juvantibus* might be made from this fact alone. There are *affections of the frontal sinuses* which present the same typical picture and cause the physician great diagnostic and therapeutic perplexity.

Besides true neuralgias, there are radiating pains in the head due to disease of isolated portions of this structure. When a unilateral pain in the temporal region is complained of, it is always wise to have the TEETH examined. In caries, particularly of the upper and lower molars, there may be excessive pain in the temples which increases during the night, even though the tooth itself does not show that it is diseased. Some time ago I saw the extraction of a tooth relieve pain which, localized for eight weeks in the temples, had robbed the patient of sleep, and had made her extremely uncomfortable. Several physicians had in this case diagnosticated "atypical headache." DISEASES OF THE PHARYNX and of the MIDDLE EAR may also cause unilateral localized pain which at first resembles headache. When a patient is brought to us with the report that he suffers almost constantly from increasing pain upon the side of the head, in front of the ear, or immediately behind it, the examination of the ear and, in the latter case, also of the pharynx, should never be neglected. If we find in these areas a pathologic process which can be removed, the pain in the head will soon cease; all will agree with me that, especially in the treatment of headache, rational therapy should be based upon a careful examination.

We should never fail to secure an accurate description of the symptoms from the patient himself. Here, too, I have often seen young colleagues err, for, having in mind the precise histories which they have heard in the Clinic, they often fail to give the patient sufficient time to gain the composure necessary for most persons when they relate their observations of themselves.

Pains are subjective sensations, and their extent depends entirely upon the person who experiences them. He alone is able to estimate their severity; objective examination will reveal to us the cause, but never the intensity of the symptoms. Therefore, it is always well to permit the patient to complete his description. When we shall have learned to what causes the various forms of head pain are due, we will have learned more from the descriptions of the patients than from their examination.

I repeatedly encounter the statement that patients exaggerate their pain. Now we possess no standard by which to estimate the suffering which a certain affection causes some one else. I therefore believe it right to assume that a person suffers just as intensely as he says he does. At all events, we must be humane provided nothing directly indicates that the patient is a malingerer or exaggerates. I have frequently seen hysterical patients who suffered severely from lack of attention to their symptoms, and whose cases were never sufficiently studied. We will help ourselves and our patients much more if we devote to these cases the same consideration that we do to others.

Since the extent of the pain, or the intensity of its expression, depends entirely upon the sensibility of the patient, in my professional opinion the entire nervous condition of the patient must be taken into consideration. By a neglect of this truly self-evident fact, stupendous errors have occasionally been made. For example, here and there a mere "HYSTERICAL HEADACHE" is described as being of peculiar type, while the consideration of cases belonging to this category reveals only that hysterical persons, and also other hypersensitive people, by their constant and loud complaining when attacked by severe headache, often cause the physician to fail to recognize or to erroneously explain a picture otherwise familiar to him. No doubt all of the cases of "hysterical headache" which have been brought to me could be explained as due to a cause which was felt in an exaggerated way or so described. Only such cases may be designated as true hysterical headache as arise without a local lesion, and purely from the outward projection of central disturbances. That such cases exist has not yet been proven and it is even unlikely, for up to the present time no area in the brain is known to be merely the sensory sphere for the cranium.

A particularly instructive case of "hysterical" headache is the following: A young woman, aged 25, was attacked during pregnancy by headache which so increased in a few days as to become excruciating. The seat of pain was first said to be the left temple and the vertex of the parietal bone; but, gradually, the pains felt during the day and especially increasing during the night until they became almost unbearable radiated over the entire left half of the head; they extended to the left side of the neck which felt as if it were forcibly compressed, and thence the pain shot to the shoulder and the left arm, attacks which might almost be regarded as epileptic recurring repeatedly. At first there was vomiting. The attacks were more rare after a few weeks, but the intensity remained the same, and this increased until labor set in, about three months after the onset of the headache. During this time everything was done to relieve the attacks. Nothing was of use, nothing relieved the distressing pain. The physicians in attendance considered every diagnostic possibility from simple functional headache to intracranial disease. Finally, after the labor it was decided to examine the teeth. Two were found to be carious, and after their extraction the symptoms suddenly and completely disappeared. The patient had passed through a long period of suffering from which she might have been exempt if, on account of the temporal headache, the teeth had been at once examined as I have just advised. When I saw the patient a year later, in consequence of the reappearance of severe headaches—

this time after a psychical irritation—I found the anemic, nervous woman to have a typical, hysterical weakness of the arm with anesthesia in this area.

Our knowledge of the past history of the patient must be supplemented by a report as to the frequency, the type, and the duration of the first attacks, also as to the period of life when the first attacks appeared. Naturally we must ascertain also what diseases have preceded and—a very important point—to what extent neuroses or psychoses, particularly epilepsy and headache, have occurred in the family. While the patient is giving us this information, we have opportunity to acquaint ourselves with their general constitutional condition, the circulation of the blood in the head, particularly in the conjunctiva, their psychical habitus, sensitiveness, etc.

Then the head should be examined; first the exit of the nerves, and then the muscles of the neck and their insertion into the occipital bone are to be palpated. Simultaneously the lateral cervical vertebral column should be examined. An examination of the pupils as to their size, reaction to light, and power of accommodation must be included, and the local examination is completed by a mild percussion of the head which may be done with the finger. In most cases the history and this simple examination will indicate our subsequent procedure.

The first question which we ask all patients who consult us for headache refers to the *nature of the pain.* I usually say to them, "We physicians discriminate between *headache* and *pressure in the head.* To which would you refer your symptoms?" In many cases I learn at once that no actual pain is felt, but that my patient complains only of PRESSURE IN THE HEAD. This disagreeable symptom is an accompaniment of neurasthenia. Tired persons, those who are overworked, persons who perform mental work in close rooms, or even those who work too steadily, also those who are hereditarily weak, who have over-exerted themselves either bodily or mentally, those who have had severe diseases—influenza here plays an important rôle—and exerted themselves too soon afterward—all may suffer from head pressure, a sensation which is felt in the entire top of the skull, which may be so extreme as to prevent all work, and is the most prominent symptom among persons who are otherwise debilitated. There is no positive finding in the head to account for the sensations of these patients. Provided there are no other symptoms which directly point to neurasthenia the patient's description of his case is the only aid to the diagnosis. This description is invariably uniform; there is a sensation of severe pressure especially early in the morning upon awakening, and this continues during the entire day, sometimes ameliorating toward night. The *general treatment of neurasthenia* will be discussed in another article. Here I shall only remark that a few measures have proven very serviceable, especially for the pressure in the head. These patients should never rise from bed upon an empty stomach; they should take a little bread early in the morning, or even a piece of chocolate will suffice; the intervals between eating should never be longer than two hours, and late at night directly before going to sleep they should take food, perhaps gruel or zwieback, so that the interval between the night and morning meal is lessened. For these intervals between meals English biscuit are very serviceable because they can be so readily taken during the day. Early in the morning, at mid-

day, and in the evening I prescribe a lukewarm douche of the forehead given for a few minutes with a Weber's eye douche from a low standing vessel to which a rubber tube and sprinkler are attached. Some patients also use with decided advantage a 10 per cent. menthol pomade once or twice during the day. The numerous water fanatics are, primarily, to be restrained from frequently *douching the head, and cold douches must be absolutely prohibited.* For those patients who are able to give up their occupation for a time, a stay in a sanatorium or a residence in the mountains or at the seashore will benefit the neurasthenia. On resuming their occupations, the treatment must be continued as long as symptoms exist or when they reappear.

By far the majority of patients with pressure in the head require *psychical treatment.* They are almost all depressed and morose; occasionally they cherish a secret fear that they are beyond aid, that they are in the first stages of softening of the brain. The fear of this has probably driven many of these patients to commit suicide, and thus escape a dreadful future. These we may greatly help by serious advice; not by pronouncing the affection as very slight; yet at the same time, we may conscientiously maintain that *pressure in the head is not one of the precursors of paralysis,* that, so far as I am aware, it never precedes this disease. The symptoms of the paralytic which resemble neurasthenia must be referred to other regions.

The *prognosis of the affection* is not grave provided the patients can decrease their hours of labor and interrupt it entirely several times during the year. I have observed that it is better to take frequently a short time for rest, than, as is the custom, to take from four to six weeks once a year. Of course, patients in whom this affection has existed for a long time will do well to stop work for a greater period.

If we learn from our patients that they do not suffer from pressure but from actual pain in the head, we must ascertain whether this is due to a chronic affection, whether it is relatively persistent, or whether it appears in paroxysms between which there are intervals of perfect freedom. In most cases the *affections running their course with headache* may easily be differentiated from *migraine.* The attack of migraine with its aura, its sudden onset, its relative increase, its prostration, and the vomiting which is almost invariably present, is a typical, circumscribed, clinical picture. Much confusion has arisen from the fact that many physicians do not discriminate between this and other forms of headache. The pain in the head in migraine plays a predominant, but not the only, part. Before concluding this article we shall discuss this affection, and shall first consider those diseases which, although of diverse nature, are included in a common group based upon the symptom of headache.

To make a diagnosis we should not for a moment forget that *headache is not a disease, sui generis,* but merely a symptom. I believe it a serious omission in the development of our scientific knowledge of headache that this point has been so rarely considered. We could scarcely have attained our present accuracy in the diagnosis of diseases of the lungs if the symptom (cough) had been persistently studied as an affection in itself. Perhaps it is due to the incorrect methods which have previously obtained that we only to-day differentiate a number of maladies in which headache at first constitutes the sole recognizable symptom.

CAUSES, COURSE, AND TREATMENT OF HEADACHE

It has previously been impossible accurately and theoretically to describe headache. We are familiar with some of its main causes, and simultaneously with a knowledge of the causes we have obtained an insight into the methods of cure. Therefore, at present it appears advisable to regard the entire condition from an *etiologic standpoint*.

A. FORMS WITHOUT MARKED ANATOMICAL DISTURBANCE

Many perfectly healthy persons suffer occasionally from *headaches of short duration* but of marked intensity if they undergo unusual mental or bodily strain, or when they do not rest after such exertion. In some they occasionally occur after remaining up all night, in others after excesses of any kind, particularly after indulgence *in Baccho*. Others suffer from violent headache after any unusual bodily exertion, such as mountain climbing. Others cannot endure being suddenly awakened, and after such awakening they have headache. In others this follows the omission of their accustomed midday nap. I can endure an intense mental strain, but am certain to suffer from headache if, after such strain, I must for any reason curtail my hours of sleep.

We know also that some persons are especially sensitive to intense irritation of the organs of special sense, in many headache follows a loud noise, exposure to an intense light, or, in others, an intense odor which they find especially unpleasant.

Under some circumstances mild headache may occur without the person having been subjected to any strain, as, frequently, after mental shock and often after gastric disturbances. In some persons an empty stomach predisposes to an attack. A physician, a friend of mine, is always attacked by headache if he is obliged to see patients before breakfast.

In these slight ailments the individual learns how to protect himself from attacks, and for this cause seldom consults a physician.

Occasionally, however, the causes enumerated may produce *chronic headache*, which varies according to the nature of the individual attacked. We shall first consider minutely some of the common and important forms of headache.

HEADACHE OF CHILDREN

Children are often brought to us complaining of persistent presure pains in the forehead, behind the eyes, and in the parietal region. When we inquire into their mode of life we frequently get reports of badly ventilated sleeping rooms and schoolrooms, that the child has a seat near the stove. that he is daily awakened at an early morning hour, or that he goes to school with an empty stomach or after a hasty or very light breakfast. The little baker boys who are obliged to deliver bread very early in the morning suffer from severe headache. Once I saw this form of headache pass directly into an epileptic attack, and observed the disappearance of both symptoms when the child was sent to another school to which it could go without taking an early morning train, as with the first one.

Sometimes we find the cause of headache in childhood to be **a too constant use of the eyes.**

On entering school, and as they progress in their studies, more continuous use of the eyes is required, and this occasionally produces pain in the frontal region and in the back of the head which may be directly attributed to exertions at accommodation. Not always, but in many cases, an error in refraction, occasionally also a weakness of the muscles of the eyes, especially of the internal recti, may be found. The pain not only appears when the eyes are exerted, but once present is persistent, increasing during the day and disappearing at night. During the holidays this almost invariably ceases. These little sufferers cannot always be helped by the oculist, for what they require is a lessened use of the eyes; but the physician may help them greatly by prescribing a roborant diet, by ordering walks, and by the use of the lukewarm frontal douche which I previously advised for another purpose. Their posture in reading and writing should be observed, and if this be faulty the parents should be urged to correct it. In the most severe cases the children are generally nervous, sometimes they vomit, are restless during sleep, and occasionally impress us as suffering from grave disease. I have observed some cases in which meningitis or chronic hydrocephalus was suspected. Careful examination, particularly a record of the temperature and, in doubtful cases, the result of rest in bed for several days, always led to a correct diagnosis. Especially in little children who cannot accurately describe what they feel, it is very necessary to determine the type of the headache. If we ascertain from our investigations that there are acute attacks with free intervals, migraine should always be suspected, and this suspicion will be confirmed if we find a similar affection in one of the parents or in a brother or sister. Migraine is a distressing affection because it may persist throughout life, and it calls for the greatest care in treatment. The regulation of the diet, the absolute prohibition of alcohol, the strengthening of the body, and the conscientious treatment of the individual attack are primarily important. It is not unlikely that we can do something to abort the attacks if we begin the treatment in earliest youth. I shall later discuss these conditions more in detail.

When a child complains of headache we must never neglect to inquire about its *sleep*. We will then learn not only of serious disturbances, of nocturnal crying, of restlessness and even of night terrors, but therapeutically, by measures calculated to deepen sleep much good may result. The bedroom and the bed must be considered, and only after practicable changes have led to no good result should other remedies be tried. A warm bath before going to bed will often suffice, but no stimulating measures should be combined with this as is so frequently the case; no ablution should be performed, nor should they be washed or rubbed off. They should remain perfectly quiet in the bath for from ten to fifteen minutes. Sometimes drugs are necessary. In this case I usually give a tablespoonful of the following mixture:

℞	Kalii bromati	10.0
	Choral hydrat	2.5
	Antipyrin	2.5
	Syrupus cortic. aur	20.0
	Aqua ad	150.0

This dose is calculated for a child aged about ten; younger children should receive only one-half to three-fourths of a tablespoonful. This dose is never to be repeated the same night, but should be given only upon successive evenings.

If in the adult persistent and severe headache which does not conform to any of the well known types leads us to suspect a constitutional or an intracranial affection, it is even more important to bear this in mind in the case of children. The examination of the urine is especially important as well as that of the eye-ground. *Nephritis* after infectious diseases, which may develop very insidiously, may first be considered, then chronic *hydrocephalus* and *tubercular meningitis.*

THE HEADACHE OF ADOLESCENTS

Headaches due to pathologic conditions of vision occur up to the age of puberty, and they cannot be sharply differentiated from what is ordinarily called **cephalæa adolescentium.** Boys and girls up to eighteen years of age often suffer from excruciating and almost continuous headache. The pain is felt in all parts of the head, but is especially severe in the forehead and behind the eyes. It usually lessens during the night and increases during the day. Actual paroxysms do not occur, but the persistency is characteristic. Those who are attending school are tired and morose, they try to study but are constantly seeking the physician to get relief from their sufferings which are a decided drawback to the best years of their lives. As far as my experience enables me to judge, students of the advanced grades of school are more often attacked than those in the lower grades.

In later life these headaches produced by strain on the part of the eyes are rare, but some occupations which require great power of accommodation may lead to this condition. For instance, I saw very severe symptoms in a prominent wood carver who did very fine work such as the carving of small parts of machinery and the like; I saw the same in embroiderers, and once very tenaciously in a diamond merchant. Diamonds are assorted according to their value, and in deciding this the size, the cut, the luster, and many other conditions come into consideration which, even with excellent judgment and long practice, make constant demands on the power of accommodation. In the last case, after every possible measure had been fruitless, complete rest of the eyes and the strengthening of the constitution by a sea trip produced such marked amelioration that it was practically almost a cure. To-day the patient is again able to follow his very trying occupation.

These affections are not incurable, but, unfortunately, the most effective remedy, the removal of the cause, is not always possible.

Residence in the open air, if possible in a mountainous region, and bodily exercise which requires but little strain upon the sight, are very efficacious. But, of course, we cannot expect that a treatment of this kind for four weeks will produce any noteworthy results. To attain this several months are necessary, and these, unfortunately, the patient cannot always give. Many such a martyr to school and examinations must rely upon other remedies until the goal of school life has been attained, when, before he settles down to an occupation, a prolonged residence in the country or, even better, a winter in

a high altitude or a sea voyage may be possible. We should insist, however, that if during the school life or the apprenticeship such a long vacation cannot be taken it must be as soon afterward as is possible. The patient is then saved much suffering in later life. To ameliorate the difficulty exemption from study or, whenever possible, sports, and lukewarm baths with cold affusions should be advised. Cold ablutions which are so often used, and the douche which is also often advised, I have frequently known to do more harm than good. The *lukewarm frontal douche* is sometimes excellent for these cases. It should, however, fall from no greater height than three feet. The effect often lasts for hours, and this is worth considering. Before resorting to *antineuralgics* I must say a word of warning. They relieve only for a very short time, and must be given too frequently.

Sometimes the use of the *constant current* has proven very effective; however, not for any length of time. A broad electrode is placed upon the nape of the neck and a similar one upon the forehead (the latter being the positive electrode) and the current is gradually increased to one milliampère, which is applied for a few minutes. The question of suggestion, lately so frequently discussed whenever electrotherapeutic successes are mentioned, I do not care to enter upon; I will merely state that in my severest cases no other method gave me such good results.

Cephalœa adolescentium is often coincident with severe pressure in the head, from which occasionally it cannot be differentiated. Unfortunately attacks of true migraine sometimes occur in the same persons. This circumstance alone constrains the physician to do all in his power to relieve the condition before it assumes a chronic form which may persist throughout life.

The factors which produce headache are especially intensified when they affect anemic or chlorotic persons.

HEADACHE OF THE ANEMIC

In this form of headache the intensity of the pain corresponds with the debilitated condition of the patient, and the intervals between the attacks of pain are very brief. The most serious forms I have seen were in girls who had made great efforts to pass a teacher's examination, and soon afterward began their arduous work, which, if conscientiously performed, gives them little time for recreation. This severe form also occurs in poor, under-nourished women who by anxiety and frequent pregnancies, by overworking for their children, by insufficient food and unhygienic mode of life, have reached an unfortunate state of general debility. The majority of these patients, before being brought to me, had gradually fallen into the habit of taking enormous quantities of the greatly abused analgesics, with only temporary effect, but usually enough to relieve the pain so that they might do their daily work.

In these cases only *rest and forced feeding* are of any avail. Since this treatment cannot well be carried out at home, especially when there are small children in the household, the patient should go to a hospital or similar institution. The mere rest from their ordinary duties, which they have until then performed, often has a favorable effect. The patients should be put to

bed. After a week of absolute rest, when they have regained the habit of sleep and the appetite begins to improve, a partial rest cure may be instituted —the patient being kept in the room alone several hours during the day with a window open. During this time they should be placed upon a strengthening diet, and preparations of iron, etc., should be given. I believe that the mineral waters containing arsenic and iron—Levico or Roncegno with which I have had long experience—are usually very beneficial. A tablespoonful in half a wine glass of water should be given after each meal. This should be stopped during the menstrual period or if digestive disturbances appear. From the second to the third week the rest treatment may be relaxed. The patient may have cool ablutions early in the morning—in the first week this is still too severe—and then may be allowed to walk for a gradually increasing time or to be in the open air. All hand work and reading are to be strictly prohibited. The diet should be plentiful, and should contain easily digested fats and carbohydrates.

It is often difficult to adhere to this plan of treatment, but I strenuously advise its continuance, for it is the only one which will bring about permanent recovery. A humane physician who realizes the exigencies of life, and who knows the importance of such recuperative measures, can often by obtaining the assistance and cooperation of his well-to-do patients make it possible for the poor ones who are struggling for the means of existence to carry out this plan of treatment. Mere advice, of course, will never suffice.

The headache of the anemic has caused much theoretic consideration. Here it seemed possible to recognize the cause of the pain, and two explanations have been offered, one of which was a poisoning of the nerves by toxins which circulate in the patient in consequence of abnormal metabolism. From this absolutely hypothetical conception, from which even to-day there is no shadow of proof, the idea has arisen that there are actually poisons which produce headache, and that, for many persons, merely their presence in badly ventilated rooms or wherever a large number of persons have congregated is enough to produce an attack. The opinion is constantly advanced that anomalies of metabolism play a great rôle in the development of headache; for instance, that in persons of sedentary habits—however, quite rarely —headache is due to deficient excretion or to insufficient combustion, particularly of nitrogenous foods. So far as I am able to judge, this view needs further confirmation. True, experience shows that in persons who eat too much and who take insufficient exercise habitual headache is occasionally relieved by the correction of these habits. The hypothesis has also been suggested that anemia of the nerves will produce headache. This I believe to be possible, but another factor must be added, since, in the majority of anemics, the headaches are not persistent. We shall revert to this discussion later.

That abnormal distention of the blood-vessels of the cranium may produce headache is quite certain, for there is a *form of headache which depends upon vasomotor paralysis in the region of the head.* When I inhale nitrobenzol, and even if I merely pass a chemical laboratory where this agent is manufactured, my face begins to redden, my carotids to pulsate, and I feel a temporary but excessively severe sense of pressure within my head. The same effect may be produced by, and be persistent for a long time after, the

inhalation of a single drop of amyl nitrite. Personally I am so sensitive to these agents that, by a sudden headache, my attention is often attracted to "well corked" bottles which contain amyl nitrite.

VASOPARALYTIC HEADACHE

Vasomotor headache which may be artificially produced is a constitutional affection in some persons, and is among the severest forms of headache of which we have any knowledge. The faces of the patients are very red, and here and there are suffused by waves of deeper redness, the lids appear somewhat swollen, the expression denotes suffering and exhaustion. They tell us that headache is really constant, with the exception perhaps of a few weeks in the year when they are in the mountains and free from all care and excitement. Pains are felt in all parts of the head, but are especially severe on the top and in the temples, where it seems as though the head would burst. There is a painful sense of pressure behind the eyes, which seem to bulge forward. The intensity varies, the patients rarely admit that there are days absolutely free from headache, but all of these unfortunates report that at times, perhaps before the menses or when the atmosphere is heavy, the pains become intolerable. The sufferers then, in spite of all their efforts, are obliged to suspend their work and go to bed where they lie moaning. Night does not always bring relief, and often in addition there are a number of other symptoms which might be designated as neurasthenic. Great debility, disinclination for any kind of work, anorexia, distressing dreams with a fright in awakening; occasionally actual hallucinations are present. Often during the comparatively easy periods there is intense pain at the top of the head as if it were bearing a 100 lb. weight, or as if the top of the skull were being crushed. This pain at the top of the head is also experienced as the only symptom of headache in otherwise healthy persons, or it may be associated with other forms of headache. As it is most severe and most frequent in the vasomotor form, it is likely that in other cases it depends upon a distinct vasomotor disturbance. Occasionally it is accompanied by edema of the vertex of the skull.

Examination of patients with vasomotor headache reveals that, besides the special sensitiveness of the scalp and the vertebræ of the neck, the entire skin of the body is covered with red blotches, also striæ due to the pressure of the clothes, or that such blotches and striæ may be produced by gentle stroking of the skin, and that this persists much longer than in other patients. The thought is at once suggested that we are dealing with a weakness in the innervation of the blood-vessels. Eulenberg has termed these cases *paralyses* of the *sympathetic*. They all persist for a long time. Sometimes they occur in persons who also show other nervous disturbances, particularly in members of families with a hereditary taint. We know, too, that the same symptom-complex may be produced at any period of life by trauma of the head; not rarely it is a partial *phenomenon of traumatic neuroses*. The visible, vasomotor weakness of the skin does not in itself produce headache, and the process may occasionally attack the skin alone and not implicate the vessels of the head, for I know quite a number of persons who present this symptom without suffering from headache. Inversely, with a feeble action of amyl

nitrite, vasomotor paralysis occurs only in the head, not in the skin or body, and we are familiar with the fact that the marked rushes of blood to the head from which women frequently suffer during the menopause are usually unaccompanied by pain. We must, therefore, assume that an increased flow of blood to the head causes headache only when it affects the *interior* of the cranium.

As a rule, only the traumatic cases are hopeless. In these vertigo, loss of memory, dizziness and psychical depression are added to the headache, and gradually—certainly in some cases—a transition to degenerative processes of the cerebral vessels takes place. The other cases are probably all curable. If nothing is done to relieve them, they gradually disappear in the course of years. I have repeatedly been led to believe that the gradual change in the mode of life common to every person here plays a rôle. I have not seen a single case after fifty years of age. In the prognosis, naturally, the differentiation from diseases which run a similar course is important, and, so far as my experience goes, only brain tumor comes into question. Perhaps further observation will permit a *differential diagnosis*; but when we see an especially severe case it is sometimes impossible to make this immediately. I remember seeing once a robust young man who died from brain tumor, and who, at the first examination, twenty-four hours before death, complained only of severe vasomotor headache without any other symptom. Therefore, the examination of the eye-ground should never be neglected.

Most cases run a favorable course, and may be checked or relieved by proper *treatment*. A good laxative at the onset is always advisable. This treatment probably causes the vessels of the abdomen to take up relatively larger quantities of blood. Hydrotherapeutic measures, such as cold foot-baths, walking in the water, and douches to the legs act in the same way. I have rarely seen good results from exercise, which has been recommended on account of its influence upon the distribution of the blood; in fact, one of my worst cases occurred in an enthusiastic bicycle rider. This patient, however, always felt better while riding, but most patients are unable to exercise on account of their pain. Whenever possible such patients should be sent to the mountains, and those regions should be selected which have a cool atmosphere without great variations in the temperature; for example, Rigi, Beatenberg, Mürren, Wengen and Obersdorf in Thuringia; the high altitudes in the Southern Schwarzwald also fulfil these indications, although to a less extent than the Swiss regions.

These patients are all peculiarly *susceptible to thunderstorms*. They instinctively feel their approach, even long before, and suffer greatly during the storm. Regions in which thunderstorms are numerous are always badly borne by them. For this reason valleys, even valleys of high altitude, should be avoided. Nor is a *residence at the seaside advisable*. Whether the lower air pressure in both cases plays a rôle we do not know. Even at home much may be done for the patient. Among drugs ergot has been particularly recommended for obvious reasons. Only once have I seen benefit from its use, and perhaps for this reason I do not permit my patients to take it for any length of time. On the contrary, I recommend tincture of nux vomica, and also advise giving to these patients in addition for a long time a milligram of arsenious acid three times daily. The electric current is often found to be

beneficial. My results, however, have not been so positive that I can join in unqualified praise of it. Nevertheless, I have seen some good effects from increasing currents applied to the side of the neck, so that I think its occasional use may be advisable. The analgesics always relieve for a few hours, but never actually cure the affection.

B. HEADACHE DUE TO ORGANIC DISEASE

Up to the present time, the varieties of headache that have been described form a somewhat uncertain foundation so far as the actual etiology is concerned, and we gain more accurate knowledge if we consider *those forms which depend upon organic changes and not merely upon disturbances in function.*

(1) The hairy scalp is exceedingly rich in receptive nerves. We may convince ourselves of this by touching a single hair, which will suffice to produce a sensation. Any one who knows the infinite number of fine, nervous plexuses surrounding the bulb of a hair, any one who has seen the fine processes like the points of a crown forming rings around the hair will easily understand this sensibility. The crown surrounds the hair, and no matter which way this curves it always touches one or more points of other crowns. Under any ordinary circumstances which increase sensibility, and also after severe headache of any origin, **hyperesthesia of the hairy scalp** may be produced and may also attack deeper tissues (hair-pain, clavus). The terminal branches of the trigeminal nerve and of the upper cervical nerves extend through the inner layers of the skin. Various painful disturbances may occur in their course, either because the nerves themselves are damaged, or because disease and irritative conditions in other regions of the skull are disseminated into these nerves. True neuralgias, as a rule, are easily distinguished from diffuse or localized headache, but it is different with the painful sensations produced by disorders of the eyes, diseases of the nose, or of the antrum of Highmore. Catarrh of the frontal sinuses may be felt as diffuse, severe, frontal pressure, and many diseases of the eyes, from insufficiency of the external muscles to glaucoma, usually first betray themselves by headache.

(2) Below the skin lies the galea. This, and probably the periosteum which is directly adjacent, may be the seat of extraordinarily severe headache which is designated as indurative headache.

INDURATIVE HEADACHE

This, probably the *most frequent form of headache,* seems to be almost unknown to the majority of physicians although it has been described in text-books for decades, and although we possess some excellent, especially therapeutic, investigations concerning it. Among the cases that I have seen, which far exceed one hundred, few had been properly diagnosticated and treated. Almost invariably the affection had been taken for migraine or an allied condition. And, indeed, those who have written monographs upon the subject have not attempted to sharply differentiate it from migraine. This distinction is, however, very easily made, and the differential diagnosis influences the treatment which differs greatly in the two affections. In the form under consideration it consists chiefly of massage, hence, as masseurs

did not differentiate sharply between the two affections, it was repeatedly observed that the method which gave excellent results in one case was of absolutely no use in the other, and some physicians who made a study of migraine declared the reports of other physicians untrustworthy because they maintained that they had cured many cases of this severe affection by massage.

The majority of patients with **indurative headache** are women. It is probable that frequent local chilling in consequence of washing the hair here plays a rôle, for it is certain that refrigeration may produce the disease.

The onset is sometimes characterized by symptoms which resemble meningitis.

Some time ago I was called in consultation to see a robust young man; he lay moaning, with all the signs of agonizing pain in the head and the nape of the neck, and had been three days and nights without sleep, with repeated attacks of nausea. Every movement of the head caused such pain that the patient cried aloud. He lay in bed, his neck rigid, and did not dare to move. The experienced family physician had thought of meningitis, but had begun to doubt his diagnosis on account of the absence of fever. Palpation of the head revealed that all the muscles at their insertions and the neighboring galea were extremely sensitive, while the forehead and the vertex of the skull were only slightly hypersensitive. The painful region was perhaps slightly swollen; at all events, hard, extremely sensitive areas could be detected in it. The history revealed that this man, a few days prior to the appearance of what was for him a very severe illness, had been overtaken by a hail storm while riding a bicycle. During the evening he was uncomfortable, felt chilly, and two days later the headache appeared. Here the view that refrigeration had led to a localized muscular affection of the head was quite justifiable. A warm bath, hot poultices to the nape of the neck, and repeated large doses of sodium salicylate brought about such rapid amelioration that upon the next night the patient slept, and after a few days he presented himself during my office hours completely cured.

This was a sudden and unusually severe case of the disease. In most instances the acute onset is overlooked. The patient consults the physician because he is tormented persistently by very severe headache. Of course there are free intervals, but these are usually in the summer; these patients suffer more or less during most days of the year. Sometimes the headache is felt merely as pressure which occasionally increases, but attacks of very severe headache are not rare, they recur at long or short intervals, and closely resemble attacks of migraine. They differ from this affection in that there is no sensitive aura, in the great rarity of nausea and in the absence of vomiting, as well as in the circumstance that most cases arise in later life, while hereditary migraine almost invariably comes on in early youth. But there are exceptions. Of course we cannot say that persons who suffer from migraine may not subsequently develop an indurative affection of the scalp. I have seen two such cases. The most important differential factor is the objective condition. In migraine painful areas of the head are rarely found except during the attack, but here the opposite is the case. One who understands palpation of the head and the muscles of the nape of the neck, and especially one who can palpate them without making too firm pressure, will find in all of these cases that the points upon the skull where the trapezius, the splenii, sometimes also the sternocleidomastoid and scaleni are inserted are sensitive, uneven, and nodular. This unevenness often continues far into the galea. It is hard, as though something were deposited in the

muscular substance, or as if an induration were present. Sometimes there are spindle-shaped sensitive nodules in the points of insertion or in the belly of the previously mentioned muscles, and the periosteum of the upper vertebræ, especially of the transverse processes, is invariably sensitive to pressure.

Proceeding to palpate from the head downward we will find other areas of the trapezius very painful, and will invariably hear from the patient that when the pain is most severe it radiates to the shoulder-blade and the region of the deltoid, which is another symptom that does not occur in migraine. Whether these conditions are directly due to muscle indurations—these hard points are usually regarded as such—or whether, as I suspect, there are only radiating sensations from the suprascapular nerves passing through the deep muscles of the neck, is uncertain. Neither do we know whether true swelling of the lymph-glands and, above all, swelling of the sympathetic of the neck, occurs in this affection as has been maintained by some authors. I have never been able definitely to demonstrate this. As a rule the pains are restricted to the region of the affected muscles and to the fascia, but not infrequently there is very acute pain in the forehead during the attack, although nodules are but rarely noted in the frontalis. Possibly we may here be dealing with irradiation of sensation, radiating pains.

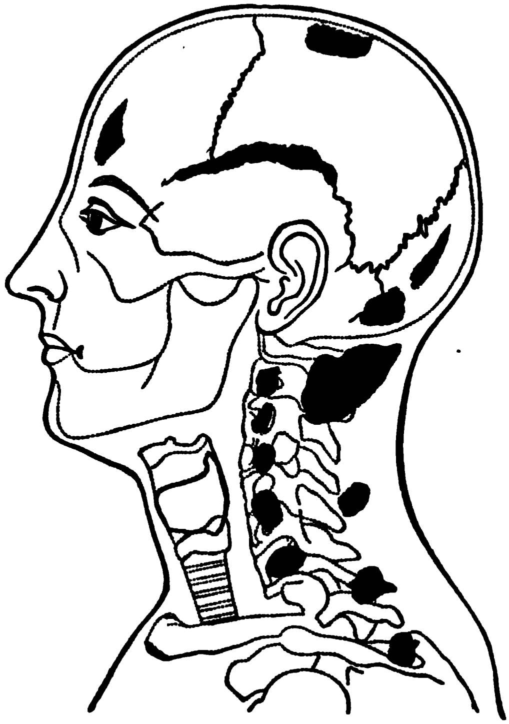

FIG. 194.—THE POINTS UPON WHICH "INDURATIONS" ARE MOST FREQUENTLY FOUND.

The examination of the insertions of the muscles should never be neglected in any case of headache.

Since we know that thermic influences play an important rôle in the etiology of the disease we can almost always detect the causative factors.

Thus, an elderly lady who had been suffering for years from severe headache became in the course of time completely debilitated, for no treatment gave her any relief. She was compelled to give up her flourishing business, and to be content with more moderate means. Upon examining the head and neck, the previously mentioned indurations were found, and the history showed that she had been suffering in the winter for years from innumerable "attacks of cold" caused by the opening of the door which led into her store. Here suitable therapeutic measures brought only amelioration; a cure was impossible.

If the physician does not bear in mind this frequent form of headache. he may fall into grave error. Some years ago a lady who usually lived in a sub-tropical region consulted me for an unbearable "neuralgia" of the head which distressed her day and night. She had long been treated for malaria but without result. Examination revealed at once that the patient had all over her head and the upper part of her body sensitive, subcutaneous, and intramuscular nodules. Here also the cause was easy to recognize. The patient, who was born in Germany, had exposed herself to a strong draught while wearing very thin clothes, as she believed that only in this way could she endure the humid atmosphere.

This last case, as many previous ones, I treated carefully in various ways, also with the electric current, and brought about decided improvement, but I was unable to cure any one until I learned that in the more chronic cases only one remedy was effective, namely, massage. If I had earlier known the publications of the Swedes Henschen, Helleda and Norström, I would the sooner have been able to help my patients. These investigations are still not sufficiently appreciated in medical literature.

There is no doubt that the most frequent and most distressing forms of headache may be etiologically understood by a careful investigation, and with this knowledge a rational method of treatment may at once be conjoined.

The *treatment of indurative headache* is quite simple. It requires patience on the part of the physician and of the sufferer, but it is effective. In the acute cases, before going to bed we give at intervals of an hour three doses of one gram of sodium salicylate, or one-half a gram of salophen may be given four or five times. The disagreeable secondary effects as well as the sweating then occur during the night. Besides this treatment, I make the patients wear nightcaps in the winter. It is also well for the patient to apply for several hours at night hot cataplasms, peat compresses or thermophores, etc., to the back of the neck. No time should be wasted by using analgesics to ameliorate the pain. In such cases these drugs are often abused. None of them has as permanent an effect as the preparations of salicylic acid. After a few days I employ *massage*. At first the muscular insertions are stroked toward the body with the thumb; this is quite tiresome, and the first attempts are also painful to the patient. Especially hard areas must be massaged, and then should follow firmer stroking with the hand, not only of the induration upon the head, but also of all the muscles of the neck, in particular the trapezius and the splenii. The nerves should at first be only gently stroked. Later they will bear firmer pressure, and even a vibratory pressure with the tips of the fingers. Each manipulation should consume from ten to fifteen minutes, including the brief pauses which are necessary. Frequently a surprisingly rapid recovery is brought about; in more protracted cases, however, the treatment must be longer continued and the most experienced masseurs estimate from six to eight weeks as necessary. As the *galvanic current* is unquestionably beneficial, it may be combined with massage. I have never seen any good results from spa treatment. As, however, it is often necessary to send the patients who have been debilitated by pain or by the treatment away from home, regions that are protected from the wind should be chosen. I have repeatedly known a sojourn at the seashore to do actual harm.

Instead of massage, I have used *vibration* with suitable *apparatus* and secured good results. I have been satisfied with this treatment, but it must be cautiously used in persons with *arteriosclerosis*, and the vibrating manipulation should not be attempted upon their heads. Besides the treatment which I have proposed, in older cases I have frequently seen good results from potassium iodid.

The work of the physician is not completed when he has relieved a headache of this kind. He must acquaint the patient with the fact that the pain is caused by getting chilled and must insist that the mode of life be changed so as to avoid the effects of cold. Many a headache in a *bald head* has been permanently cured by wearing a wig. After the conclusion of this treatment I always give the patient a few doses of sodium salicylate, about a gram, and tell him that if the pain returns he is to take immediately three or four powders, is to go to bed, to apply hot compresses to the nape of the neck, and to cover himself warmly while in bed. By these measures we frequently succeed in aborting a fresh attack.

Some of these patients have severe pains in other parts of the body which they believe to be neuralgia, etc., yet a careful examination shows them to be "*rheumatic nodules*," similar to those found upon the posterior part of the head and neck. These patients are regarded as hysterical, hypersensitive persons who are constantly complaining of pain. The skin should always be palpated when any one complains of vague pains now in one, now in another part of the body.

(3) Headache due to disease of the galea and the insertion of the muscles is most common, but is rarely due to disease of the bones of the skull. This has always an extremely local character, so that confusion with the diffuse forms of headache will seldom occur. Palpation and, if necessary, careful percussion will rapidly facilitate our recognition of thickened or softened areas. *Periostitis traumatica and syphilis* produce these conditions. Such organic affections need not immediately be thought of if a person complains of pressure pain or even of pains arising spontaneously; for instance upon the vertex of the skull. The vertex of the skull, particularly in the hysteric, is frequently the seat of severe pain which may even be increased by pressure. The patients sometimes liken the sensation to that of a nail being driven through the head, and the affection has therefore been called *clavus hystericus*. In rare cases there is also pain which is localized in the **sutures of the skull**. It is steady and is increased by pressure. One of my patients, a robust, military man, has suffered so greatly for years from this insignificant affection that he has repeatedly requested surgeons to trephine his skull, and once had the periosteum loosened from the vertex of the skull without any benefit. So far as I know, improvement can be brought about only by general strengthening measures and by the employment of cold locally, also of ice, ether spray, chlorid of ethyl, etc. No sufficiently prolonged trial of the galvanic current has as yet been made.

(4) The *cerebral dura mater* is so rich in nerves that disease of this structure, or merely its subjection to pressure, will often cause very acute pain. Intracranial **periostitis chronica** and **pachymeningitis interna** are relatively the most frequent forms. We think of the latter with its relapsing hemorrhages when severe headache occurs in old persons, sometimes

introduced with vomiting, sometimes occurring in paroxysms with free intervals, occasionally combined with a disturbance of consciousness or paralysis of some cranial nerves. Disorders of speech may also transitorily appear. That analogous processes occur under some circumstances is well known; perhaps it is here worth our while to especially consider influenza, in which pachymeningitis is occasionally added to other intracranial affections.

At the autopsy of persons who have suffered for years from agonizing headache, we not infrequently find upon the vertex of the skull or laterally to this area firm localized adhesions of the dura to the top of the skull; these have perhaps been caused by trauma, or may occasionally be attributed to syphilis. The abnormal venous circulation caused by the formation of new Pacchionian granulations within limited dural areas, particularly in old persons, appears to cause headache.

Of course, these are the more frequent occurrences. If in cases of very severe headache pain is felt within the head, it is always well to consider the possibility of the dural nerves being affected; since, except for the branches of the fifth nerve at the base of the brain inside of the skull, sensory nerves are probably contained only within the dura mater.

There is a true **hyperesthesia of the dural nerves.** In these cases, there is no, or perhaps only very slight, headache during bodily rest. As soon as the patient begins to walk, to ascend a staircase, to jump, or to perform any other bodily exercise, he has a distinct sensation of pain within the skull. He feels that "his brain reels," is distinctly aware of palpation over the sensitive dura, that is, he feels the variations in pressure of the liquor cerebrospinalis. At other times there is actually continuous pain, and we hear that cough, pressure, etc., always produce aggravations. We often hear him say, "It appears to me that with each attack of cough my head will burst." This hyperesthesia is noted under varying circumstances; sometimes in very debilitated persons, particularly during convalescence from severe disease. Rest combined with good nutrition, etc., has an excellent curative effect. The same pains are experienced by persons who suffer from *chronic constipation.* These patients with headache are cured by proper diet and by treatment at Marienbad, Kissingen, etc.

The pia mater probably contains only the nerves intended for the vessels but as it is closely adjacent to the dura, and since nearly all inflammations run their course with exudates and increase the intracranial pressure, it is evident that all forms of leptomeningitis may directly produce headache by pressure upon the dura which is so richly supplied with nerves.

(5) The *brain.* It is probably only by the transmission of pressure that the *headache* occurs which accompanies all *diseases of the brain which limit the extent of this organ.* For the *brain itself,* at least upon its surface, is *insensitive.* I saw a little boy who had been trephined cry loudly when his bandage was changed, but I noted with astonishment that he was perfectly quiet and engaged with toys which had been given him while a knife was several times plunged through the normal cortex to discharge an abscess. Certainly there are portions of the brain the irritation of which produces disturbances of sensation and pain which are then conveyed to the periphery, such as the opposite arm or opposite leg. The area from which painful sen-

sations are transmitted to the top of the skull may be theoretically assumed but has not yet been found.

An organic **intracerebral disease** or a severe **uremic cerebral irritation** must always be thought of when attacks run their course with fever, when the pupils are very widely distended or unequal, or when headache is almost continuous, and vomiting constantly recurs. Accurate investigation of all the aspects of the case then naturally becomes our duty.

In *brain tumor* headache may for a time cause the greatest perplexity in diagnosis. It is extremely severe, occasionally persists for a long time as the only symptom, and is usually confined either to the forehead or to the occipital region. Often other severe symptoms appear, such as rigidity of the pupils, disturbances in gait, and vomiting, and these leave little doubt as to the origin of the severe pain. Nevertheless, I have seen a few cases of excruciating headache which continued for many months before it was possible to diagnosticate tumor. I remember a patient who, after years of suffering, succumbed to a tumor in the region of the island of Reil, and who only a short time before death was seized with severe attacks of headache between which there were intervals absolutely free from pain.

In cases of persistent headache it is always wise to examine the *eye-ground,* and several *examinations of the urine* should be made. Contracted kidney, especially, is often accompanied by headache which we may fail to recognize as a partial symptom of this affection because the amount of albumin in the urine is sometimes almost infinitesimal. In addition to possible albuminuric changes, under examination with the ophthalmoscope we should particularly note the condition of the papilla (neuroretinitis, stasis papillæ).

The treatment of these severe intracranial headaches is often exceedingly difficult. The best results are obtained by continuous treatment with cold, by means of an ice-bag and the like, by rest in bed, and in some cases, particularly when changes have been produced in the dura, by the administration of potassium iodid and purgatives. Of course, wherever possible, we should treat the underlying affection itself. Symptomatically, the various antineuralgics which are to be described under the treatment of migraine are for a time of use.

SYPHILITIC HEADACHE

Headache in the various parts of the capsule of the skull or of the brain may be caused by syphilis. Upon the whole, however, relatively few of the numerous cases of headache are due to this cause. Nevertheless, if the physician is consulted by one who was previously free from severe headache, and who without a known cause is attacked by severe, localized pains here and there at the top of the skull, pains which are particularly worse at night or after excitement and rarely cease, it is well to remember that in the tertiary stage as well as in the initial stages of general syphilis this symptom-complex is frequent. *Syphilitic headache* is extremely severe. The excessive pain, which scarcely ever remits but occasionally even increases, rapidly debilitates the patient and renders him incapable of exertion. We sometimes find these unfortunates crying, kneeling in bed, and even fearing to touch the pillow. Sometimes a definite part of the cranium is particularly sensitive

to pressure, sometimes even one-half of the head. Autopsies and the careful analysis of their findings have demonstrated that all of the parts surrounding the brain may be attacked by syphilis. We recognize a leptomeningitis syphilitica which especially implicates the pia mater, and a diffuse and miliary form of pachymeningitis gummosa which lead to many adhesions between the dura, pia, and top of the skull. We also know that there is a syphilitic osteitis which being specially developed between the dura and the top of the skull produces in the course of time many osseous deposits upon the latter structure. Not infrequently gummata form in the bones of the skull and increase its size. Many of these rapidly disintegrate, and thus produce large openings in the top of the skull. The physician who can make a correct diagnosis in this case, which is not at all difficult, performs a beneficent act. In a *differentio-diagnostic respect*, only *lead poisoning* and the various forms of *infectious leptomeningitis* can be confounded with it. The former very rarely leads to severe headache, and may be differentiated by the history (colic, the occupation, etc.) as well as by the usual lead line on the gums. Meningitis almost always attacks the pia of the cervical cord, and causes rigidity of the neck. On account of the usually increased pressure it is often accompanied by vomiting and slowing of the pulse, which is not apt to be the case in cerebral syphilis. The temperature record will often suffice to differentiate meningitis from the more chronic syphilitic affection. If we are certain that infection has preceded, and other causes of headache are excluded, we should not temporize with symptomatic remedies in these severe cases, but give upon the first night a hypodermic injection of morphin in order to secure rest for the patient, and then as soon as possible institute antisyphilitic treatment. These are the cases in which we should not delay; more than one life has been lost by too long waiting or by a too conservative treatment. Iodin as well as mercury should be given at once in large doses.

This review of the causes of headache and its different varieties may awaken the thought that it is difficult to decide in the individual case with which variety we are dealing. This is not quite true. For, if we exclude the usually recognizable intracranial affections and the exceedingly common form of headache due to induration, only the headaches of children and adolescents remain as relatively frequent forms. In my opinion at least two-fifths of patients with headaches suffer from the indurative form, one-fifth from other forms. The remaining two-fifths are afflicted with migraine, an affection now to be described.

MIGRAINE

Primarily we must regard it as certain that headache of the migraine type occurs in several forms of disease which are perhaps related. It may appear in epilepsy, at the onset and during the course of relapsing paralysis of the oculomotor nerve, in tobacco poisoning, perhaps also in severe forms of hysteria. Most frequently, however, these pains assume the form of a definite, circumscribed disease, *typical migraine*.

The patients who suffer from migraine report that they do not have headache constantly; that, on the contrary, there are sometimes intervals of months, often shorter periods of relief, then quite suddenly, usually early in

the morning, an attack of headache sets in with general prostration and malaise which rapidly increases to such an extraordinary degree that they are incapable of attending to their work; this attack usually lasts the whole day, but becomes milder toward evening, often after preceding vomiting. When the paroxysm is over there is usually complete euphoria. Occasionally the patient who, even late in the afternoon, was suffering severely, is met in the evening in company or at the theatre.

During an attack of migraine many interesting details come to light. First, we almost invariably learn that the disease is well known in the family of the patient. We find that one or another member of the immediate family has similar attacks, also that the parents, sometimes even the grandparents, have suffered from it. Where the type is unknown among the other members, we often ascertain that other neuroses have appeared in the family, especially epilepsy, congenital degenerations, and neuralgias. Of course, there are also cases in which such investigations reveal nothing.

The affection frequently appears in extreme youth; usually, however, it develops only after the responsibilities of life have been assumed. We can scarcely doubt that predisposition, mental, and even bodily exertion may produce the disease. It is also known that it may be caused by prolonged, debilitating diseases and privation. More than one of these patients accurately dates the onset of the affection from the time of his convalescence from some infectious disease. They all know what circumstances produce the individual attack, but they cannot determine the general cause. There are times when the slightest cause or change of condition will produce headache; for instance, just before the menses, a long railroad journey, being present for some time in a close, badly ventilated room, or during great heat; in some cases migraine follows slight constipation; and anger, excitement, the use of the smallest quantity of alcohol or tobacco, an unusually early awakening in the morning, and the omission of a meal will produce a severe attack of migraine.

Whether true migraine ever arises from disease of the nasal cavity and may be relieved by treatment for this is not yet certain; the same is true of the frequently maintained relation of migraine to disorders of accommodation and of the ocular muscles. But there are some patients in whom an attack is produced by a strong light such as they must encounter at concerts and in theatres, and in others an attack follows any work requiring close sight.

It is obvious, therefore, that in migraine we may combat the predisposition as well as the occasional causes of the individual attack. Many children, headache in whom has been previously described, certainly have a *predisposition to migraine.* Strengthening of the body, the avoidance of over-exertion, careful ventilation of their rooms, particularly their bedrooms, is very important. It is self-evident that children should not be allowed alcohol in any form, but I particularly desire to mention it here. Persons with a tendency to migraine should consider this when choosing an occupation. I have seen many a one prevented by this disease from carrying on his work who might have prospered in some other calling. Migraine is most frequently found among professional people, especially among the brain workers, and we possess excellent descriptions by prominent authorities who have themselves

suffered from the disease. Most patients soon learn what will produce an attack; nevertheless, there are a few rules of general application. Among the most important is this, that persons who frequently suffer from migraine should never exert themselves on an empty stomach. I advise, just as in pressure headache, that food be partaken of every two hours even if only a little zwieback or a swallow of milk. The interval between the evening and morning meals should also be lessened by taking food before going to bed. If the patients upon awakening, or even during the night, feel that an attack is threatening, *without raising their heads* and while still in the recumbent posture they should take some food, milk, for example. I believe that an attack may frequently be prevented or aborted by this method.

In some persons an attack of migraine announces itself the evening before its appearance by lassitude, irritability, an incapacity for arduous work. In most patients there are distinct prodromes early in the morning, so distinct that the patients almost invariably know what will happen during the day. They awaken with numbness in the head, and feel extremely tired. They have a distinct consciousness that sleep was heavy and deep; others again have disquieting dreams, and waken feeling completely worn out. Headache appears immediately on raising the head or during dressing, but does not reach its acme until a few hours later, although the pain constantly increases in severity, and is almost exclusively unilateral. It is felt deep in the head, "as if the head were splitting." There is pressure and a sense of burning behind the eyes and in the eyes. In the frontal sinuses, even in the antrum of Highmore, there is pain. To this is added a feeling of great prostration. Energetic persons and those in whom the attack comes on slowly still attempt to carry on their daily work, but in many cases are finally compelled to abandon it. Then a hypersensitiveness of all the senses develops; light, sounds, odors, etc., are distressing; all attempts at accommodation, reading, and any hand work become impossible, and the symptoms frequently increase to such an extent that the patient must be left alone in a quiet and darkened room. The appetite is lost, many patients suffer from retching, and almost all from vomiting, either early or, more frequently, toward the end of the attack. Watery discharges suddenly appear. In the majority of cases these severe attacks last from eight to twelve hours, therefore for an entire day. Of course all are not of the same intensity; some attacks are mild, especially if proper therapeutic measures are instituted. But, unfortunately, there are also extremely severe cases in which an actual *status hemicranicus* develops in consequence of the attack lasting for several days and nights. Directly after its cessation, which may be quite sudden, many patients feel perfectly well. Weaker persons, naturally, still feel debilitated by the sufferings which they have undergone.

Examination during an attack always reveals anomalies of the circulation: The face is usually pale; in some cases, however, extremely red, and the conjunctivæ are injected. Many patients complain of cold feet throughout the attack and, in fact, are inclined to chilliness during the entire period. The pupils are usually somewhat contracted which is an important symptom, since, as a rule, under other circumstances of great pain they are dilated. In some cases there is a difference in the size of the pupils. The palpebral fissure is generally narrowed.

I believe that we may occasionally prevent such an attack of migraine, and have previously mentioned some of the measures to be employed for this purpose. Some drugs appear to be useful, and first among these is potassium bromid, probably because it induces sleep and has a generally quieting action. After a particularly exhausting day, or on a day when the patient notes any of the prodromes of migraine, also before a period during which, according to his own observations, he is particularly predisposed to attacks, two or three grams are given in solution before going to bed, and this treatment should be continued for a few days. A combination with 0.5 chloral hydrate appears to me justifiable. If early in the morning there are signs indicative of an attack—the patient is usually familiar with these signs—after some food has been taken in the recumbent posture one of the analgesics should be administered. Their number is exceedingly large, but I have gotten along so well with the earlier ones, antipyrin and phenacetin, that I have but little experience with the others. Antipyrin seems to have the most certain action in the majority of persons, more so than phenacetin. The dose of either is from 10 to 15 grains. I have often noted that the best results were not secured from these excellent remedies because the patient had not been informed that he must remain absolutely at rest for a full hour after their administration. If it is possible for him to sleep during this time, the attack is aborted or is very mild. If this be impossible, if the attack of headache has set in, we have remedies with which to relieve it but we cannot abort it. Some patients recover the sooner if they resist the attack as long as possible, carry on their work, and try to occupy their minds. If we consider the subjective aspect of the sensation of pain, it appears perfectly rational to divert the attention as long as possible. Absolute rest with self-observation is calculated to add to the difficulties. Often, however, the general prostration is so great as well as the pain that nothing but rest is possible. In this state everything has a disturbing effect, even the ingestion of food, which must be forced, for voluntarily the patients eat nothing. A number of remedies calculated to influence the circulation in the head are useful, but we are not yet so sure of their action that we can order now one, now another remedy. We must still experiment. Heat is acceptable to most patients. Even a bandage applied tightly around the head affords some relief; possibly the artificial pressure is active here by producing anemia of the skin. Rubber bags filled with hot water or thermophores are excellent, others are benefited by hot foot baths. Nitrite of amyl was formerly much employed. It is, however, of such doubtful utility, and may occasionally increase the pains so extraordinarily, that it is no longer used; neither is nitroglycerin which has a similar effect. Only caffein and the analgesics, out of the numberless drugs that have been advised for migraine, have maintained their position. Some years ago an elderly gentleman consulted me for migraine. For many years he had gone for advice to celebrated physicians of America, England, Germany and France. His collection of prescriptions formed a consensus of the opinions of the medical faculty of the world as to the treatment of migraine. Almost the entire materia medica was represented, but only one remedy continually recurred, and this was *caffein* and *pasta guaranæ* containing caffein. There can be no doubt that this remedy, particularly in combination with an analgesic, will afford decided relief. I order

℞ Antipyrin	0.5
Pasta guaranæ	0.3
Caffeini citr.	0.02

F. pulv. d. t. dos. Nr. S.: One or two powders at intervals of an hour.

Large doses of potassium bromid during the attack are also advised by some authors. I do not sanction their employment for they induce vomiting, perhaps on account of the quantity of fluid necessary to dilute them and to prevent the potassium bromid from having a disagreeable and corrosive effect upon the gastric wall. They also increase somnolence after the cessation of the attack; sometimes, but very rarely, caffein produces palpitation of the heart.

A word in regard to *morphin*. I admit freely that I cannot get along without this remedy in the treatment of the severest forms. The attack has so extremely depressing an effect upon the psychical condition of the patient, the pains are so great, the general disturbance from vomiting and, in fact, also from the limited ingestion of food is so extreme that an injection of morphin is often quite justifiable. Morphinism need never be feared so much as to make us, for this reason, withhold from the patient a remedy of the highest value. Indeed, I am aware of no single case that has developed from the employment of this remedy in migraine. The pain and nausea are greatly lessened by the injection of 0.015–0.02.

The treatment of the intractable forms will severely tax the skill of the physician. Besides the treatment of the attack, it is occasionally necessary to remove the patient from his usual surroundings and place him under more favorable hygienic conditions.

I know a lady who suffered so much that she was obliged to leave her husband and child, and spend a whole year at the seashore and in the mountains to regain her health. In the main, she attained her object. The attacks, which at first occurred several times a week, have become very rare and have almost ceased.

In addition to ordinary and general strengthening remedies, and the previously described precautionary measures, food, etc., *arsenic and iron* may be employed with advantage; I know of but one other remedy which will lessen the number of attacks, and this is the *galvanic current*. I have frequently employed it, and have notes of many cases in which it proved of value.

A very active woman, the superintendent of a large business, had almost daily attacks during her busy season. Here I succeeded, not once but repeatedly, in prolonging for several days the periods free from pain. There was no doubt of the success. I resorted to galvanization, applying weak currents to the neck.

As it has been repeatedly asserted that true migraine disappears after the *cure of nasal disease*, I always examine the nose in severe cases, but I must admit that I have seen no case permanently cured by operation upon the nose.

A number of migraine patients are not attacked primarily by headache, but by *derangements of vision*. In these patients the condition is usually

designated as "*flittering scotoma,*" and to this the attack of true migraine is added, although it may in rare cases be absent or very slight, for instance, there may be merely numbness in the head or frontal pressure. We must, therefore, assume the cause of the disordered vision and of the headache to be the same, but they do not always produce these symptoms in combination. Attacks of headache without disturbances of sight are more common than flittering scotoma without headache. The disordered vision is always an early symptom, a sort of *aura of the attack of migraine.* It appears upon the side of the head opposite to that which is attacked by headache.

The subjective ocular symptoms are usually described by the patients, especially by those who study their symptoms, as floating dark spots in the visual field, the borders being serrated and illuminated. Others see only the illuminated edges, or they may complain of dimness of vision. The latter may be the precursor of manifold disturbances of sight, concentric limitation, hemiopia, even actual amaurosis fugax. The affection is at first limited to one eye only, but it is quite certain that in most or in all cases the other eye also becomes implicated. Such an attack of scotoma may last for a few minutes, or its duration may be an hour.

The same cause which produces migraine also produces other *brain symptoms,* but more rarely than disturbances of sight. Here it must be emphasized that transitory disturbances of sensation in one-half of the body may appear, even transitory aphasia. After prolonged attacks of migraine, especially if they are frequent, motor disturbances are observed, such as hemiparesis, also contractions in the extremities.

The clinical picture of migraine is of course circumscribed, but the physician who investigates the nature of the disease will find these occasional additions to the symptom-complex highly interesting. The fact that *epilepsy* may begin with a flittering scotoma, and even with a true attack of migraine, is of great significance. There are also attacks of migraine in which the visual disturbances, the debility which rapidly ensues, and the numbness in the head play so great a rôle that the malady may be confounded with an abortive epileptic attack.

The relations of epilepsy to migraine are still somewhat vaguely understood, but from what has been reported we may conclude that these diseases are either occasionally due to the same cause or, what is much more likely, that the same predisposition of the nervous system produces them.

It has been previously remarked that migraine is sometimes combined with other forms of headache, especially often with the headache of children and with indurative headache. I mention this again because of its diagnostic importance.

THE THEORY OF HEADACHE

Having described the important forms in which headache plays the predominant rôle in the symptom-picture, in some being regarded as the cause, and in some having been shown to be the causative factor, the questions arise: WHAT SYMPTOMS ARE COMMON TO ALL THESE FORMS? HOW DOES HEADACHE ARISE?

All forms of headache become manifest in the region of the trigeminal

nerve. Only in isolated cases are the sensory branches of the upper cervical nerves affected. These nerves first run to their ganglia (Gasserian ganglion, intervertebral ganglion), and thence as "roots" to the central organ. *Numerous observations have proven that the irritation of a sensory nerve, of its ganglion, or of its root produces pain.* It is just as certain that pain is then felt in the area of distribution of this nerve. As to the involvement of the roots, so little has as yet been reported that I shall cite a few cases. A celebrated musician, throughout his life, suffered frequently from excruciating headache which usually radiated to the back of the head. At the post mortem, I found embedded in the roots of the upper cervical nerve an old cicatrix which involved particularly the region of the posterior horn.

The roots penetrate the central apparatus, and from the pons to the cervical cord those which supply the trigeminal nerve gradually become more scanty and finally enter their terminal nuclei. The terminal nuclei of the cervical nerves lie a little above their points of entrance. In spite of the fact that many diseases of the pons and the upper spinal cord have been studied, *nothing has as yet been revealed which unquestionably proves that a disturbance of the intracerebral portion of the root produces pain. A priori,* this is not impossible, but conditions which produce pain are apparently very rare or they never exist.

The nerves of the central tract pass into the terminal nucleus of the sensory nerve, which runs to the corpora quadrigemina, and on to the thalamus opticus. It is quite conceivable that a disturbance within this tract would be communicated to the periphery as pain. But, in spite of the fact that many researches have been made, *distinct pain in the path of individual nerves after a lesion of the central tract has never been observed.* It is, however, known that when the sensory tracts which unite and are enclosed in the caudal-ventral portion of the thalamus are subjected to definite impressions, very severe pain is produced throughout the other half of the body. This has been proven by autopsies.

From the region that has been mentioned nerve tracts pass to the thalamus and the cortex of the brain, and the terminal portions in the thalamus unite with those of the cortex. Yet, notwithstanding the fact that cerebral localization has been minutely studied in the human brain and in animals for the last thirty years, no area of the cortex has yet been found in which the trigeminal nerve alone is present. Irritation of such a region must, under some circumstances, produce pain in the area traversed by this nerve.

On surveying this path we conclude that *isolated irritative symptoms are practically communicated only from the peripheral area of distribution of the nerve, and thence to the supposed cortical field of the same.* In all other areas of the central apparatus, the paths intended for other nerves lie so close to the small central tracts that any disturbance must certainly involve a large area containing many fields of innervation. This may at once be applied to the question of headache. As headache is rarely accompanied by pain in other parts of the body, the areas between the nerve roots and the cortex of the brain may be excluded as the apparatus of transmission.

We now turn to the discussion of the nerves and the cortex. It has been stated that no cortical disease is known which produces pain. If we assume such to exist, two other assumptions must follow. First, that there is a closed

trigeminal field; second, what is certainly most unlikely, that isolated portions of this field, for instance, only the definite fields intended for the fibers of the trigeminus at the top of the skull, are diseased, which form a brace of very hypothetical views. Even if the assumption of a cortical headache were tenable, *it is at present absolutely unjustifiable to assume that its point of origin is the cortex.* Naturally the perception of pain is quite a different matter. This can hardly take place anywhere except in the cortex.

That very severe headaches may be caused by an affection of the peripheral branches of individual cervical nerves is taught by the previously described base of "rheumatism" in the muscles of the head and neck. What about the fifth nerve? In so far as typical headache is concerned we may at once disregard a localization in the root or in the Gasserian ganglion. If this really occurred, headache would occasionally be associated with pain in all other branches of the trigeminal nerve, and this, as we know, is not the case. The same objection might be made to the view of a localization in the main branches.

Only the view remains that somewhere in the cutaneous or dural branches which supply the attacked area lies the pathologic cause. No doubt rheumatic affections or indurations of the head often lead to direct damage of the cutaneous branches of the fifth nerve, for the pain is not only felt in the posterior part of the head but also very acutely over the eyes and in the temporal region. The dural branches of the trigeminal nerve must, however, be frequently damaged. Many patients distinctly state that intense pain is felt within the head, especially behind the eyes; in the most severe forms of headache, in migraine, and with an increase of intracranial pressure, other dural nerves, the branches of the pneumogastric and the sympathetic, are implicated, as is proven by vomiting and various changes in the pupils. This damage must, as a rule, simultaneously affect all intradural nerves, for the headache is of diffuse character, and this especially distinguishes it from neuralgia. We therefore arrive at our second conclusion that the dural branches of the fifth nerve form the point of attack in intracranial headache, and that all the dural nerves are uniformly attacked, at least all upon one side.

So far we are upon an anatomical and sure foundation.

Of what *nature* is the lesion of the dural nerve?

It is certain that increased pressure within the skull is manifested by headache, but pressure alone probably does not cause the disturbance. We not infrequently see local pressure upon the dura without the production of pain, and we often note that in tumor of the brain the headache increases and decreases without corresponding change in the size of the tumor. There must be another factor conjoined with cerebral pressure. Such, for instance, as the disturbance produced in the circulation of the blood and the lymph within the tissues surrounding the dural nerves. It is known that these disturbances may cause acute nerve pain. The severe neuralgias in the extremities due to the contraction or occlusion of individual vessels and the neuralgias caused by an increase of venous blood in the sciatic and other nerves are excellent examples of this. If, however, anemia, and probably also venous hyperemia of the dura and of the pia may produce headache, many of its well known clinical forms are explained. I refer to vasomotor headaches in

those persons with abnormal vasomotor irritability, and to the headache occurring during menstruation. Headache from the inhalation of amyl nitrite, which unquestionably dilates the vessels of the head, should not be forgotten. *Hyperemia in the area of the dura, therefore, may produce headache, as may also local anemia due to pressure.*

These considerations bring us, somewhat in advance of the explanation of headache, to the discussion of intradural and extradural organic diseases. When we consider the forms of headache not produced by severe organic changes, we are entirely in the realm of hypothesis. Obviously we must here consider also anemic conditions not due to pressure but to vasomotor disturbance, to constriction or dilatation of the nerve vessels. It is quite conceivable that headache may be produced reflexly either because abnormal irritation (as from the eyes, from the nose, or the ears) is conducted to a normal apparatus and produces more or less intense vascular spasms, or because otherwise normal irritation may produce the same effect in an abnormally irritable nervous system. The most severe vascular spasms would consequently be observed in hereditarily debilitated persons, in whom, as is well known, migraine is common, its paroxysmal character and its intimate relation to epilepsy having for some time been explained by this view. In migraine the pallor of the face so frequent during the attack, the pale eyeground, and the general vascular spasm which causes chilliness are distinctive. Moreover, the disturbances of sight, the occasional disturbances of speech, and the rare epileptic attacks during the painful period find explanation in the assumption of vascular spasms which are not limited to the dura. Since DuBois-Reymond explicitly reported his own attacks of migraine which he explained by this theory, the malady has been much discussed. His hypothesis, however, has lately failed to be generally accepted. The investigators who have most closely studied the disease in the last few years, Liveing, Gowers, Möbius, do not believe the proofs brought forward sufficient to meet the possible objections. In the circumstance that dilatation of the blood-vessels also produces headache I see no contradiction. Any disturbance of the circulation, no matter of what nature, will temporarily damage the peripheral nerve.

Nor do I concur in the objection that migraine, like all pathologic and physiologic processes, must be produced by the parenchyma cells, that the undoubted vascular disturbance is only secondary; for it has been demonstrated to be impossible to attribute the symptom-complex, "headache," to any area of the brain. But the opponents of the vasomotor theory consider this alone, and they even go so far as to locate there absolutely hypothetical irritations and processes of discharge. Still less reasonable is the claim that amyl nitrite, which paralyzes the vessels, only rarely relieves migraine produced by spastic processes. We know that paralysis causes pain. Further researches will determine whether the reported cures do not depend upon a medium dose, and whether this might not, perhaps more readily, be attained by nitroglycerin. The innervation of the head by the sympathetic nerve is so complicated a process that we cannot expect headache to be especially prominent in disease of its entire tract. But from the circumstance that none of the relatively few cases of disease of the sympathetic have been associated with migraine, etc., objection has been made to the vasomotor theory of individual varieties

of headache. Upon the hypothesis that many forms of headache depend upon vasomotor, especially upon vasospastic, processes it might readily be concluded that disturbances in various sensory areas might reflexly produce headache. This theory alone would explain toxic headache. Unfortunately we are too little acquainted with the relation of the finer vessels of the dura to those of individual nerves.

This must incite us to renewed investigations. It is quite conceivable that severe pain in these vessels may lead to conduction disturbances in other nerves. We know this to occur in paralysis, which is known as *periodic oculomotor paralysis*. There is a complete paralysis of the third nerve always accompanied by a true attack of migraine. It is usually very severe; sometimes it is the first from which a person suffers, at other times paralysis occurs only with a later attack. This paralysis may exist for months and then disappear, or, long after it has passed away, even years after, there may be a renewed attack with most severe pain. Möbius, who first described the symptom-complex, elaborated a few differential points in the picture of migraine which leads to paralysis of the ocular muscles in comparison with a typical attack. According to this author a most important point is that the attacks are very rare, that they are particularly severe, and that hereditary predisposition, as in most cases of true migraine, cannot be determined. Since, however, oculomotor paralysis has been observed occasionally in typical migraine, the question whether these affections are distinct is still a matter of discussion. Two necropsy reports of such cases are at hand. In both a tumor was found at the root of the nerve. We know that intradural tumors when situated in the motor zone, or even in other areas of the cortex within the region of the hypocampus major, often lead to occasional attacks of true epilepsy. It is therefore quite possible that they may generate other spastic symptoms at the base of the brain, and may also be the cause of vascular spasm. We then assume that vascular spasms in the area of the cerebral arteries are due to an unknown cause, probably based upon heredity, and are also directly due to a tumor of the base. The similarity of the clinical pictures as well as their slight differences would thus be explained.

It will be observed that, on the whole, we are upon hypothetical ground. If, however, renewed investigations give us any light, we must clearly discriminate between what we positively know and what is mere surmise. *We know that the agent which produces headache attacks the dural nerves, perhaps also some of the nerves of the pia. The sensation of pain is, of course, conveyed through the brain. We know that headache may be due to various causes. We do not understand the mode by which they damage the nerves, nor in what manner they produce pain. Anemia of the nerve is, at all events, a factor which may here be included, as well as hyperemia. Perhaps most forms of headache may be attributed to these two conditions.*

Naturally, the physician must not allow this theoretic explanation to forestall treatment. Empiricism has made us acquainted with a number of remedies and processes which may afford relief. The development of diagnosis indicates the points where our aid may with advantage be offered. We know this much of headache: that each individual case should interest and stimulate the physician; and we know so little of its theory that now when positive, fundamental laws are everywhere determined, the questions which have arisen should form a new stimulus to scientific endeavor.

PARALYSIS AGITANS[1] (PARKINSON'S DISEASE)

By W. ERB, Heidelberg

Our knowledge of paralysis agitans has almost wholly been acquired since the beginning of the nineteenth century. In 1817 the disease was for the first time clearly described by Parkinson, and subsequently by various authors (Todd, Romberg, Trousseau and others) with more or less exactness; in these descriptions, however, there was some confusion with different related pathological conditions, with chorea, and with other forms of tremor, especially with the intention tremor of multiple sclerosis.

It was about thirty or forty years later before the clinical history and diagnosis of the disease, as well as its nature and pathologic importance, were based upon a firm and positive foundation.

We must assign to Charcot and his pupils in the Salpêtrière the credit of having given us an almost exhaustive symptom-picture of the features of this peculiar disease; they had at their disposal uncommonly rich material which facilitated the diagnosis and, above all, enabled them to sharply differentiate it from multiple sclerosis. The work of Ordenstein (1867), which was inspired by Charcot, marks this important advance in the history of paralysis agitans.

This field has since been further developed, especially by German authors; some characteristics of the pathologic picture are still doubtful, for others a correct explanation has been found. The two principal symptoms of the disease are the following: The *tremor* and *muscular rigidity* (to define it briefly), which have been clearly emphasized; and special stress has also been laid upon the remarkable and frequent occurrence of cases *without* tremor; we now, at the beginning of the twentieth century, possess a definite, complete, and almost exhaustive symptom-picture of Parkinson's disease.

We have been less fortunate in the recognition of the pathologico-anatomical foundation and the nature of the disease. With increasing minuteness in histologic methods of investigation, renewed efforts have been made to base this severe disease, which with apparent justice was regarded as a "functional neurosis," upon a recognizable anatomical foundation. Many observers obtained more or less conclusive findings, but these have by no means as yet furnished a satisfactory explanation, and are still *sub judice*. We may assert to-day that no positive and undoubted anatomical foundation for the disease has yet been revealed, at all events none has been generally accepted. The solution of the problem is left, therefore, to the twentieth century.

[1] Synonyms: *Shaking Palsy; Spasmus Agitans; Chorea Festinans seu Procursiva, etc.*

The therapy of this intractable malady has not kept pace with its exact recognition; in its treatment we are almost as powerless as we were fifty years ago, although we have succeeded by some general procedures, which are extremely acceptable to the patient, and by a few palliative drugs (hyoscin, duboisin) in ameliorating this most distressing affection.

DEFINITION

By paralysis agitans we mean a *disease of advanced age* which is almost exclusively confined to the *motor-apparatus,* and is manifested by a *characteristic tremor of the limbs gradully increasing in severity and extent,* subsequently affecting the trunk, and also marked by *general rigidity and the slowing of muscular movements* with increasing *muscular tension*; from these symptoms almost all of the other phenomena of the affection may be derived, the *anatomical foundation of which has not been positively determined.* The disease is *of progressive character* and is usually fatal, but only by exhaustion and marasmus after a prolonged duration.

ETIOLOGY

Up to the present time the causes of paralysis agitans are very imperfectly known. Gradually, however, a few points have been clearly demonstrated to be of undoubted significance and effect.[1]

[1] In the following I shall give in part the substance of numerous original observations, consisting of 183 cases which I saw within fifteen years. Of these 183 cases, 143 occurred in my private practice and 40 were observed in my Clinic. (A number of cases which were also seen in the Clinic have not been included because the notes of the cases were not sufficiently comprehensive.)

I shall here mention these results somewhat in detail as they may be of use in the compilation of later and more full statistical reports.

Among the 183 cases there were 129 men and 54 women, which gives a proportion of about 5 to 2. In my private cases the number of men was greater than this: 105 men to 38 women, which gives a proportion of about 3 to 1, while among my clinical patients there were 24 men and 16 women, therefore an exact proportion of 3 to 2. The total number of these (40) is, however, relatively small.

In regard to *the age* at which patients were attacked by the disease, the following table is conclusive:

Onset at Age of	Men.			Women.			Total.
	Private.	Clinic.	Total.	Private.	Clinic.	Total.	
21–30 years.......	2	1	3	..	..	..	3
31–40 years.......	10	4	14	4	1	5	19
41–50 years.......	18	10	28	11	4	15	43
51–60 years.......	46	6	52	18	11	29	81
61–70 years.......	27	3	30	5	..	5	35
71–80 years.......	2	..	2	..	..	..	2
	105	24	129	38	16	54	183

From this it is evident that by far the greatest number of cases (81 of 183 cases) occurred in the sixth decade of life (the age from 51 to 60); in the next greatest

The disease may be designated as not very common; only nerve specialists often see it; but these are the patients who congregate in almshouses.

Heredity plays a certain but by no means a prominent rôle; from a moderate number (about 16 per cent.) of the patients we hear that parents or grandparents suffered from the same affection; I know a family in which three or four sisters are afflicted with the disease.

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number the onset was in the fifth decade (43 cases); in the seventh decade (61–70 years) there were 35 cases; but even prior to the 41st year of life there were 22 cases, three of these occurring before the 31st year, the youngest being 28; after the 70th year there were only 2 cases, both men. Therefore two-thirds of all the cases (124 out of 183) occurred in the pre-senile period of life, their onset being between the 41st and 60th years.

Curiously enough, the earliest period for the onset of the disease, at least in men, was observed in the lower classes, and then in those in better circumstances; among 24 men seen in the Clinic 4 were attacked in the fourth decade, 10 in the fifth, 6 in the sixth, 3 in the seventh, and none in the eighth decade, while among men in the higher walks of life 46 cases occurred in the sixth, 27 in the seventh, and only 18 in the fifth decade. Therefore the average age at which the 105 men of the higher classes were attacked = 54.4 years; that of the 24 men in the Clinic = 46.8 years. This difference is not apparent among the women. Here the average age at which those in both categories (38 from the higher classes and 16 from the lower) were attacked is almost the same, 51.2 years.

In regard to individual etiologic factors my investigations show that *syphilis* plays no rôle; among 101 men from the better classes only 11 had previously had chancre; and 8 had well defined syphilis, therefore a total of 19 per cent.; that is, a smaller proportion than would otherwise be found among men in the better walks of life (22 to 24 per cent. according to my large statistical compilation). Among the 15 men from the lower classes, of whose cases a record was kept, none had syphilis. Among the 54 women there were no reports of this disease nor was any ever suspected of having syphilis. But in women such a history is of very little value unless the facts have been most minutely investigated.

I have endeavored to ascertain whether *direct heredity* (occurrence of tremor, that is, paralysis agitans, in parents, brothers, sisters, or other relatives) was present. Among 62 men there were 10 who positively reported this, among 31 women there were 5; therefore, among 93 cases there were 15, = about 16 per cent., in whom heredity was suspected; hence this factor appears to play no predominant rôle in the development of the disease.

The effect of *bodily trauma* (wounds, shock, fall, etc.) was cited as a cause in 9 cases. In some instances, however, *psychical trauma* was probably also a factor; this condition in a more *acute* form is mentioned nine times (as a severe, "fatal" fright, fear, extreme excitement, etc.), in a more *chronic* form (as prolonged anger, sorrow, care, excitement, etc.) in 30 cases (this being the total number of cases in some of which, however, accurate reports are lacking).

In 4 cases the affection is said to have developed after an attack of *influenza*, and this does not appear unlikely, considering our experience with influenza.

In regard to other etiologic conditions—refrigeration, abuse of alcohol and of tobacco—my notes reveal nothing of value.

I shall at once add to these statistical reports a few clinical observations as follows:

In 173 cases there were reports concerning the *localization of the symptoms at the onset* of the affection. These began with tremor and stiffness in the *upper* extremity (hand and arm) in not less than 137 (77 times upon the right, 60 upon the left); in the *lower* extremity in 25 (12 times upon the right, 13 upon the left); in a *more diffuse manner* upon both sides (according to the report of the patients) in 9 cases, and especially often with stiffness; and in the *bulbar* nerve tracts, in the lips, and in speech in 2 cases.

Cases with and *without* minimal tremor occurring late were found in not less than 37 of 183 cases, among these 28 times in men and 9 times in women.

Age is a predominating factor; paralysis agitans is certainly a disease of *advancing years* although not actually of the senile period; it occurs almost exclusively after the fortieth or the fiftieth year; I know of some cases which began at seventy or seventy-one, but the rule shows many exceptions. Sometimes the disease begins *prior* to the fiftieth, and not rarely even before the fortieth year; in most exceptional cases it may appear even in the twenties; I saw three cases of this kind, the youngest being 28 years of age. Other authors have seen cases at twenty-six, at twenty-one, and even beginning at nineteen and seventeen years; Lannois reports a typical case following rötheln at twelve years of age. It is questionable in these cases whether the diagnosis was correct, and this appears the more likely since we have recognized the *hysterical imitation of paralysis agitans.*

My own experience in 183 cases shows that 118 of these began after the fiftieth year (of these, two beyond the seventieth year), 43 between the forty-first and fiftieth years, in 19 cases between the thirty-first and fortieth years (see note on page 881). At least two-thirds of all cases occurred between the ages of forty-one and sixty, therefore certainly not in the senile period. Almost exactly the same proportion (70 per cent.) was given by v. Krafft-Ebing.

The disease does not occur, as was formerly believed, with about the same frequency in both sexes; the *number of men predominates* decidedly. My figures show that in the higher classes (143 cases) the proportion is about 3 to 1; among the lower classes (40 cases) 3 to 2, and in the total number it is about 5 to 2. Gowers found it 5 to 3, v. Krafft-Ebing, among 100 cases, 3 to 2.

Whether the disease is more frequent among Jews than among other Europeans, as might appear from v. Krafft-Ebing's figures, I am unable to state.

That *syphilis* plays no prominent rôle in the development of the affection is shown by my statistics; these prove that among 116 cases there were only 19 with a previous chancre or syphilis; therefore the number of cases with syphilis was less than the average proportion usually found among the male adult population; this is sufficient proof! Among women suffering from paralysis agitans syphilis could not be determined and, indeed, was hardly suspected.

Neither can a decided influence be attributed to *alcoholism.* This would naturally be more frequent and predominate most extensively in men. The very common alcoholic tremor differs essentially from the tremor of paralysis agitans.

Other factors mentioned as causes—refrigeration, damp dwellings, insanitary conditions of life, infectious diseases and the like—have by no means been positively proven, but perhaps influenza alone cannot be wholly excluded.

Numerous recent observations—in conformity with earlier reports—have demonstrated beyond doubt that *psychical* and *somatic traumata* may produce the disease.

It has been proven that sudden and profound psychical emotion, a *severe* and *acute psychical trauma,* severe "deadly" fright, fear, horror, and the like are prone to cause the affection; among my cases there were 9 such, of which I have described 2 in detail. Even more frequent are *chronic*

psychical conditions, such as prolonged sorrow, anger, anxiety, or excitement, which have preceded the disease; such psychical lesion was indicated by the patient in 30 of my cases, and numerous similar examples are found in literature.

On the other hand there appears to be no doubt that *bodily trauma* (injuries, shock, contusions, fractures, burns, etc.) are the precursors of paralysis agitans; Walz published a long article on this subject, v. Krafft-Ebing. v. Górski, Leva, Bychowski and others have made similar observations, and cases of this kind are increasingly frequent in the literature of accidents; in 9 of my cases bodily trauma was named as the cause. These traumata were in part of mild nature; but a few weeks or months later the first symptoms of the affection appeared—usually in the member attacked by the trauma. Since in most accidents fright and fear, therefore bodily and psychical trauma, act together, the latter not rarely predominating, it is difficult to decide which of the two has had the larger share in causing the disease. In any event it appears unquestionable that one as well as the other may produce paralysis agitans, and more readily when both causes act simultaneously.

In how far a certain *predisposition* to the affection is here operative, which is quite likely, cannot at present be determined.

According to Krafft-Ebing, *over-exertion*, surmenage local, is said to be the provocative cause in some cases.

Therefore, besides *advancing years* (which alone do not explain the condition since, otherwise, the disease would be much more frequent and would appear almost as a senile phenomenon) only *psychical and physical traumata* may be recognized as in any sense positively determined causes of paralysis agitans.

SYMPTOMATOLOGY

The symptoms of paralysis agitans have been so frequently and so accurately described in all of their individual features that we may here be content with a brief sketch outlining the most salient points. In the well developed cases these are so pregnant and so uniform that the disease is recognizable at the first glance, and it is somewhat remarkable that this was formerly for so long a time grouped or confounded with other diseases.

The name of the disease indicates that in the "tremor," the "shaking," the "agitation" of the limbs the most important symptom of the disease is found. A closer study of the malady shows, however, that besides the tremor another symptom is just as marked and of as great significance, and in connection with the tremor impresses upon the pathologic picture its characteristic stamp; this is a peculiar *muscular stiffness*, a rigidity, which reveals itself by a rigid posture and by a certain slowness and difficulty of movement which frequently annoys the patient more than the tremor; a symptom, moreover, which sometimes exists alone, the tremor being wholly absent.

These two symptoms, *tremor* and *muscular rigidity*, are the ones which first appear, are the most typical of the clinical picture, and clearly reveal the individual processes of the affection. They are always present although in different degree, sometimes uniformly, at other times one, and then the

other, being predominant or absent. From this various forms of the disease arise, with which we should become familiar.

Tremor.—The tremor for the most part begins *gradually*, almost invariably in the hand and unilaterally, more frequently upon the right side than the left (77 to 60), and is accompanied by feeble, quite rapid, yet scarcely noticeable oscillations (4 to 7 in a second), first in the fingers and thumb. Either from excitement or exhaustion, or apparently without cause, it is temporarily increased, the movements gradually become more marked, more extensive but slower; finally, actual tremor occurs, and continues in a mechanical manner for hours, sometimes without an interruption for an entire day; the finer and complicated finger movements imitate those made in rolling pills, in spinning, in counting money and the like, and depend upon alternate contractions of the small muscles of the hand and forearm. The movements toward the trunk become more feeble, they usually continue to be distinct at the wrist, but are absent in the elbow- and shoulder-joint or only appear there much later; sometimes they are less prominent in the fingers, and are observed particularly in the wrist.

After the hand the leg of the same side is attacked, then the foot less intensely but distinctly, and this is later so increased that a continuous unrest develops accompanied by a beating sound of the foot upon the floor. In relatively rare cases (25 in 173) the affection began in the foot, and subsequently attacked the arm of the same side; here also the toes may be especially implicated in the movement.

In two cases I first noted a bulbar localization—in the mouth and neck, and in the speech; in nine cases the affection—tremor or stiffness—was either diffused upon both sides or at once involved an entire extremity; therefore, any mode of onset is possible.

Usually the tremor is limited for a long time, even for years, to one side; then the other hand, subsequently the other leg, are attacked; sometimes the affection begins simultaneously in both hands.

Finally—but not in all cases—the neck and the head are implicated; not rarely the affection is distinctly visible in the muscles of the lips, chin, jaw, tongue, and throat. But it never reaches such an intensity as the tremor of the head in multiple sclerosis. In very rare cases the muscles of the larynx have been found implicated (Rosenberg, Fr. Müller).

The *intensity of the tremor* varies extraordinarily in different patients, and increases from scarcely perceptible oscillations to extreme shaking of the limbs; it varies with the moods and with the general condition of the patient, is augmented by excitement, exhaustion, and the like, and it is also increased and manifested by energetic movements first in one hand, and then appearing to a still greater extent in the other.

This tremor—a point especially characteristic—*now appears when the body is completely at rest*; it begins soon after awakening, is more or less violent during the day, and *ceases during sleep*; only in very advanced stages does it prevent sleep. *It is not increased by voluntary movements* (or but very rarely), *it is therefore absolutely not an intention tremor*; on the contrary, *it may be transitorily suppressed*—at least at the onset of the affection—*by voluntary movements*; this enables the patients to use their hands for an astonishingly long period for all the finer movements, as in eating,

writing, drawing, piano playing, dressing themselves, etc. Coarser movements are not at all impeded. Naturally, in advanced stages of the disease a decided disturbance of the power of motion appears.

Muscular Rigidity and Muscle Tension.—Much more frequently this is due to the other principal symptoms: MUSCULAR RIGIDITY and MUSCULAR TENSION; the majority of the muscles of the body are apparently in a condition of increased tonus or slight tension which may be objectively determined and is combined with a peculiar difficulty in rapidly and easily bringing the muscles into play; hence, there is a *brief delay,* or, rather, a *prolonged latency of voluntary stimulation.*

As a result of this peculiar disturbance in the muscles, we have the characteristic picture which most patients with paralysis agitans present: Their posture, their movements, their facial expression, their voice, and their speech.

The *rigidity of the muscles of the face* produces the serious, surly, or tearful facial expression; that of the muscles of the larynx and the organs of speech, the peculiar indistinctness of the speech and voice which often seems to vanish; that of the muscles of the arm or hand, the slight flexure of the elbow-joints, and the characteristic position of the hand "as if holding a pen"; that of the musculature of the trunk causes the bent posture, the difficulty experienced in rising suddenly from a chair, in turning rapidly, in turning in bed, or in quickly resuming the equilibrium of the body and altering its gravity. The rigidity of the muscles of the leg causes the *stiff, spastic gait,* the bent knee, and the dragging of the tip of the foot upon the floor.

The body is generally *bent* forward; the head droops, the back is markedly curved, the arms are slightly flexed at the elbows, the legs at the knee; in rare cases, however, a more extended, stiffer, and more erect posture is seen, the head and the vertebral column being flexed somewhat backward (so-called "*extension type*" in contrast with the ordinary "*flexion type*").

The much discussed symptoms, "*propulsion*" and "*retropulsion,*" depend mainly upon this muscular rigidity which was long regarded as a constrained movement. In walking the rigid legs of the patients seem to cling to the floor, the upper part of the body is propelled forward, and the patients "hang fire" but are alike unable to regain their equilibrium quickly, or to fall, or to go forward until they find an obstruction to sustain them, or until they finally regain the necessary command of the trunk (chorea festinans!); the same condition is noticed with retropulsion. If the patient who is quietly standing is drawn back and thus caused to walk backward, it is just as difficult to stop him as in walking forward, and for the same reasons.

On these two symptoms—the tremor and the muscular rigidity—the typical picture of paralysis agitans is based; it is recognizable at the first glance, for in most cases it reappears monotonously.

Of course, other more or less constant symptoms are observed: All kinds of *vasomotor disturbances, redness and heat of the face and head,* often combined with a *feeling of heat and burning of the skin,* with an increased secretion of sweat over the greater part of the body, or else a disagreeable sensation of cold; rarely there are other paresthesias of the skin, occasionally also *more or less severe pain in various parts of the body,* and, above all, a sensation of extreme *fatigue in the muscles;* finally there is a general restlessness

of the limbs, a desire for frequent changes of position which increases with the duration of the disease and becomes actual suffering on account of the helplessness of the patient; every quarter or half hour these unfortunates long for a change of position and are unable to effect it without help. They must be raised from chair or bed, be turned a countless number of times in bed, and thus become a torment to themselves and to their nurses.

Notwithstanding all this, the psychical functions and those of the special senses are absolutely undisturbed. [Mental activity and initiative are, as a rule, undoubtedly reduced. Parkinsonians are loath to undertake the consideration of any new subject or to actively pursue mental work formerly agreeable to them.—Ed.] Except for occasional pain and paresthesia, noteworthy sensory disturbances are absent; the sphincters are intact; aside from the tremor and stiffness gross power and motility remain for a long time and only gradually decrease; the vegetative functions are normal.

Consequently *objective examination* in the early stages of the disease—again excepting the tremor and stiffness with their sequelæ—reveals nothing. The motility, the gross power, appears quite normal except for the slowness on attempting movements; but for all muscular movements—for example, in dressing, in eating, and the like—the patients require an increasingly long time; the muscles do not atrophy, and the electric contractility of the motor apparatus remains normal; but muscular tension and stiffness may be objectively demonstrated, and usually are accompanied by a moderate increase of the tendon reflexes (up to a slight foot clonus); only very rarely do the tendon reflexes appear to be diminished (on account of muscle tension) and just as rarely are they markedly increased; as a number of recent investigations in a series of cases has taught me, the patella reflex is quite normal, and shows nothing of the pathologic condition described by Babinski—perhaps quite an important proof that the affection is primarily of functional origin.

In regard to the *cutaneous sensibility*, it is usually stated to be absolutely normal; however, some authors (Ordenstein, Eulenburg, Kornilowicz, Holm, v. Krafft-Ebing, Heimann, and others) have reported disturbances of sensibility in rare cases, and upon accurate examination Karplus recently found in a number of cases all the varieties of objective disturbances in sensation which he himself designates as "very varying and inconstant"; namely, hyperesthesias and hypalgesias, also thermal hyperesthesia partly associated with hyperalgesia, usually circumscribed and not limited to definite nerve tracts, but existing for a long time; they are most likely to be developed in the extremity or on the side of the body affected by the motor disturbance, but are never very extensive; in one case he found also a well developed bilateral hyperalgesia, in another a hemi-hypalgesia; Karplus believes that we may certainly assume from this a hysterical disturbance in sensibility. In many instances, however, in spite of preceding subjective pain and paresthesia, this objective disturbance in sensibility is absent.

It would add to the interest of our conception of the affection if these findings of Karplus could be confirmed.

Frenkel has lately described a peculiar *change in the skin*—a thickening and tense adherence to the subcutaneous layers—as a very constant symptom in paralysis agitans which he has utilized to explain some of the symptoms.

Karplus tested these reports with absolutely negative result, and for this reason questions the accuracy of Frenkel's statements.

The internal organs, as well as the sphincters, the organs of special sense, of ocular movements, the pupils, etc., are normal. There is no aphasia, but there is a certain weakness of the voice with a somewhat hasty, monotonous, "vanishing" utterance of sentences as though the patient were anxious to get them out and done with; this is especially prominent in the later stages, and with the gently trembling lips gives us a very peculiar impression.

The vegetative functions are not affected; the intelligence and memory of the patient appear unimpaired.

I have known such patients to carry on a large business, conducted properly in all respects, without difficulty; even a mild disposition and good humor may be long retained in spite of the severe and hopeless nature of the affection; but, of course, to this there are many exceptions.

This clinical picture will not be found uniformly developed in all cases; as has been already stated, even the two main symptoms, tremor and muscular rigidity, show the greatest possible variations; one or the other may be less prominent; this is especially true of the tremor, which chiefly impresses its clinical stamp upon the disease. While the rigidity shows a varying intensity, although it is rarely absent, the *tremor may be completely lacking* at the onset and in the further course for years, or it may be temporarily only slightly apparent, or may be so rudimentary that even after prolonged observation it is exceedingly difficult to perceive it, yet, in spite of this, the clinical picture may be well developed and unmistakable. These are not the infrequent cases of *Parkinson's disease without tremor*—of paralysis agitans *sine tremore* or *sine agitatione*—which are of especial importance since they may lead the inexperienced observer into most unpleasant diagnostic errors, and may direct the treatment into improper channels. Such cases are not rare. Among my 183 observations I find no less than 37—therefore about 20 per cent. In these instances the first symptom of the disease was a certain stiffness, awkwardness, and weakness in one or the other hand or in a leg; or the onset was more gradual with a sensation of fatigue, a paresthesia, difficulty in executing certain movements, the dragging of one leg, and other nervous symptoms. I have seen a number of such patients in whom either beginning disease of the brain or spinal cord, slight apoplexy, spastic spinal paralysis, brain tumors, or even neuroses of writing and the like appeared likely, yet in which, frequently at the first glance, or only upon prolonged and continuous observation, the diagnosis became clear. The further course, the final appearance of the tremor, the increasing characteristic habitus of the patient, the absence of other symptoms of the diseases in question, rendered the diagnosis positive. I must admit that before I became sufficiently familiar with the affection I made several such erroneous diagnoses, and therefore I desire to call especial attention to these "*formes fruste*" of *paralysis agitans*. They are readily recognized by the peculiar position of the hands and arms, the facial expression, the posture, the gait, and the voice of the patient.

The question arises whether the opposite—paralysis agitans without *stiffness and muscular rigidity*—exists. This is quite likely; certainly in some cases there is very slight rigidity. It appears to me doubtful whether the

absence of rigidity can be regarded as proof that the tremor closely resembling paralysis agitans is of hysterical origin. This is quite commonly assumed, but the condition should be more comprehensively studied.

COURSE

The disease is generally very protracted but uninterruptedly progressive; it usually lasts for many years, five, ten or fifteen, and sometimes even longer.

Tremor and rigidity increase; the former may pass into general trembling of great intensity and duration, so that the patients can no longer sit up and can no longer lie in bed, cannot divert their minds with anything, and can only be fed with extreme difficulty; the rigidity becomes so marked that the patients are absolutely helpless, constantly require aid, and must be given a change of position continually during day and night, being a source of great discomfort to themselves and others! In fact their condition is pitiable —a condition in which death alone can bring relief. Not rarely insomnia, psychical disturbances, irritative conditions, delusions of persecution, imbecility, senile and arterio-sclerotic dementia are added to complete the misery of this existence.

Provided intercurrent diseases (bronchitis, pneumonia, influenza, apoplexy and the like) do not early terminate the life of the patient, he finally succumbs miserably with bed-sores and general marasmus.

Some cases run an exceedingly rapid course toward such an unfortunate termination; only recently I saw a lady who, in the course of a single year, was reduced to this terrible plight; on the other hand, many remain for years and decades with only the initial symptoms of the affection, and perish before its more advanced stages are reached.

There is no recovery from this unfortunate disease. Reports of individual cures are not sufficiently trustworthy, and probably depend upon diagnostic errors (confusion with hysteria!); here and there the affection may be arrested, but very rarely does a transitory improvement occur; in the most favorable cases the disease runs a protracted course to its unavoidable end.

PATHOLOGY

The **anatomical foundation** of this severe and incurable affection has recently been investigated countless numbers of times by our improved modern methods, but, unfortunately, with but little success! We must still admit that *we do not know the positive and constant pathologico-anatomical foundation of paralysis agitans.*

I must, therefore, decline to give a detailed description or to criticise the anatomical findings which have been reported by various authors, and shall be content with a brief sketch. A number of expert observers have attempted this task with all of the aids of our modern technic; in contrast to the former almost *negative* findings, modern investigators have almost invariably reported *positive* findings in the brain as well as in the spinal cord, in the peripheral nerves and even in the muscles (Borgherini, Koller, v. Sass, Ketscher, Hunt, Dana, Redlich, Sander, Nonne, Wollenberg, Karplus and

others), and most of these are inclined to regard their findings as of some significance in the pathological anatomy of paralysis agitans. That other competent observers, such as Oppenheim and Fürstner, report entirely negative results in undoubted cases by the same methods robs these opinions of much of their weight.

What has been found is about as follows: In the *brain* dilatation and thickening of the vessels, connective tissue increase, diffuse and insular proliferation of the glia, atrophic and degenerative changes in the nerve fibers and ganglion cells, vacuole formation, pigment degeneration, nuclear disappearance and partial destruction of the fine dendrite branchings of the latter; according to Nissl the same changes have been found with remarkable frequency in the spinal cord, especially in the large ganglion cells of the anterior columns, also in the *peripheral nerves*; occasionally proliferation of the neurilemma, degenerative changes in the nerve fibers, and slight fatty degeneration of the muscular fibers have been reported (even in the motor end plates, Dana) besides thickening of the perimysium and of the vessels: usually, however, the muscles were found to be normal.

Most authors regard these changes as by no means specific or characteristic; but, on the contrary, they consider their quality to be identical with the senile changes invariably found in the nervous system of the aged; quantitatively, perhaps also by their special localization in paralysis agitans, they chiefly differ from the alterations in the aged; therefore these changes are the chief expression of *an abnormal, extreme, and early senility of the nervous system.*

But the fact alone that paralysis agitans is frequently, and even as a rule, observed at a period of life when no other signs of senility are noted, which period might at most be designated as presenile, also the circumstance that the disease would necessarily appear much more frequently and invariably in elderly persons, that senile tremor, and in fact the usual signs of advancing age, are by no means identical with the clinical picture of paralysis agitans, and, finally, that the most marked senile changes in the nervous system may be present without paralysis agitans, absolutely undermines the support of this view and renders it most unlikely that the alterations found are essential features of paralysis agitans.

The circumstance that the malady apparently possesses no characteristic localization, that it is irregularly disseminated throughout the entire central (and even peripheral) nervous system, that the lesions are by no means strictly confined to the motor apparatus (motor neurons I and II), as might be expected from a symptom-complex limited almost exclusively to the motor apparatus, is still more in opposition to this explanation of the anatomical finding.

In the anatomical findings, therefore, we recognize nothing distinctive: they probably belong only to the terminal stages of a process going on for many years, the onset of which was unnoticed. It does not appear strange that in muscles which have been immoderately active for years (Dana calculated that a muscle examined by him had undergone not less than 840 million contractions during the continuance of the disease for eight years!), "slight fatty degeneration" and some connective tissue increase should be found; or if, in the peripheral nervous system and the spinal ganglion cells

of persons who have been subjected to frequent irritations, who have suffered for years or have finally succumbed to marasmus or other fatal acute disease, such degenerative changes are found as are revealed by Nissl's method under varying conditions, or if we find in the brain, under the same circumstances, vascular degeneration, glia proliferation, and degeneration of the nervous elements.

For the present these reasons permit us to conclude that the actual, unquestionable, and invariable anatomical lesions of paralysis agitans have not yet been found; perhaps in the pathologic changes mentioned above there are important conditions, but these are not positive.

Thus, pathological anatomy leaves us in absolute ignorance of the *actual localization* of the affection, and the solution of the question must be attempted from a clinical standpoint.

It has sometimes been thought that the *muscles* themselves might be the seat of the affection; in my opinion everything militates against this view—chiefly because of the reasons which cause us to locate the disease in the central nervous system, and because there are no facts which make it likely that such a peculiar spasm of the muscles could occur without an implication of the nervous system. If the report, lately promulgated, of the frequent occurrence of sensory disturbances in paralysis agitans is actually confirmed, this view will be shattered.

The same is true of the *peripheral nerves*; we have no knowledge of a disease of the peripheral nervous system in the symptom-picture of which there is even occasionally the slightest syndrome resembling paralysis agitans aside from the fact that this is not localized in individual nerve trunks but, quite differently (analogous to affections due to central causes), in motor as well as sensory regions.

Implication of the *spinal cord* might much more readily be thought of; from the earliest times anatomical changes in the spinal cord in this disease have been sought and were supposed to have been found; this is explained by the fact that the spinal cord can be investigated with more facility, and that the brain has often been neglected in the examination; the entirely negative findings, mentioned previously, would be opposed to such localization provided we recognize an anatomical lesion to be the determining factor. By analogy, one of the symptoms of paralysis agitans—muscular tension and rigidity—could be clinically localized in the spinal cord and in the pyramidal tracts; but there is no marked increase of the tendon reflexes, and the normal plantar reflex controverts this theory; a functional irritation of the pyramidal tracts might be conceived, but this might also originate from the brain. For the localization of the tremor in the spinal cord (gray anterior columns?) we possess no analogy; we know of no spinal cord affection, whatsoever its name or seat, in which primarily or secondarily the tremor of paralysis agitans or the entire syndrome occasionally appears.

On the contrary, everything in the clinical picture indicates that the *original seat of the affection is the brain*; for instance, the fact that a unilateral paralysis agitans has sometimes been found associated with coarse local brain lesions (tumors in the thalamus and cerebral peduncles, sclerosis of the hippocampus major), etc.; that a posthemiplegic paralysis agitans (analogous to chorea and athetosis, posthemiplegia) is occasionally observed;

that the affection so frequently occurs in consequence of psychical trauma; that it almost always appears unilaterally; finally, its remarkable resemblance to the symptom-complex in the most common form of hemiplegia (after apoplexy in the region of the large central ganglia and in the internal capsule). In this condition we find paralysis, contractures, and increased tendon reflexes, most marked in the arm, less so in the leg, and most feeble in the head and the face; in paralysis agitans, however, we note the same intensity of the tremor, the same rigidity and muscular tension, the same increase of the tendon reflexes! The posture and gait of the side first affected so closely resemble those of hemiplegia that cases without tremor can sometimes with difficulty be distinguished, and for this reason are frequently confounded with cerebral affections. The terminal stages of the disease, the mental deterioration, the not infrequent psychopathic complications are also in favor of this view.

Naturally this does not give us an exact localization of the process within the brain. Whether its course runs directly in the path of the cortico-spinal motor neuron or in its collateral associated tracts, whether in the motor cortical region, in the neuron cells or the axon bundles, in the corona radiata, in the internal capsule or in the central ganglia of the brain-stem—who can decide? At all events speculation is futile; it is, however, certain that at the onset of the affection there is no severe or coarse lesion in the motor apparatus, for with such a condition the unimpaired motility would not be compatible; we must therefore think primarily of finer, nutritive, molecular, or chemical changes which may subsequently cause structural lesions and profound nutritive disturbances.

If the sensory disorders which appear should prove constant, a more diffuse process must be considered; the conditions here are analogous to those of tetany, which was also considered to be a pure motor neurosis until J. Hoffmann discovered changes in the sensory nerves and those of special sense. This furnishes a clue and guide to any one capable of investigating the *actual nature of the malady.*

But of this we are still absolutely ignorant. We do not recognize the true foundation nor the nature of the disease from the anatomical changes; even the obvious and very plausible hypothesis that the affection is a premature and extreme senility of the nervous system has been rejected for important reasons previously stated. Modern experience and opinions in regard to many endogenous toxic effects of glandular origin—I refer particularly to Graves' disease, myxedema, tetany, Addison's disease, and similar ones—may lead us, and in fact have done so, to regard paralysis agitans as a glandular autointoxication little as we can reconcile this view with the usual psychic and traumatic origin of the disease. But this very interesting theory is scarcely more than a possibility; no definite facts support it, and therapy based upon this idea (Dana) has been entirely without result.

The hypotheses of exogenous, infectious, rheumatoid or simple toxic action, which, after a long time, exert a destructive power upon the nervous elements only (perhaps with advancing age or other deleterious condition) are at this time visionary.

To develop these hypotheses further would be futile; I coincide in the opinion of Jolly, v. Krafft-Ebing, Wollenberg and others that at this time

we must accept *the view of an originally functional, fine, nutritive, molecular disturbance* principally localized in the motor apparatus, but probably also in the central nervous system. That during the long course of the malady this derangement may finally lead to visible nutritive disturbances, to atrophy and degeneration of the nervous elements and their surroundings is not remarkable, and finds its analogy in other diseases of the nervous system (commotion, traumatic neuroses, some psychoses, etc.).

How these molecular disturbances arise, how they are produced by the causes of the disease, is a complete mystery; paralysis agitans is, in the main, a rare disease. The etiologic factors said to be its cause, advanced age, psychical and somatic traumata, emotional conditions, refrigeration and the like, are so common and so generally existent that in the persons who are attacked by paralysis agitans there must still be an unknown factor, an existing tendency or endogenous peculiarity which predisposes them to this disease. The disease is not particularly apt to be associated with hereditary conditions, nor with a special or general neuropathic predisposition; the processes of involution both in the male and female, vascular degeneration and perivascular proliferations which have been thought of, appear to have but little significance. How, and during what period of life this predisposition arises is therefore still a mooted question.

DIAGNOSIS

In the great majority of cases, even in their first stages, the *diagnosis* of the affection is very easy. In the developed cases the picture is so typical and characteristic that the malady is recognizable at the first glance—even when no tremor is present; usually, when the patient enters the room, a correct "instantaneous diagnosis" may be more easily and positively made than in any other disease; I need only refer to the preceding description.

As a rule, the typical cases with tremor may readily be differentiated from other forms of tremor, particularly from the toxic forms (alcohol, coffee, absinthe, lead, mercury, etc.), by the quality of the tremor and the simultaneous muscular rigidity with its characteristic sequelæ apart from the recognizable etiology; also from *neurasthenic tremor* which is of dissimilar nature and unlike in appearance; from the *tremor in Graves' disease* (by its different mode of appearance and accompanying symptoms); from tetany, etc. The differential diagnosis of even senile tremor, as a rule, causes no perplexity. It is different in nature, form, and localization, and the other symptoms of paralysis agitans, the vasomotor disturbances, the rigidity, the typical posture, the voice, the facial expression, etc., are absent; nevertheless a senile tremor may sometimes resemble that of paralysis agitans (transitional forms?). Since the classic researches of Charcot's adherents, the *intention tremor of multiple sclerosis* can no longer be confounded with the tremor of paralysis agitans, and it now appears scarcely worthy of discussion; nevertheless the differential factors may be briefly enumerated! These are embodied in the nature and manifestation of the tremor. In paralysis agitans the tremor comes on during rest and ceases with voluntary movement—at least transitorily; in multiple sclerosis it is absent during rest and appears upon voluntary movement, increases the more the greater the effort to make

exact movements and to near the goal; in paralysis agitans it is fine and rapid, appearing with the effort at complicated movements as imitative tremor of the hands and fingers, while in the trunk, the neck and head it is absent or only slightly noticeable; in multiple sclerosis it is a coarser, slower tremor, with marked excursions, being especially prominent in the trunk and in the head. Although there are cases of advanced paralysis agitans in which the tremor becomes coarser, "shaking" comes on and even continues with voluntary movement, and although multiple sclerosis occasionally causes a tremor resembling paralysis agitans, these diseases differ so markedly from each other, and their other symptoms are so pronounced that confusion is hardly possible. Paralysis agitans is a disease of advanced age; besides the tremor, it primarily presents only the symptoms of muscular rigidity and slowed movements with their typical consequences; multiple sclerosis occurs chiefly in youthful and middle-aged persons, and, besides the tremor, shows a number of other symptoms (headache, nystagmus, scanning speech, spastic paresis of the legs, sensory disturbances, affections of the bladder, ataxia, optic atrophy, psychical anomalies, etc.) which are completely foreign to paralysis agitans; these readily permit differentiation. [Exceptional cases of paralysis agitans present a distinct intention tremor in early stages of the disease, and tremor in the limbs apparently uninvolved may frequently be demonstrated by directing the execution of somewhat difficult movements, such as extracting the contents of an inside breast pocket, reaching behind the back to the interscapular area, etc.—Ed.]

The differentiation from certain forms of tremor in *hysteria* is sometimes difficult. It appears that hysteria may actually "imitate" paralysis agitans. Such cases have recently been studied and described by Ormerod, Greidenberg, Rendu, Oppenheim, Dutil, and Béchet, and, finally, by v. Krafft-Ebing. Indeed, an astonishing resemblance to paralysis agitans is found. Only minute observation of such cases reveals that we are dealing with hysteria. Hysterical stigmata are present although not invariably (characteristic sensory disturbances, attacks, ovaralgia, anesthesia of the pharynx, etc.), the tremor often appears suddenly, is widely distributed after a psychical trauma, presents great variations, may disappear entirely, and does not increase but remains stationary for years; it is more polymorphic, and extends rather to the proximal part of the limbs, is not checked by the will, but rather increased, as also by excitement; on the other hand, typical muscular rigidity and slowing of movements are absent as well as the characteristic posture, the position of the hand as if holding a pen, the facial expression, the gait, etc., so that usually a differentiation is still possible. Nevertheless, many cases of this kind and others of hysterical origin are obscure and cannot be accurately classified.

More difficult—at least at the onset—is the recognition of the cases without *tremor*; here great perplexity arises as to the diagnosis, and errors are prone to occur. Nevertheless, if we bear in mind the occurrence of paralysis agitans without tremor, these forms also, as a rule, will be distinguished by the characteristic posture, the habitus and facial expression of the patient, and occasionally slight tremor will be subsequently discovered. Moreover, the symptoms of those diseases which come into question (usually of the brain on account of their unilateral development!) will by no means be so marked

as to remove at once all doubts. But a considerable time may occasionally elapse before we are sure of the diagnosis; advanced cases, even those *without tremor,* may be correctly diagnosticated at the first glance.

PROGNOSIS

The prognosis, as is evident from what I have stated, of the course of the disease, is hopeless. According to universal experience, the affection is progressive and incurable. It is true this advance is often extremely slow, and its occasional arrest and transitory improvement not rarely afford the patient a certain degree of comfort. In the individual case a relatively favorable prognosis can only be given after prolonged observation; that is, we may perhaps assume that the patient will very gradually and with comparatively little suffering approach the inevitable termination of the disease; other cases, however, may sometimes pursue a rapid course and terminate fatally in one or two years.

The few isolated reports of *cure* must be regarded with extreme skepticism, and they probably depend upon erroneous diagnoses, especially upon confusion with hysterical forms of tremor; nevertheless, I think we should not entirely exclude the *possibility* of a cure, particularly in young persons.

TREATMENT

Considering the prognosis, we can attain no brilliant results in the *treatment* of this distressing affection; nevertheless it may be said that, notwithstanding the incurability of the disease, we may in mild cases retard the progress of the malady and even arrest its course, and in severe cases the sufferings may be alleviated and existence rendered tolerable, at least for years.

In an experience of many years, a number of measures were shown to be efficacious; these I published in 1898, and I can add but little to these to-day.

As a rule, a *causal indication* cannot be followed; heredity cannot be removed, psychical and physical traumata cannot be prevented, sorrow cannot be assuaged nor care lessened, and other causes, as a rule, are unknown.

We must, therefore, primarily fulfil the *indicatio morbi* by combating the disease itself.

In the first place we must strictly regulate the patients' *diet and entire mode of life.* They must be moderate in eating and drinking, must subsist upon simple, nourishing, but varied food, must take little alcohol, no strong tea or coffee, and must not smoke to excess; cold sponge baths, tepid ablutions, and lukewarm baths are beneficial; all mental exertion and excitement, everything which strains or fatigues the nervous system, should be avoided; moderate outdoor exercise, frequently repeated during the day and for as long a time as possible is advisable, also moderate indoor gymnastics and mild massage; in the summer an outdoor life and in the winter a residence in the South is highly desirable.

There are three remedies from which I have frequently seen favorable results: First, *arsenic,* which is unquestionably a nervine and alterative of

the first rank, and in some neuroses (chorea, tic, Graves' disease, etc.) has produced brilliant results. Its favorable influence upon hematopoiesis, metabolism, the nutrition of the skin, the nutrition and function of the nervous system is unquestioned, and it has also been frequently employed in paralysis agitans; it was first advised as a remedy for tremor in general, but it is probably more effective as a nervine and tonic. The form in which it is to be administered appears to be immaterial, whether as arsenious acid, in solution, in pills, granules, or as Fowler's solution; whether subcutaneously with all necessary precautions, or in any combination, is immaterial. I usually give Fowler's solution combined with equal parts of aqua foenic. and tincture nux vomica (6 to 15 drops three times daily), and find that this mixture is well borne by the stomach.

Lately I have often employed *sodium cacodylate* which permits the introduction of arsenious acid in organic combination and in relatively large doses without disagreeable consequences; it is best given subcutaneously (0.05 gram daily with occasional pauses) or internally either in solution or in granules; the preparation used must always be fresh.

Electricity has also been employed in various ways; I have most confidence in *bipolar faradic baths* of indifferent temperature and moderate intensity continued from 10 to 15 minutes, and these I order three times a week; I have little actual experience as to the value of galvanic baths, but they have been advised by different authors. Alternating with the baths I usually employ *galvanization* of the head, of the back of the neck, and of the sympathetic, as well as of the back and of the extremities, and, it appears to me, with some degree of success.

From my large experience, I advise as a third remedy *mild hydrotherapy*, best when combined with forest and mountain air; everything that can prove exhausting is to be avoided, and only full lukewarm baths, gradually passing to cooler half baths with slight friction and affusions, partial washings, etc., are permissible. These measures are usually very acceptable and refreshing to most patients.

The cooler, indifferent spas (Johannisbad, Schlangenbad, Ragaz, Wildbad, etc.) have a similar effect, but we must not exceed bath temperatures of 33.5° C., since, as a rule, at higher temperatures the springs have an unfavorable effect upon the patient.

The combined or alternate employment of these three remedies may be advised; but it is evident that, besides these, occasionally other nervines and tonics (iron, quinin, strychnin, glycero-phosphates, silver nitrate, bismuth, valerian, etc.) may be employed. [Persistent massage, combined with full passive movements and active but gentle exercises, may do much to liberate the contractured postures, reestablish a normal gait, and retard the disabling physical features of the disease. A faithful, intelligent and tactful massage operator is essential to success. Passive exercises by means of the Zander apparatus are also useful.—Ed.]

Symptomatic treatment which has for its object the amelioration of the distressing symptoms of the disease—the tremor and the muscular rigidity—is of paramount importance. That this can only temporarily relieve, the affection being incurable, does not diminish the value of these palliative remedies, and among them we possess some which are often satisfactory and sometimes

brilliantly successful. Chief among these is *hyoscin,* which was first employed by me; this is best given in the form of hyoscin hydrobromate, but only in a fresh and reliable preparation. It should be given subcutaneously, and is exceedingly effective in small doses; it decreases the tremor, the restlessness of the limbs, and the rigidity for a number of hours, and makes life bearable to the suffering patient. I have seen most astonishing results from this, and have continued the use of the remedy for years without its losing its effect; an increase in the dose was sometimes necessary, but only very rarely were there any disagreeable secondary effects; the remedy was seldom ineffective, but this is sometimes the case, and many patients cannot take it at all.

The dose is calculated according to *decimilligrams*—hence we must be *cautious with our prescription!*—and two to four decimilligrams injected once or twice daily are sufficient; sometimes I give the full dose in the morning and one-half as much in the evening which has an excellent effect upon the tremor and induces sleep.

The remedy may also be given internally in pill form, but larger doses are then necessitated, and the action is not quite so certain and prompt.

Duboisin, recommended by Mendel as less dangerous and quite as effective, acts similarly to hyoscin. But in my experience this is the case only when it is given in large doses (6 to 12 decimilligrams daily divided into three doses); it occasionally produces unpleasant secondary effects. Under any circumstances we possess in these two remedies excellent palliatives in paralysis agitans, and Oppenheim's opposition to their use (Text-Book of Nervous Diseases) appears to me as incomprehensible as unwarranted.

Many other palliatives have been advised and extolled; the salts of bromin, atropin, hyoscyamin, ergotin, strychnin, veratrin, valerian, cannabis indica, etc., deserve little confidence, and this applies also to the narcotics and hypnotics which, of course, cannot be entirely dispensed with in the distressing terminal stages of the affection.

Organotherapy, which according to the hypotheses previously mentioned might perhaps be useful, has been variously tried, most thoroughly it appears by Dana; he used the thyreoid, the thymus, the hypophysis, the braïn, the testicle and adrenal extracts in tablet form. The results were absolutely negative except with thyreoiodin—which aggravated the disease! Nevertheless, I do not regard these results as conclusive.

"*Vibration therapy,*" which was first employed by Charcot after patients had reported to him the favorable and palliative effects of a railroad journey, has been carried out partly by means of a so-called "tremor chair," partly with a head mask which was caused to oscillate by means of an electro-motor; I mention this merely to add that it is of no real therapeutic importance; the same is true of *nerve stretching* and *suspension* which have occasionally been resorted to here and there; the results at best are very questionable.

In conclusion I believe I have shown that notwithstanding our advancing clinical knowledge of this peculiar and severe affection, our recognition of its nature and pathogenesis is still incomplete, and our attempts at treatment and cure are still exceedingly unsatisfactory. It is to be hoped that in the present century more fortunate results may be achieved.

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ATHETOSIS

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This *disease*, or—more correctly—the *symptom athetosis*, was described in 1871 by Hammond.

We understand by the term an abnormal and peculiar movement, independent of the will, and usually appearing in the extremities, the upper being chiefly implicated, the lower to a less extent; the phenomena are always most conspicuous in the distal ends. These conditions are also noted in the muscles of the face, in rare cases the palate and tongue being involved; the contractions are usually unilateral, occasionally bilateral.

The condition belongs to the group of true cerebral diseases which occur chiefly during youth. In a few cases, like symptoms have been observed in patients with disease of the spinal cord; and in a few obscure cases the necropsy was negative. The following is the history of a typical case:

In a *girl, aged 6*, this remarkable symptom-complex revealed the most common type of the disease. The mother related a history, usual in such cases, of hereditary predisposition and of injuries during birth; there was no report of hereditary syphilis; she stated that the child was normal at birth and was well and strong. At the age of twelve months, without assignable cause, it was attacked with fever, preceded by chills; the physician was unable to make a positive diagnosis. After a few days the child entirely recovered; in the course of time, however, the mother noted that the right extremity was but little and imperfectly used; some weeks later the movements slightly improved. But a certain stiffness of the right extremity was noticed, and, gradually, peculiar movements in the right hand. When the child began to walk it dragged the leg; now and then the mother observed that the toes contracted. Upon close questioning it was learned that in the child's second year there were now and then epileptic attacks, she did not develop well mentally, and now distinctly showed psychical defects. The little patient was said to have no pain. It was late before she learned to control micturition. When examined, however, there were neither bladder nor rectal disturbances. Examination revealed a slight weakness of the right facial nerve supplying the mouth, and a little deviation of the tongue to the right, the other cranial nerves being normal. The child's psychical development appeared to be greatly retarded, but speech was normal. The right side showed distinct paresis, most marked in the right upper extremity. In walking the right leg was dragged. In this member as well as in the upper extremity there was slight atrophy and moderate contracture with distinct increase of the reflexes; sensation was normal.

We have here the familiar picture of spastic hemiplegia which often follows the febrile diseases of infancy, and the cause of which, according to Benedikt, Vizioli, Strümpell and others, is usually *encephalitis*.

Following this description of the preceding condition, we now turn to the main symptom, the peculiar movements which were conspicuous in the child at the onset

of the disease. We noted that the upper arm was adducted, the elbow-joint was peculiarly and slowly flexed, and extensor movements alternated. At the shoulder-joint the upper arm sometimes changed its position from adduction to abduction; now and then this was quite marked. In the intervals there were slight movements of pronation and supination in the elbow-joint.

The wrist-joint was usually hyperflexed, frequently with a tendency to a radial position. It was markedly resistant to passive movement; occasionally the position changed, but not to a great extent. The disturbance, however, had its principal seat in the hand, the fingers of which frequently were more or less flexed; the thumb was often tucked slightly under the fingers. In these abnormal movements the small muscles of the hand were almost invariably implicated, but the long finger muscles were rarely at rest. Sometimes the fingers were distally flexed, the thumb opposed, the patient unsuccessfully attempting to extend the phalanges. When I tried to bring about extension passively, I succeeded only by exerting considerable force. After the lapse of scarcely a minute, the rigidity ceased, the fingers were slowly extended, but such extreme hyperextension of the finger appeared as a normal person could scarcely bring about voluntarily.

During passive motion, a surprising condition was discovered in the fingers. The phalangeal joints were hypermotile, and resembled those of articulated skeletons in which the joints are substituted by rubber bands. It was also noted that the finger movements were not always uniform, the unrest being sometimes more marked in one, sometimes in two or three fingers, while at times all moved in one direction, sometimes in the opposite. Occasionally there was complete extension, at other times hyperextension of the basal phalanges with flexion of the terminal phalanges, sometimes the inverse. Now and then the fingers were separated, then adducted—all this with a sluggishness which sometimes simulated voluntary movement. These movements have been somewhat aptly compared to the motions of the tentacles of a polyp. From time to time there were intermissions of varying length, and during these pauses the little patient could perform voluntary movements; it was stated that no abnormal movements occurred at night.

If the foot was examined while in a position of equino varus, it appeared at first to be motionless; but on prolonged observation, and occurring independently of the will, there was slow dorsal and plantar flexion of the toes, occasionally of other joints; these movements were less energetic than those in the hand.

This is the *type* of movements the *symptom-complex* which we designate "*athetosis,*" and we now ask, *How do these abnormal movements due to cerebral disease* differ from other similar conditions?

Among motor irritative phenomena we recognize the following: *Hemitremor, hemi-paralysis, agitans, hemi-chorea, and tic.* It is needless to enter into a differentio-diagnostic discussion of tic and tremor, for these conditions bear no actual resemblance to each other. On the other hand, we must give some consideration to the symptom of "*choreic contraction.*" This explanation is necessary since choreiform movements are not rarely an accompaniment of the spastic hemiplegia of infancy.

A glance at the following table, constructed by v. Monakow after that of Greidenberg, will show us the various points in the differential diagnosis of hemichorea and hemiathetosis:

Hemichorea.	Hemiathetosis.
1. Face, trunk and all extremities are attacked; in any case, all extremities.	1. The forearm and hand (fingers), as well as the lower leg and foot (toes) are chiefly or solely attacked; the other parts of the body show slight paresis with increased muscular tension.

2. The movements are active and manifold, they resemble tremor, are more marked with intended movements, and are purposeless. After short stimulation there is a brief transitory relaxation of the muscles.	2. The movements are slow, rhythmical, and hyperextensile; there is elastic resistance; the contortions appear to be voluntary; the joints are fixed, although with varying intensity, during the movements.
3. Contracture is often absent; frequently the limbs are flaccid. No deformities are noted in the joints.	3. Contracture, i. e., involuntary fixation of the joint, is common, but is subject to continuous change; there are deformities in the joints.
4. Hemianesthesia common.	4. Hemianesthesia rare.
5. Rest during sleep.	5. Rest during sleep not invariable.
6. Intended movements and attempts to suppress the unrest increase the tremor.	6. The movements are limited by the will to only a slight extent.

If these points are borne in mind the typical case just described must be considered as athetosis. But we do not maintain that such a differentiation is always possible. Sometimes there are similar conditions which one observer regards as athetosis, and another, perhaps, as chorea. Literature furnishes examples of cases in which there were choreic movements as well as athetosis, also of cases in which athetosis passed into chorea, or *vice versa.* The transitions are so gradual that, in the localization theory which will later be described, almost all authors who have written upon this subject fail to regard the necropsy findings of these conditions distinctive.

In the question of localization, another condition has repeatedly been studied in disease of the brain in adults; this is manifested by the peculiar movements usually seen in one extremity alone, but which by their slowness, their awkwardness, and their intermissions, closely resemble athetosis; they differ, however, by giving us the impression of an intended movement, by being sometimes just as marked (or even more so) in the shoulder- or elbow-joint as in the wrist-joint, as well as because there is no hyper-flaccidity of the finger-joints. Here we are dealing with *a condition midway between athetosis and a sort of constrained movement,* a variety of constrained movement which has nothing in common with that occasionally observed in severe neuropathic conditions in consequence of constrained ideas.

This description exhausts the main symptoms of the affection; but we must consider some peculiarities not observed in our typical case. An important point is the distribution. In some cases other muscle groups are affected, most commonly *the muscles of the face* (although this is rare).

In well marked cases we observe a continuous, peculiar, sluggish, and grotesque muscular contraction of the face, sometimes a tonic rigidity appears for a short time and then gradually disappears. In these cases the branches of the facial nerve which supply the mouth are chiefly affected; more rarely the other branches. Rare as these symptoms are in the *well developed forms,* there is some indication of them in most cases. Then we note a slight contraction, a slight drawing of the muscles to the affected side. The slow contraction of the tongue, of the hard palate, or of the platysma is exceedingly rare.

So far we have spoken only of unilateral symptoms. We must not, however, fail to mention that there are also *bilateral cases.* In some the extremities only are affected, in others also the muscles of the face. I accidentally

saw such a case in a Greek street beggar in Patras. The symptoms were very peculiar. The upper extremities moved continuously, assumed the strangest positions, and marked contortions of the face occurred now on one side, now on the other, sometimes on both. Occasionally there were brief tonic spasms which gave to the face an almost frightful expression.

PATHOLOGICAL ANATOMY

In discussing the pathologic anatomy, we find that in many of these cases there is a bilateral *cerebral affection*; some of these belong to the group of obscure "idiopathic" athetoses without anatomical finding.

Among the *other symptoms of athetosis,* we must mention *pain,* from which some of the patients suffer more or less severely. As a rule, any symptoms may appear which correspond to the underlying condition. For instance, in infantile athetosis there may be hypertrophy of the muscles, paralysis of the various cranial nerves, psychical anomalies, epileptic spasms, disturbance of the reflexes, contractures, etc. Naturally these disorders vary according to the underlying affection. They differ greatly in adults when they are the result of an apoplectic disease of the brain (particularly hemorrhage, softening, embolism); they differ also in tumor, in progressive paralysis, in meningitis, and in abscess.

This consideration leads us to the more minute *specialization of the conditions which cause athetosis*; and we have repeatedly mentioned that they are chiefly associated with the *spastic hemiplegia of infancy.* Athetosis with congenital paralysis may appear immediately after birth; when paralysis develops subsequently it rarely appears at once, but usually after weeks or months, and when the paralysis has partially or almost wholly disappeared, when the contracture has become more or less distinct, and when the reflexes are increased. In very rare cases athetosis alone, not actual paralysis, can be determined.

PATHOLOGY

The *pathologico-anatomical findings* in infantile hemiplegia are only revealed to us by cases which originated in a distant antecedent period; recent cases rarely terminate fatally. In the former we find foci of softening, cysts, defects in structure, porencephalia (Kundrat), microgyria, or anomalies in the cerebral membranes; in other cases there may be diffuse sclerosis. The initial affection may be due to embolism, and sometimes, especially in children with fatty degeneration of the arteries, to hemorrhage and thrombosis. We have already spoken of encephalitis which is in many cases supposed to be the cause. It is probable that pre-natal defects of development play a rôle in the forms acquired in intra-uterine life.

Athetosis after apoplectic hemiplegia is extremely rare in adults. Here the causes are the same as in the sudden hemiplegias of adults: Thrombosis, embolism, and, more rarely, a rupture of the vessels due to various etiologic causes.

According to Gowers, hemianesthesia is an accompanying symptom in the majority of cases. After the twentieth year, however, this symptom is rarely associated with the disease. I have seen it but twice in adult hemiplegia,

once in the case of a merchant, aged 24, who as a young man used alcohol to excess and was syphilitic; suddenly he became unconscious, and on awaking there was a right-sided paralysis which subsequently somewhat improved; gradually typical athetosis developed in the right hand. The second case was that of a laborer from Jerusalem, aged 24, who suffered from mitral insufficiency, and presented a clinical picture similar to that of the preceding case; this was apparently due to embolism.

In the scale of frequency, *tumors* follow the affections which have been described. I possess microscopic preparations of a brain tumor which produced similar symptoms. It is true the case was not one of typical athetosis, but it had exhibited the previously described peculiar, constrained movements. It was the case of a woman, aged 62, who was under my observation in the Clinic of Professor Nothnagel from the 3d to the 24th of January, 1903.

The patient had measles when a child, and in her twenty-fifth year articular rheumatism. In the beginning of November, 1902, she lost appetite, was occasionally dyspneic, and obviously became weaker and more debilitated. About the middle of December there was a peculiar paresthesia in the right hand, gradually a certain degree of unrest became noticeable, and objects fell from her hand; this unrest soon became so great that the patient was unable to hold anything still. The right upper and lower extremities steadily became weaker. Later the patient suffered from vertigo, which confined her to bed. There was no vomiting, no loss of speech nor of consciousness, no bladder or rectal disturbances, and no symptoms of alcohol or syphilis.

When examined upon the 5th of January, the patient seemed to be well informed but very talkative, complaining much, and constantly making grimaces. She drew up her forehead, blinked her eyes, contorted her mouth, and periodically produced a laughing expression, chiefly by overaction of the muscles in the right side of the face. The tongue was incessantly moved about. The branch of the right facial supplying the mouth was less active than the left. The pupils were of equal size and reacted sluggishly; the eye-ground was normal. The speech was somewhat indistinct, and had a nasal tone.

The extremities were freely movable, both actively and passively (particularly upon the right side), but were weak. While the left side was absolutely quiet, the right upper extremity was in constant motion. All of the large joints were involved in this motion. The hand was raised, extended, flexed, or rotated, the movements showing no purpose and being rapid, after which the extremity would again become quiet and return to its original position. On any attempt to suppress the movements, they were increased. Upon intended movement a slight degree of ataxia was noticeable. Sensation was normal. Knee-jerk and Achilles tendon reflex were also normal. The gait was uncertain, and with the eyes closed she tottered. There was a moderate goiter. In the left breast was a tumor the size of a pigeon's egg and painful on pressure. The patient also suffered from arteriosclerosis and bronchitis. There was enlargement of the glands in the inguinal region. She rapidly grew weaker, became demented, and developed incontinence. The speech was low and indistinct. Catarrhal pneumonia set in with a gradual rise in temperature, the patient succumbing on January 24th. The necropsy, performed by Dr. Störk upon the 25th of January, 1903, revealed the following: Scirrhus carcinoma of the left breast, chronic tuberculosis of the lungs and of the glands, diffuse colitis. Many segments of tapeworm. *A conglomerated tubercle the size of a hazelnut in the left thalamus opticus.* The histologic examination, made in Professor Obersteiner's Institute, showed that death was actually due to a tubercle which largely occupied the lateral portion of the optic thalamus, and compressed an area of 8 to 10 mm. in the middle of the peduncular portion of the internal capsule. The spinal cord was intact.

Some years ago I saw a similar case. It was published by Dr. Pineles in his article, "The Functions of the Cerebellum." It was the case of a man, aged 60, who suffered from headache and moderate vertigo: later his locomotion began to be uncertain; there were also peculiar involuntary movements of the left upper extremity. Examination

revealed slight nystagmus and mild disturbances of coördination in the left extremities, but otherwise the motility was intact. "When the patient was asked to extend the left arm or to perform any movement which necessitated the spreading of the hand, he made movements as if grasping at something, and the fingers were flexed and extended in a slow and quite rhythmical way. Occasionally there were movements in the elbow-joint as though the patient were reaching for some object. This slowly resulting spreading of the fingers closely resembled the play of the muscles in athetosis, and could only be controlled by a great effort on the part of the patient. Indications of this disturbance of movement were also evident in the left toes."

After a few weeks' illness the patient died of tuberculosis. The *autopsy* revealed *a tubercle the size of a walnut in the left cerebellar hemisphere.* On section it was evident that the neoplasm exclusively occupied the left half of the cerebellum and the middle of the quadrangular lobe.

While athetosis is relatively frequent in brain tumor, it is extremely rare in *abscess.* A. Berger, whose excellent compilation we shall frequently refer to, mentions only a single case of this kind. We note that the symptoms may appear in paralysis as well as in exceptional cases of compression of the brain.

In this discussion we have several times casually referred to *localization*; it is now time to consider this theoretically and practically. The number of published necropsies of athetosis and hemichorea is not very large, and among these are some which cannot be utilized on account of the multiplicity of the foci. In others again the autopsy finding is inconclusive.

As a clear illustration of a case which fulfilled all requirements we may consider the one histologically examined by Berger, and published in the *Jahrbuch für Psychiatrie,* Bd. XXIII, page 214. The patient was a man, aged 61, who was admitted to Nothnagel's Clinic for carcinoma of the stomach, and there succumbed. In his third year, apparently after a fright, he was stricken with right-sided paralysis; this soon improved, but peculiar permanent contractions persisted in the face and right arm, and became more marked with the lapse of time. As a matter of fact, in addition to a certain debility of the right hand typical athetotic movements appeared in the right half of the face as well. The *necropsy* disclosed a large carcinoma of the stomach; in the brain was a finding which I shall briefly sketch. A cavity in the posterior portion of the left nucleus lentiformis was almost filled with calcified masses about the size of a cherry. Upon *histologic examination* the internal capsule and the other parts of the pyramidal tract were seen to be perfectly normal. In reviewing the cases which maintain their position notwithstanding the sharpest criticism, we find exclusive foci in the thalamus opticus 11 times, also 3 cases (including my case mentioned above which has not been previously published) with foci in the thalamus and visible destruction of the internal capsule. Capsular foci were twice found, in 2 cases the lenticular nucleus was affected without implication of the internal capsule, in 3 cases the lenticular nucleus was the seat of the focus and the internal and external capsules were implicated, and a circumscribed focus in the cortex of the brain was only once found. Furthermore, we have just learned from Pineles's case that, under some circumstances, a lesion of the cerebellum may produce movements resembling those of athetosis, and this author collected several similar cases from literature. A case described by Halban and Infeld is very interesting: A girl, aged 15, from her first year had shown ophthalmoplegia upon the left side and right-sided hemiplegia

with spasm and hemichorea. The autopsy revealed a focus in the left tegmentum which had destroyed the greater portion of the red nucleus.

The *results of autopsy* still differ so greatly that it is evident the condition is far from uniform. These diverse results of necropsy have given rise to numerous and equally diverse *theories*, a few of which I shall briefly mention. A very peculiar view, hardly accepted in Germany, was promulgated by Charcot and his adherents. They believed that besides, and without doubt anterior to, the posterior nerve fibers in the corona radiata which convey sensory impressions, there is a layer of fiber bundles which possess peculiar motor properties, disease of which gives rise to hemichorea. Gowers regards disease of the optic thalamus as the cause. Anton believes that certain forms of chorea (not all) are to be referred to disease of the lenticular nucleus and corpus striatum, by the destruction of which inhibition disappears; some authors (Eulenburg) have suggested theories for its possible development from the cerebral cortex. Eisenlohr maintains, on the basis of an autopsy, that the spinal cord is responsible for choreic movements. In regard to the manner in which foci of the cerebellum produce athetosis, Pineles states the following: The cerebellum with its three important communication systems is to be included among the subcortical portions of the brain; therefore in the innervation of movements it gives off important constituents. Disturbances of motion occurring in man after disease of the cerebellum are also due to the fact that the power to regulate voluntary movements is lost; this, under normal circumstances, belongs to the cerebellum, and principally affects the hemisphere of the cerebrum upon the other side.

Later authors, such as Anton, Halban-Infeld, and Berger declare that all attempts to localize the phenomena in a circumscribed area are futile. The first to express this view were Kahler and Pick, according to whom these pathologic movements may be produced in any area as well as in the compact pyramidal bundle. v. Monakow believes that abnormal irritative processes on the part of the interbrain which are transmitted centripetally to motor centers of the cortex are necessary for the development of the condition.

Berger suggests a hypothesis by which these conditions may be made to conform to a uniform principle: If we investigate simple *hemiparesis*, we find that this may occur anywhere in the large motor tract from the cortex to the peripheral nerves, but it may also be produced by disease of the cerebellum. Is it not conceivable that, like paralysis, disturbances of movement may also be produced by the affection of several different cerebral regions? This appears to be favored by the fact that foci are found in almost all cerebral regions, in the cerebellum, and in the spinal cord. Berger does not state whether there is direct irritation of the pyramidal fibers or whether the irritation is transmitted by centripetally running fibers.

Besides the symptoms of athetosis in severe cerebral changes, those forms must be discussed in which either (*1*) *no anatomical lesion can be determined* or (*2*) in which affections (*a*) of the *spinal cord* or (*b*) of the *peripheral nerves* are the cause. The first group again leads us to the consideration of bilateral athetosis; this usually attacks all of the four extremities, it rarely spares the face, and it is usually indicated at birth; later onset is rare. The defective intelligence and the dragging, indistinct speech are usually conspicuous. As a rule, rigidity is very marked, paresis is slight. In rare cases

the musculature of the trunk and pharynx as well as respiration is implicated. Hypertrophy of the muscles is said sometimes to occur. The *results of autopsy* are still very scanty and inconclusive. Concerning the point which at present interests us it must be emphasized that anatomical findings have occasionally been absolutely negative; for instance, Déjérine and Sollier found nothing but an anomaly of the cerebral convolutions which Oppenheim also observed. In some cases of *unilateral* athetosis no lesions were found at the necropsy.

Under some circumstances movements resembling athetosis may be referred to *spinal diseases*; this is shown by a case of Eisenlohr's in which, however, the brain was not thoroughly examined. Years ago several authors, Rosenbach, Audrey and others, reported similar conditions in tabes. For years I have had under my care an official, aged 70, who, while a young man, contracted syphilis; in 1899 he was attacked by tearing pains and paresis of the lower extremities. While motion became normal after an energetic inunction treatment, the pains persisted; gradually moderate ataxia of the legs developed and the pupillary and patellar reflexes failed—conditions which did not improve under continuous mercurial treatment. In this man, almost from the onset of the affection, independent of the will, very slight movements were produced in the toes particularly upon the left side. Similar conditions are said sometimes to occur in *myelitis* and *poliomyelitis*. Several authors have described this symptom in *multiple neuritis*.

The **prognosis** is grave; recovery is not to be expected, and there is rarely improvement. Electricity, baths, massage and gymnastics are the curative agents, and these should be tried whenever possible.

TETANY OF ADULTS

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HISTORY AND CLASSIFICATION

The *chief symptom* of tetany is the appearance of bilateral, tonic, and intermittent *spasms* which are often accompanied by pain; they almost invariably occur during full consciousness, and are first visible in the hands, which show the peculiar position assumed by an obstetrician. They often affect the lower extremity, occasionally the muscles of the larynx, of the face, and of the jaws; more rarely the muscles of the chest, the abdomen, the neck, and the tongue. Now and then there are spasms of the diaphragm and of the muscles of the eye; spasms of the bladder and rectum, at least in adults, are almost unknown.

In the *sensory sphere* we seldom note any disturbance except the above mentioned pain; at most it is insignificant. Still more rare is any disorder of the organs of special sense. It is an interesting fact that in many cases pressure in the region of the brachial plexus between the paroxysms will evoke spasms (Trousseau's phenomenon). The *electric*, and especially the galvanic, contractility of certain motor nerves is invariably increased (phenomenon of Erb and Hoffmann), sometimes also of the sensory nerves. We often note an excessive, *mechanical* excitability of certain motor nerves (Chvostek's phenomenon), occasionally also of the sensory nerves (Hoffmann's phenomenon). The *mind* is usually unaffected. Mental disturbance is observed only in exceptional cases. Among the relatively rare phenomena, *trophic secretory* anomalies deserve mention: excessive perspiration, reddening of the skin, swelling of the joints, slight edema, loss of hair and of the nails, discoloration of the skin, the appearance of urticaria, herpes, polyuria, and glycosuria. The *pulse* is often accelerated. In some forms of spasm there is *dyspnea*; in others an occasional *rise of temperature* is noticed. The *tendon reflexes* may be increased; occasionally they are absent. *Epileptic attacks* not infrequently occur between the periods of contraction.

For the first clear description of the disease, as sketched above, we are indebted to Steinheim of Altona (1830) and to the French author, Dance. Soon after this the French (Constant, Murdoch, and others) made a study of tetany in children. Following this, Trousseau made a comprehensive research into tetany in adults. It was only in the early 70's that the German clinicians (for example, Kussmaul and Riegel) began to master the problem. Then came Erb's pioneer work on electrical contractility, and soon afterward other treatises by his pupils (Hoffmann, Fleiner, etc.). The works of Schultze should also here be mentioned. In 1878 Chvostek of Vienna de-

scribed the phenomenon which bears his name. From that time on Viennese physicians closely studied this remarkable affection. In Billroth's Clinic, Nathan Weiss made the memorable discovery of tetania strumipriva, a discovery which gave a renewed impetus to the far reaching experiments on the thyreoid gland. This was followed by my detailed report of the most important phenomena, the appearance of psychoses in tetany and the epidemico-endemic foundation of the disease. Prior to my investigations, N. Weiss and v. Jaksch had made a notable report on tetany in working men; subsequently Chvostek, Jr., and H. Schlesinger investigated its symptomatology. After the treatises of Abercrombie and Babinski, a voluminous literature on tetany in children appeared. I refer to the researches of Ganghofner, Escherich, Hochsinger, Loos, Kassowitz, Mann, and others. At present we shall limit our discussion to the main theme—tetany in adults. According to its occurrence we classify it as follows:

(1) *Tetany in healthy young working men.* This is most apt to appear at certain seasons, and in certain cities (Vienna and Heidelberg); it chiefly attacks young men, and preponderates in certain trades (cobblers and tailors).

(2) *Tetany in gastrointestinal affections.*

(3) *Tetany in acute infectious diseases* (enteric fever, influenza, angina tonsillaris, etc.).

(4) The rare cases of tetany caused by *poisoning* from certain substances (chloroform, morphin, ergotin, and lead).

(5) Tetany of *the puerperium* (during pregnancy, labor, and lactation).

(6) Tetany following loss or absence of the *thyreoid gland.*

(7) Tetany *in connection with other nervous diseases* (Graves' disease, tumor, syringomyelia, etc.).

Group 1 contains the most common cases of tetany in working men. Even the early French authors had noticed the prevalence at certain times of tetany in children. Subsequently, like observations were made concerning adults (Delpech, Verdier, Aran, and Rabaud). The observations of Weiss and v. Jaksch gave us the first intimation that this applied also to the working men of Vienna. The first fairly comprehensive statistics were compiled by me from the admission cards of the General Hospital of Vienna, and are herewith presented. The 368 cases admitted were distributed throughout the year as follows:

Month.	No. of Cases.	Month.	No. of Cases.
January	45	July	5
February	56	August	5
March	87	September	4
April	75	October	5
May	33	November	12
June	19	December	22

It is evident, and is noted every year, that there are few cases in July, August, September, and October. In November there are slightly more. The number of cases steadily increases throughout December, January, and February until the acme is reached in March and April; it distinctly declines in May, and more markedly in June. In other countries where tetany of working men prevails similar observations have been made. My study of

the admissions to the General Hospital also revealed the peculiar fact that certain years bring many cases, others but a few.

Another table gives a compilation of the occupations. Among 399 men there were 174 cobblers, 95 tailors, 26 carpenters, 20 locksmiths, and 19 lathe-workers; of the remaining 65, 1 to 5 were found in each of various other occupations. It is interesting to compare these figures with the percentage of workmen in the total population of Vienna; for instance, in 1894 the cobblers formed 1.3 per cent. of the whole population, and furnished 43.6 per cent of the cases of tetany in the Hospital; therefore the cases of tetany were out of proportion to the percentage of the total population, being 41 times as numerous. A similar ratio was observed among the tailors. In comparison with this it is noteworthy how very rarely builders were affected by the disease; my statistics show only two masons; there was but one factory hand. These figures will at once strike us as insignificant on comparing them with the enormous number of persons engaged in such occupations in a metropolis like Vienna. It must also be remarked that tetany is not observed among the higher classes of Vienna.

The persons attacked by the disease are usually only those of a certain age. My statistics show that 83 per cent. of the cases were between 16 and 25, and only about one-eighth of the number had advanced beyond the 25th year.

We must bear in mind an important fact which has already been mentioned, that the malady is by no means equally distributed throughout the world; in the cities in which it was formerly rare it is now common, and in those in which it once prevailed it is now almost extinct. I have for some years devoted considerable attention to this peculiar distribution of the affection, both by personal observation when I traveled as well as by questioning many foreign colleagues who came to Vienna.

I have scarcely ever had a case from the country, notwithstanding the fact that I have a large number of hospital patients from lower Austria and other provinces, and observers in the principal cities of the various provinces have reported but little concerning tetany among the laboring classes. To my argument the objection has sometimes been raised that tetany is not reported outside of Vienna because unrecognized, hence the opinion prevails that the disease is rare elsewhere—an objection which is shown to be without foundation since a number of neurologists have accepted medical chairs in cities of the provinces after having had for years the opportunity of studying tetany in Vienna, and have thus been enabled to recognize it when observed in the cities to which they removed. Outside of Vienna cases have been reported by relatively few physicians—by v. Jaksch in Prague, by Kraus, by v. Wagner and Anton in Graz, and Mayer in Innsbruck; Schultze, who observed many cases in Heidelberg, never saw the disease in Bonn. The rarity of the affection in many other German Clinics was also demonstrated; for instance, in Berlin, Hanover, Leipsic, and Kiel. The same exemption from the disease is noted in France, Italy, Scandinavia, America, and Japan. While such cases are rarely observed in Russia, yet actual but mild epidemics have been twice reported (Minor, Wermel); Vaughen described a limited epidemic among Italian laborers in New York.

In Paris tetany of workmen is a great rarity. In 1891 Charcot told me he

well remembered seeing a few cases in his youth, but that after about 1860 he observed none. Is it not remarkable that the Viennese physicians should have been so backward in taking up the study of this disease? Skoda knew the affection well, one of his clinical lectures on the subject having been preserved. If the cases had at that time been as frequent as they now are, the fact would certainly have been commented upon by so prominent a clinician as he. I have made many inquiries among Viennese physicians who were attached to hospitals during the years between 1860 and 1880: they all assured me that at the time mentioned such diseases were exceedingly uncommon.

We therefore consider that not only the epidemic but also the endemic nature of the affection is fully proven, and we will now demonstrate similar conditions in the other forms of tetany. A few instances are recorded of severe cases of the disease being observed in one and the same family.

Group 2 includes those cases in which the symptom-complex is noted in the course of severe *gastrointestinal affections.* Most interesting are those greatly discussed cases which are combined with gastrectasis—a combination which Kussmaul described many years ago. The progress of these cases is usually very rapid, and they are often fatal. As a rule the underlying affection is severe and the dilatation marked, the cause often being pyloric stenosis, occasionally stenosis of the small intestine. From a theoretical standpoint it may be confidently asserted that my statement, made in 1890, that such cases are most likely to occur during the tetany months has been confirmed. Tetany is not invariably produced by severe gastrointestinal affections, as has been stated, for a few cases of moderate severity have been reported in perityphlitis, in helminthiasis, and in acute gastritis.

Group 3 includes the cases which occur in the course of *infectious diseases.* Aran described tetany which appeared in enteric fever, and similar cases have been frequently reported. Now and then it has been noted in other infectious diseases, as in measles, influenza, tonsillitis, croupous pneumonia, and acute articular rheumatism.

Group 4 embraces those cases in which the spasms are produced by a *nonorganic* poison introduced into the system from without. The earliest cases belonging to this category were probably caused by ergotism, and perhaps, if individual observations are reliable, also by alcohol, chloroform, morphin, lead, phosphorus, and carbonic oxid.

Group 5 is more readily understood: it is *the tetany of maternity.* Since the time of Steinheim and Dance cases of this kind have been known to appear after childbirth, and Trousseau has described an actual epidemic in nursing women.

Among 61 cases (49 from literature and 12 of my own) 23 occurred in pregnant women, and the majority of these in women who were in good health. A few were suffering from maladies not connected with the puerperal state; in others intercurrent affections existed—for example, goiter, enteric fever, ergotism, and variola—the relations of which to tetany were not without interest. Tetany usually appeared between the sixth and eighth months, occasionally between the fourth and fifth. Relapses in subsequent pregnancies are not rare. In 10 cases the disease appeared after parturition; generally, however, during lactation, sometimes in the early, sometimes during the later, course.

In 1846 Delpech reported two mild epidemics, a fact which is the more remarkable because subsequently tetania lactantium absolutely disappeared in Paris. In 1856 Verdier expressed his surprise that so many cases occurred in winter. Of 52 cases in which I ascertained the date of onset, 39 occurred between January and April, and only 13 in the remaining eight months. These conditions, therefore, remind us of workman's tetany. Most interesting of all is the circumstance that, in the pregnant, even partial strumectomy may produce tetany, whereas under other circumstances the operation is rarely followed by this disease.

We now come to the much discussed **Group 6**—*tetany with thyreoid insufficiency.* The fact, of scientific value, that acute and fatal tetany often follows total extirpation of the thyreoid gland was made known in 1880 by Nathan Weiss, of Billroth's Clinic in Vienna. Many surgeons have reported similar conclusions; it is true the frequency of these occurrences rapidly decreased, for surgeons learned to appreciate the danger of such an operation. Nevertheless isolated cases were still reported, either because total extirpation was unavoidable, or because even partial strumectomy was not always devoid of consequences. Pineles reported 16 cases of this kind. The fact was noted that Vienna furnished many more cases than other cities; the season did not appear to exert any special influence. Some cases make it seem likely that tetany may also occur in *congenital deficiency* of the thyreoid gland, but this is not yet quite certain.

To complement the forms of the disease **Group 7** may be mentioned; here I include cases the nature of which is still very doubtful, and in which the typical attacks of tetany appear in the course of other diseases of the nervous system. In a few instances this form of tetany has been very clearly described as combined with Graves' disease; and here a thyreogenous development comes into question with both affections. The appearance of tetany in spinal disease (syringomyelia) as well as in brain tumor is very obscure.

ETIOLOGY

In the presentation of the *etiology* of this disease we must admit that our knowledge is very limited. The only form in which we are certain of the cause is tetania strumipriva. That the extirpation of goiter is not rarely followed by fatal tetany has already been stated. When this fact became known years ago, doubts were expressed whether the loss of the thyreoid gland could actually cause the disease. Suppuration of the wound, injury to the recurrent laryngeal nerve, and similar conditions were thought of, and doubts were removed only by resorting to animal experiments. Schiff's researches which had remained unnoticed were now taken up; he had demonstrated that strumectomy would kill a dog, but that death could be averted if a thyreoid gland were grafted into the abdomen. Eiselsberg removed the thyreoid of a cat, and sewed it into the abdominal cavity; the animal continued to live, and only succumbed when the ingrafted organ was subsequently extirpated; v. Wagner-Jauregg pointed out the analogy between animal and human tetany, and a number of investigators confirmed the foregoing observations.

Further discussion of these experimental findings is probably to-day un-

necessary. By untiring labor, and upon the basis of these studies, as well as by researches in myxedema, a number of prominent investigators have proven that the thyreoid gland furnishes a secretion which is formed from the blood, and in the blood it has an antitoxic effect upon the toxic products of metabolism (Ewald).

The view just described has recently been considerably modified; it is not the absence of the thyreoid gland which produces tetany, but the loss of its epithelial bodies. The chief exponent of this theory is F. Pineles, whose reasoning we shall now follow: The auxiliary organs of the thyreoid gland—the so-called glandulæ parathyreoideæ—were first described by the anatomist Sandström, and Gley and Moussu first studied their functions. But only when the internal glandulæ thyreoideæ contained within the thyreoid gland were discovered in 1895 by A. Kohn of Prague was the condition actively discussed. Upon the basis of this study Vassale and Generali on the one hand, and Pineles on the other, came to the conclusion above expressed. Besides the results of the animal experiment, Pineles pointed out that in thyreoplasia or congenital myxedema, which forms a definite pathological picture (extreme dwarfism, idiocy, and myxedema), the epithelial bodies are always intact; tetany has never been observed in such individuals. (See volume "Diseases of Metabolism and of the Blood," article "Myxedema," p. 179.)

Furthermore, Pineles attempted to prove by the study of thyreoid gland operations in adults that tetany shows itself only when the epithelial bodies are destroyed. It is certain that the ominous symptom-complex appears when, in partial extirpation, the isthmus or the upper portion of the lateral lobes has been allowed to remain. If we bear in mind the fact that in man the epithelial bodies are located in the lateral lobes of the thyreoid gland; that in strumous degeneration of the thyreoid gland the relation of the epithelial bodies to the thyreoid is altered; that in the removal of both lateral lobes we must weigh the probable effect of extirpation or lesion of the epithelial bodies; and that the epithelial bodies are usually located in the lower half of the lateral lobes near the point of entrance of the inferior thyreoid artery (even considering the change of position in goiter), the view seems very reasonable that the upper parts of the lateral lobes which adjoin the point of entrance of the superior thyreoid artery contain no epithelial bodies. The greatest danger from removal or injury of the epithelial bodies therefore exists in those cases of partial strumectomy in which the lower parts of the lateral lobes have been extirpated, and the isthmus or upper portion of the lateral lobes has been allowed to remain. In fact a review of the cases indicates that those patients are most susceptible to tetany in whom the lesions of the epithelial bodies are most extensive, or in whom these bodies have been extirpated. Pineles attaches value to the fact that when extirpation affects the lingual portions tetany does not result. These patients probably have no cervical thyreoid; nevertheless they have epithelial bodies.

I mention these recent views on account of their intimate relation to the subject under discussion; but further research will be necessary in order to decide this question. We notice how many explanations are still offered in the discussion of this disease, and in the "other groups" of tetany there is probably still less of accurate knowledge. Let us first consider the most

common form, the *epidemico-endemic form of occupation tetany.* So many remarkable facts have been compiled regarding this variety of the disease that it seems as though at any moment the enigma of its nature might be solved. Researches have been carried on for years, yet neither other observers nor I have advanced beyond the theory which I developed in 1890. At that time, in quoting a suggestion of v. Jaksch's, I proposed the hypothesis that this form of the affection must be produced by some factor which appears only at certain seasons and in certain localities. In corroboration of this view I urged the following considerations: 1. Its epidemico-endemic appearance which has been fully described above; 2. The not infrequent onset with fever; the course, occasionally with high, occasionally with subnormal, temperature; 3. The intercurrent *acute psychoses,* which were first described by me, and were afterward repeatedly reported; 4. The occurrence of several cases in one family, which has been a few times observed; 5. The statistical proof that in some years there are many, in other years but few, cases; 6. The sudden appearance of groups of these cases in places where formerly (perhaps also subsequently) the disease was unobserved. Here the decline of the affection in Paris is noteworthy, as well as its rarity in Vienna until about 1870. The idea has been abandoned that the form of manual labor is of consequence, for why should the cobblers of Vienna only be attacked, and not those of Paris? Why do we see so few seamstresses with these spasms, and so many tailors? Nor can exposure to cold be considered the cause, when in much colder countries—for example, Scandinavia and England—occupation tetany is rarely seen.

Valuable reports of thyreoid tetany (Schultze, Hoffmann, and Pineles) have led to the question whether the previously mentioned organs (the epithelial bodies) here play a rôle. In favor of this idea it was stated that goiter is not rare in Vienna; in Steiermark and the Tyrol it is much more common, yet in these places tetany is particularly rare. In the tetany of maternity a powerful influence has been ascribed to the thyreoid gland, and the increase of goiter observed in pregnancy—even its appearance during this state—has been urged to support the view. Here we must call to mind the remarkable demonstration of Lange that pregnant cats require more thyreoid gland substance to keep them healthy than other cats; if more than four-fifths of the thyreoid gland is extirpated they are seized with tetany, which improves under the administration of iodothyrin. I must also refer to the fact that if partial strumectomy is performed in women during pregnancy many of them develop tetany. Although we have attempted to ascribe to the thyreoid gland an influence in the tetany of maternity, this organ cannot be the actual causative factor. It must be reiterated that only in certain regions, and even here only at certain seasons, do these cases arise, and epidemics have repeatedly appeared and disappeared suddenly. Occasionally we note that a combination of maternity and acute infectious disease precedes the spasms of tetany. I believe that maternity is merely a predisposing cause. It often forms a basis for the same diseases of the nervous system as do the infectious diseases: for chorea, polyneuritis, acute transitory mania, and optic neuritis.

Only in this way can I regard maternity as predisposing to tetany, only this rôle can I ascribe to the acute infectious diseases. There must be an auxiliary agent; this alone would explain why this combination should be

noted only in definite regions and at certain periods. We do not know why tetany appears after the introduction of poisons (morphin, chloroform, etc.); cases are so rare that no explanation has yet been offered. On the contrary, gastrointestinal tetany (which is not so rare) furnishes a fruitful field for hypothesis. The best known are those of Fleiner and Albu. Fleiner has lately brought forth Kussmaul's theory, in which it is assumed that the predisposition to spasm noted in severe gastric affections is caused not only by autointoxication and hyponutrition, but chiefly by a great loss of fluid which the weakened and parched organism sustains for a short time prior to the appearance of the spasmodic attack in consequence of processes generated in the stomach. The fluid lost to the organism collects in the stomach, where, combined with the gastric contents already present in a state of fermentation, and the development of gas, it causes an enormous distention of the diseased organ. This is distressing but endurable, and in itself does not produce spasms, probably because it occurs gradually; if, however, it is suddenly relieved by severe vomiting or the introduction of the stomach-tube, the over-distended gastric wall contracts violently and immediately causes spasms. Fleiner, therefore, ascribes to these irritations (vomiting and the introduction of the stomach-tube) merely a final and causative effect, but does not seem to regard them as the actual primary cause (as authors have previously done).

Another hypothesis originated with Gerhardt, and was concurred in by Albu and many others. It is based on the idea of an autointoxication of the organism from stagnant, decomposing, and fermenting masses in the dilated stomach.

But we should remember that those who have advanced these theories have proposed only hypotheses; none has furnished final proofs. This has been unsatisfactory, and the latest authors upon this subject (Rudinger and Jonas) have returned to the view which I advanced in 1890, when, in my first monograph, I called attention to the fact that the majority of cases of tetany occur in the typical "tetany months." It is true I could then compile but few cases from literature; but with the appearance of additional reports, and the increasing prominence of this factor, the more likely does it appear that the changes in the organism due to severe gastric affections form only a predisposing basis for infection from without.

SYMPTOMS

Having sufficiently discussed the occurrence and etiology of tetany, we shall now study the symptoms. Most prominent among these are the following: (1) **Tonic, intermittent spasms of the transverse striped muscles**; the upper extremity is first affected, the fingers being contracted, and reminding us of the form which the hand of the obstetrician assumes for introduction into the vagina; the hand often appears as though actuated through the ulnar nerve; now and then other positions are observed. Sometimes there is flexion at the wrist-joint, more rarely at the elbow-joint; the shoulder-joint is often adducted. In many cases the lower extremity is first attacked, there is plantar flexion of the joints of the feet and toes, and occasionally extension at the knee. In rare cases there are intercurrent contractures of the muscles of the face and those of mastication. The muscles of the chest, of the back, and of

the abdomen, are now and then implicated, occasionally those of the larynx (this is different in children), the diaphragm, the pharynx, and the eyes; not infrequently there are spasms of the tongue, particularly on yawning. In the adult the transverse striped muscles of the bladder and rectum do not appear to be involved. Tonic rigidity is usually symmetrical—rarely unilateral. The attack may last some hours, or, in rare cases, days. As a rule, the causative factor is unknown, but psychical stimulation, rheumatic influences, or bodily fatigue, can sometimes be demonstrated. In many cases there is true intention spasm—a fact to which Schultze first called attention: intended movements are increased to tonic spasm. Slight tremor and moderate motor weakness are often observed in the extremities of these patients as auxiliary phenomena.

(2) **Trousseau's Phenomenon.**—Trousseau discovered a method by which, even in the period free from attack, we may produce the spasm of tetany, or, if already present, may increase it. According to him, we may accomplish this by exerting pressure in the affected members, either upon the principal nerve trunks or the vessels, in such a way as to arrest the arterial or venous circulation.

Trousseau's discovery was soon everywhere confirmed. The test is more frequently successful in the upper than in the lower extremity; the phenomenon is seldom seen except in tetany, but it can be demonstrated in two-thirds of the cases of this disease. For decades the actual cause was much discussed. While some authors attributed it to compression of the artery and consequent anemia, others maintained that the spasm was the result of irritation of the nerve from pressure. Years ago I settled this dispute by a simple animal experiment: In dogs attacked by tetany after extirpation of the thyreoid gland I not infrequently noted Trousseau's sign as well as extensor spasms and electric hyperirritability, for pressure in the region of the crural artery during the attacks free from spasm will evoke extensor spasms. When I laid bare the crural artery and the crural nerve, no matter how strong the compression of the vessel, I was unable to elicit spasm, but the slightest touch upon the nerve at once produced it. These experiments, which were recently repeated by Kashida in Tokio, show most distinctly that the phenomenon is the result of nerve irritation. This is also favored by the observation that unilateral compression will often produce bilateral spasm, and that contractions are sometimes caused by pressure at other points in which there is no large vessel (for example, upon the ankles); thermal irritations may produce spasm (Kashida).

To produce this phenomenon the upper arm should be spanned with the hand, and the four fingers should be pressed with considerable force into the region of the plexus. Sometimes the spasm appears immediately, at other times after one to three minutes; instead of compression, we may constrict the arm with a bandage or a piece of rubber tubing.

In addition, there are cases of "latent tetany" in which, besides Trousseau's phenomenon, we note electric and mechanical hyperirritability without spasm.

(3) **Electric Hyperirritability** (*Erb's phenomenon*; *Hoffmann's phenomenon*) of the motor nerves, to which Benedikt and Kussmaul had previously called attention, was first accurately studied by Erb. The first galvanometric

measurement was made by Nathan Weiss. In 1887 I published the first thorough study of the condition of the muscles, at that time little understood. Simultaneously with these appeared Hoffmann's reports concerning the motor and sensory nerves.

Since then it has been frequently remarked that no other disease so invariably runs its course with marked hyperirritability; the nerves of the upper extremity, especially the ulnar nerves, are the ones which reveal the well known phenomenon. It is chiefly *galvanic* irritability which causes these conspicuous changes. Cathodal closure contraction is often produced by the feeblest currents, 0.05–0.1 milliampère; and cathodal closure rapidly induces tetany. The anodal contractions are soon evoked. While, in the norm, we can never produce anodal closure or anodal opening tetany, no matter what the strength of the current employed, in tetany it can be caused even by relatively feeble currents; occasionally we observe a cathodal opening contraction which never occurs in the norm—and, in rare cases, even cathodal opening tetany. The constancy of this increased irritability to the galvanic current is equalled by its inconstancy to the faradic—a peculiarity to which I called attention in 1887, and which has since been frequently confirmed. In many cases we observe the phenomenon studied by Chvostek, Jr., and described by Hoffmann: on galvanic irritation of the sensory and motor nerves, even with very feeble currents, the patient has distinct sensations which are often radiating; i. e., they produce paresthesia, which follows the anatomically familiar distribution. Both Chvostek, Jr., and I reported increased irritability of the auditory nerve.

(4) **Mechanical Hyperirritability** (*Chvostek's phenomenon, Hoffmann's phenomenon*).—Chvostek, Sr., first pointed out that, in tetany, lightning-like contractions can be produced in the face by tapping over the course of the facial nerve; sometimes they can be evoked in other motor nerves, but not with the same constancy. This sign furnishes a most significant guide. In typical cases contraction is caused by a slight tap in front of the ear, and occasionally by mild stroking of the cheek (Schultze). In less marked cases, contraction of the angle of the mouth and the ala nasi on the same side can usually be produced only by a percussion blow below the pons zygomaticus; occasionally it will appear only in the angle of the mouth. But it must be emphasized that Chvostek's symptom may be absent, particularly in chronic tetany; inversely, as I showed in 1886, and as Schlesinger, Schultze and others agree, it is by no means pathognomonic; it may sometimes be demonstrated for years in normal persons, and more frequently in those who are neurasthenic, hysterical, or epileptic; nevertheless, in those not suffering from tetany it never assumes the marked degree which is common in so many—although not in all—of those afflicted with this disease.

Hoffmann pointed out a similar condition in the sensory nerves: tapping upon those areas which in neuralgia often appear as pressure points will not infrequently produce a powerful sensation which may radiate along the course of the nerves. I have often evoked this sign in patients with tetany—occasionally also in those who did not suffer from it.

(5) **The Sensorium.**—In the malady under consideration consciousness is usually retained unless there is a severe, underlying affection; for example, severe gastrointestinal disease.

In 1889, when connected with Meynert's Clinic, I published for the first time three cases of acute psychosis in tetany—a condition which is theoretically interesting; similar ones were reported by Kraepelin, Hochhaus, Westphal, and Hirschel. Besides these severe psychoses we now and then observe a transitory state of excitement.

(6) **The organs of special sense** are but little affected; spasms in the muscles of the *eye* are occasionally observed, and were closely studied by Kunn; sometimes there is cataract formation. Now and then narrowed pupil and dilated pupil have been emphasized. Some cases of pupillary rigidity have been reported. Inflammation of the optic nerve has been described as an exceedingly rare occurrence.

Pain is the most prominent *sensory disturbance.* Although some cases run their course without this symptom, nevertheless it frequently accompanies the spasm, and often persists longer. Sometimes the pain is of great intensity, sometimes it is slight—often there is only a more or less unpleasant paresthesia. This symptom is by no means rare, and in time we learn to recognize a *forme fruste* of this affection, the only subjective symptom being formication. In contrast to the common appearance of the symptoms mentioned is the rarity of objective sensory disturbances; most cases run their course without these. If present, there is, as a rule, only slight hypesthesia of the finger-tips or the fingers, perhaps of the hand, rarely of the entire upper extremity. The large nerve trunks are occasionally sensitive to pressure.

(7) As already stated the **temperature** is sometimes increased, even during the attacks which are not due to an underlying and acute febrile disease; subnormal temperatures also are now and then observed.

(8) Occasionally there are disturbances of the **circulatory and respiratory organs** which are attributable to muscle spasms. In the forms running an afebrile course tachycardia is sometimes noted.

(9) In adults I have never observed **disorders on the part of the urinary organs,** but a few cases in children have been described. *Polyuria* and *polydipsia, albuminuria* and transitory *glycosuria* are rare occurrences.

(10) **Trophosecretory anomalies** are more common. In some cases there are outbreaks of sweat and reddening of the skin, as well as slight edema. In occupation tetany the patient's face is often slightly swollen. Herpes and urticaria have been observed. Alopecia and changes in the nails are more interesting, since they reveal a transition into myxedema. Change in the nails is a conspicuous symptom, which, however, is seen only in severe and chronic cases. Cataract of the lens has been reported by Meinert, Peters, Wettendorfer, and others. Upon prolonged observation a few cases show atrophy of individual muscle groups (Hoffmann and Max Weiss).

(11) In many cases the **tendon reflexes** are normal; sometimes they are increased, but the absence of the patella tendon reflex during the paroxysm has also been observed. Absence of the reflexes may continue after the cessation of the paroxysm.

(12) We not rarely meet tetany patients who suffer from **typical epileptic spasms**; in some cases this is certainly a mere coincidence, but it occurs so frequently that we are forced to conclude that it is not always accidental. An internal connection is strongly favored by the fact that tetanic and epileptic spasms have been observed after strumectomy in persons who were

previously free from nervous disease, and that animal experiment occasionally reveals similar signs. Here it may be emphasized that some authors include among the symptoms of tetany the hysterical attacks now and then observed.

DIAGNOSIS

The diagnosis of well developed tetany in the adult is extremely easy; the characteristic spasms with a free sensorium, the demonstration of Trousseau's sign, and the electric and mechanical hyperirritability remove all doubt. As an aid to the diagnosis, we note at certain times and in definite localities that special underlying conditions (severe gastrointestinal affections, strumectomy, maternity) and the peculiarity of the occupation (of cobblers and tailors, most of whom are still young) are factors. We can, therefore, be brief in discussing the *differential diagnosis*.

Some decades ago much was written of the differentiation from *tetanus*, but nowadays, when both affections are so thoroughly understood, it is needless to consider this at length: the etiology of tetanus is trauma, a factor which plays no rôle in tetany. In tetanus the muscles of mastication and of the nape of the neck are chiefly affected while the muscles of the hand remain intact; reflex irritability is greatly increased—a peculiarity not noted in tetany—while in the former affection we do not find Trousseau's phenomenon nor electric and mechanical hyperirritability. The differential diagnosis from epilepsy will rarely cause perplexity because in this disease symptoms appear which are foreign to tetany: the characteristic aura, loss of consciousness, clonic contractions, incontinence, post-paroxysmal confusion, etc. But it must be borne in mind that there are mixed forms of epilepsy and tetany.

There is actually but one disease—*hysteria*—which causes difficulty in diagnosis. Recent studies have shown that the previously mentioned neurosis, in which everything may be simulated, may produce a picture closely resembling tetany; here I refer to the reports of Blazizek, Schlesinger, Krafft-Ebing, Nikolajevic, and Curschmann. The last author has recently devoted two very interesting articles to this question. If patients with this affection (and they are almost invariably females) suffer from tonic tetanic spasms, and if the accompanying triad of phenomena is absent, the differential diagnosis will occasion no perplexity. But in some cases investigators have also observed mechanical hyperirritability and Trousseau's phenomenon; the former symptom is of limited importance, as it is not rare in other affections, and is quite common in hysteria.

The diagnosis is much more obscure when symptoms resembling Trousseau's sign are present, but these symptoms differ so widely from the genuine sign that Krafft-Ebing has referred to them as the "pseudo-Trousseau sign." H. Curschmann gives the following points of differentiation: in pseudotetany this phenomenon does not appear slowly and gradually, but immediately at the moment of compression. The nature of the spasm is peculiar; for example, there may be a paradoxical and spasmodic position of the fist, with tonic contraction of a few of the muscles supplied by the median nerve. In most cases suggestive measures (for example, pressure on the ovary) may produce the same effect as the above mentioned methods; but in genuine cases

we never see electric hyperirritability—a fact which is calculated at once to decide any doubtful case.

In conclusion, a word in regard to the differentiation from myotonia congenita (Thomsen's disease), which must be considered because, as already stated, intention spasms now and then occur in tetany, and because, as Schultze has recently shown, electric and mechanical reactions of the nerves and muscles may occasionally be produced which resemble Thomsen's disease. But the facts that in tetany muscular hypertrophy is absent, and that the symptoms which remind us of myotonia are very rare, while in myotonia spontaneous spasms, Trousseau's phenomenon, and marked electric hyperirritability of the nerves are wanting, will render the diagnosis easy. Thomsen's disease is congenital, and usually progressive. Tetany is acquired late and is often curable, and those symptoms which remind us of Thomsen's disease often disappear simultaneously with the spasms.

This discussion of the differential diagnosis is based upon the supposition that we are considering a *well-developed* form of the affection. There are, however, *formes frustes*. One of these is marked by the appearance of paresthesia, of Trousseau's phenomenon, and of electric and mechanical hyperirritability.

The conditions are less clear in an affection which I described in the year 1887 as "*tetanoidie.*" In the spring of this year, in which an uncommonly violent epidemic prevailed, I saw four cases—mostly in young workmen—with paresthesia of the fingers, and occasionally with severe facial nerve phenomena, in none of which was there spontaneous spasm, nor could this be produced by pressure, and only the appearance of extreme galvanic hyperirritability distinctly revealed the connection; similar cases were subsequently reported by many authors. That such cases may not rarely be confounded with other conditions—for example, with neurasthenia, hysteria, acroparesthesia, occupation neuroses, etc.—is quite conceivable. If an epidemic prevail in a city in which there is usually more or less tetany, and a patient come to us with the foregoing subjective symptoms, the occupation (that of cobbler or tailor) or an underlying condition (maternity, strumectomy, or severe gastrointestinal affection) may alone lead us to suspect tetany. When this suspicion is once awakened, the demonstration of Chvostek's sign and Erb's symptom will soon clear the situation.

PROGNOSIS

In turning to the *prognosis* we must first state what we know of the duration of tetany. There are cases in which the spasms occur but once, and last for only a few hours; usually they appear acutely and have a duration of weeks, during which time the attacks do not always occur daily, but are noted at intervals. In the stage of onset rigidity lasts merely a few hours, subsequently for many hours; in the intermissions the person feels quite well or complains merely of lassitude or paresthesia. Although months may pass in which the health is perfect, yet relapses occur, frequently during the months of February and March; these cases have been grouped by v. Jaksch under the name of "*acute relapses.*" In examining such a patient whom I had asked to call upon me after some years, I was struck with the fact that very few

of them entirely recover. The majority, even between the paroxysms, are not perfectly well. They suffer from paresthesia and a feeling of constriction, and not rarely present Chvostek's, Trousseau's, or Erb's symptom; sometimes there is cataract, alopecia, or shedding of the nails, as well as general nervousness. Some show the picture of chronic tetany, which has already been described.

The mildest cases are those which follow acute infectious diseases and intoxications. In the tetany of workmen very mild attacks are common, but a large contingent of these patients not infrequently have relapses into chronic forms. In the tetany of maternity we often observe a fulminant onset, sometimes the attacks are brief, at other times more prolonged, and parturition often terminates the affection. Relapses are not unusual, especially if the woman again becomes pregnant, also after labor, or while nursing her child. A chronic condition in this form is not rare. Two fatal cases have been reported.

The tetany of gastrectasis occupies a unique position: here the attacks are usually very severe. As a rule, death occurs after a few days, more rarely in the course of weeks—perhaps not on account of the tetany, but because of the underlying grave condition. Occasionally such patients recover. It has already been mentioned that tetany following total strumectomy is often fatal. In other cases the attacks are merely transitory, and there is complete restoration to health. In a third group tetany occurs with myxedematous symptoms, or myxedema follows a transitory tetany, and in these cases spasms appear only rarely or not at all.

PATHOLOGY

Pathologico-anatomically the findings in the adult are not convincing. Ignoring the earlier conclusions which were only macroscopically arrived at, we must state comprehensively that the few positive findings implicate merely the *spinal cord.* For instance, N. Weiss found changes in the ganglion cells of the anterior horn, Langerhans described vascular changes; these semi-positive findings are offset by the negative ones of prominent authors. It may be mentioned parenthetically that recent investigators have also postulated spinal cord changes in the tetany of children. The origin of this affection in the spinal cord is favored by the symmetrical bilateral appearance of tonic spasms and the increase of the reflexes which is occasionally demonstrated. Implication of the brain is denoted by the spasm of certain cranial nerves (the facial, the motor trigeminal, and those supplying the ocular muscles), intercurrent psychosis, and epilepsy.

TREATMENT

The *prophylaxis* of tetany is self-evident. True prophylaxis can come into question only in tetania strumipriva; total extirpation should be avoided, and, in accordance with recent advice, the epithelial bodies should not be excised. In women who suffer from tetany during pregnancy, conception should be avoided; should they again become pregnant, we should advise them to remove from the city in which tetany prevails. This advice should also be given to young workmen who suffer from the acute relapsing form.

Those predisposed to tetany should give careful attention to the digestive tract—a rule which also applies to those in whom the affection has already appeared. In gastric tetany, internal *treatment* is the most satisfactory. Lavage may be cautiously employed, but Fleiner particularly warns us against considering any operation. Nursing women should wean the child, and whenever tetany threatens during pregnancy the latter, under all circumstances, must be interrupted. For obvious reasons much was expected from thyreoidin; although the results have so far not been satisfactory, yet in doubtful cases I should at once make a trial of this form of medication. Our treatment, therefore, is still symptomatic. In desperate cases morphin cannot be avoided; chloral hydrate and the bromids also come into question. The salicylates, antipyrin, pyramidon, and similar remedies are useful to relieve the pain. Undoubtedly a symptomatic value attaches to electrotherapy: galvanization of the medulla spinalis by means of medium currents (4 to 5 milliampères) and labile galvanization of the extremities sometimes appear to be beneficial. The pain and paresthesia are often mitigated by the faradic brush. I have seen relief from protracted warm baths, and now and then from packs. Lukewarm or cold douches, bandaging the extremities at night, and the application of ice bags to the back, may also be tried. Abandoning the occupation sometimes produces a remarkable and very rapid improvement; absolute rest in bed is necessary in the serious cases.

THOMSEN'S DISEASE (MYOTONIA CONGENITA)

By L. v. FRANKL–HOCHWART, Vienna

L. K., a servant, aged 28, is the patient whose history will now be detailed. Upon the 10th of May, 1904, she was admitted to Nothnagel's Clinic, and reported that her mother had died in the preceding February from inflammation of the lungs. The mother was said to have suffered during childhood from the same disease which this patient presented, but her symptoms were much less marked. At fifteen the mother was attacked with articular rheumatism, also with a disease of the bones which produced curvature of the legs. At thirty she suffered from an attack of apoplexy with right-sided paralysis of the extremities, and shortly before her death had a second stroke of paralysis affecting the left side. The father of the patient was an alcoholic, and was said to suffer from hematemesis. The patient's aunt had epilepsy; the maternal grandfather was neurotic, but the patient knew nothing definite as to the nature of his disease.

In childhood the patient herself had diphtheria, scarlatina, and a pulmonary inflammation; she did not begin to walk until 4 years of age, and until then was quite feeble. Later she developed more rapidly, and while attending school was a comparatively robust child.

As far back as she could remember she had suffered from muscular stiffness which hindered motion, and made her incapable of any work which required skill, such as embroidering, sewing, etc. If she grasped an object firmly with her hand she could not immediately let go of it, and after doing so the fingers were only slowly extended and as if they were flexed by a spasm. If she knelt upon the floor, or sat for some time upon a chair, she was scarcely able to rise; her knees would be quite stiff and she could not extend them. The legs were like wood, and could be moved only with difficulty, so that her first steps were very awkward and uncertain. Gradually the gait would become normal, and would continue so provided the patient did not move about too long. After walking on an even surface for about half an hour she felt a tired sensation which rapidly increased and forced her to sit down in order to avoid falling. The ascent of steps was extraordinarily difficult because it was almost impossible for her to lift up her legs, and in descending she felt uncertain, and was constantly afraid of pitching forward. During excitement the patient performed all movements in a perfectly normal manner, and as if she had never been ill. In occasional quarrels with her playmates she fell down on the slightest push.

During cold weather she noted an aggravation of the ailment; she was always better in summer than in winter; during the menstrual periods and in the mornings she was less well than at other times. When she rose early, she could hardly drag herself from bed because of the stiffness in her limbs. She was worse at night only when she had been very active during the day; otherwise she was comparatively well in the evening. During the periodic exacerbations of her illness, speech and mastication were occasionally impeded, particularly if her mouth had been for some time closed, in which case syllables and words were at first enunciated with difficulty, for her tongue was as heavy as lead and appeared as if paralyzed; sometimes she was obliged to chew very slowly because she had to overcome rigidity of the jaw which sometimes continued as long as fifteen minutes.

The mental power as well as the memory of the patient was good so that, in spite of her infirmity, she advanced at school, and helped with the housework at home as

far as she was able. For a few years before coming under observation she had been employed in a button factory where easy work was assigned her. She had to regulate the lever of a metal press, but even this simple task was difficult for her to perform as it always required some exertion for her to remove her hand from the machine quickly and at the right time, and there was danger of her hand being caught in the machinery.

According to the patient's report, in the course of years her condition gradually became worse, that is, the symptoms had of late become more noticeable. She came to the hospital because of frequent attacks of syncope and spasms during the preceding three months. She attributed these to grief over the death of her mother, because, in three days after this occurred, she was several times attacked by spasm of the vocal cords which lasted several minutes (seven). The throat felt as if suddenly constricted. Her face became red, then blue, and speech was impossible. Eight days later, after some further excitement, she was attacked by general tremor and extensor spasms. While these lasted, the teeth were pressed firmly against each other, but the tongue was not bitten; there was loss of consciousness, but neither vomiting nor incontinence; the attacks lasted five minutes, after which there was great lassitude; memory returned after the lapse of half an hour. After that time there were four attacks of syncope, always after excitement. Immediately prior to the attack the patient several times had a sensation of fulness in the stomach and throat; it appeared to her as if bands were rising from the stomach. There was dyspnea, cold perspiration appeared on the face and hands, and loss of consciousness soon followed. There was neither paralysis nor disturbance of speech after the attacks, there was no derangement of the organs of special sense, no sensory disturbance, no pain; exceptionally slight pressure in the head, vertigo, and palpitation of the heart were noted. The menses appeared during her fifteenth year, but nothing anomalous was noted.

We pass to the *examination of the patient.* She was of medium size and graceful, with a moderate development of fat; her skin was pale, but she showed no other anomaly. The *internal organs* were normal. The urine was free from pathologic constituents; nothing of importance could be detected in the bones. It may be stated briefly that the organs of special sense functioned normally, and that no disturbances were revealed by the most minute sensory tests. Mentally the patient was normal, but was inclined to slight irritative conditions. She had no attacks of syncope while in the hospital. All of the *tendon reflexes* were normal. Dermographia was noted. We now turn to the apparatus which the patient most complained of as being disturbed —the *muscles.*

The muscles of the nape of the neck, particularly the trapezius and the sternocleidomastoid, were powerfully developed, and the shoulders were very muscular. Considering the graceful structure of the body, the development of the muscles of the back was conspicuous: These were by no means too plump, but the contours were as prominent as in an athlete under training. The arms also showed unusual development for a woman. The circumference of the upper arms was 27.5 cm.; the prominence of the biceps and the deltoid was quite conspicuous. The muscles of the forearm were just as markedly developed, and the thenar group was extremely prominent. The shoulder could be raised and the scapulæ approximated very rapidly. The arm could be quickly raised to its full height above the shoulder-joint. Testing the motor power, it was at once evident that the arm could be depressed without meeting resistance. Upon repetition the power increased, so that on a fifth or sixth test the arm could be depressed only by exerting considerable force. The lateral movement of the arm was excellent, the fall being sudden. The elbow-joint could be moved the more readily the more it was exercised. If the arm at the elbow-joint was extended to its maximum, and the patient was told to flex it, she could do so only very slowly as if overcoming strong resistance. Upon repetition of this exercise there was improvement, so that on the third or fourth attempt flexion could be performed with ease when the arm was extended to its maximum.

If the forearm was flexed to its utmost for some time, and the patient was then directed to extend it, this was promptly done and without difficulty. The same result attended the movement of the elbow-joint and the wrist-joint.

The fingers could be well extended, and were rapidly flexed from a position of extreme extension. But when flexed to an extreme degree, they could be extended only

slowly and with great effort as if controlled by a spasm. If the patient held an object, she could let go of it only slowly and after considerable effort.

In the *vertebral column* there were no pathologic peculiarities.

In the *lower extremity* the hyper-development of the muscles at once became conspicuous. The vasti and the gastrocnemii were especially prominent, and were very hard to the touch. The circumference of the middle of the right thigh was 50, of the left 49.5 cm.; the circumference of the calves bilaterally was 36 cm. The patient could quickly rise from a recumbent to a sitting posture, and if she were placed upon the floor could easily rise. The lifting of the leg extended at the hip-joint was at first very slow but was quicker on repetition, and gradually became normal; the power which was slight at first constantly increased; when vigorous movements were intended, slight spasm appeared; like symptoms occurred on all movements.

The test of the *mechanical irritability* by means of the percussion hammer was very interesting. Tapping the nerve trunks produced no reaction. But the results were very different on making short, sharp taps on the muscles. On the trapezius this soon caused a deep groove to form which, three to five seconds after stimulation had ceased, slowly and sluggishly disappeared. Similar results were noted in the deltoid, the biceps, and in the muscles of the forearm; there was no response in the triceps. This phenomenon was very conspicuous in the hyper-developed thenar muscles; on tapping these the thumb showed opposition, grooves being observed which in disappearing produced slight waves. Similar conditions were observed in the rhomboid, the infraspinatus, and in the vasti, and were especially marked in the gastrocnemii.

In the tongue, which was not hypertrophied, contractions appeared on firm depression but there was no rigidity. In the muscle groups innervated by the *cranial nerves* (the muscles of the face, the eyes, and of mastication) no pathologic changes could be determined. The pupils functioned normally. Electrical tests revealed the following: Different nerves, such as the crural, the facial, the peroneal, the median, etc., could be stimulated both by galvanism and faradism. Minimal contractions were everywhere produced by currents corresponding to the normal; galvanic irritation with brief induction quickly produced contraction in the muscles, but this lasted no longer than normal. In a few nerves a remarkably prolonged contraction could be produced with somewhat stronger currents; for instance, if the ulnar nerve were periodically stimulated the hand at once assumed its characteristic position. On opening the interruption electrode, the extremity did not at once return to its normal position but for some time retained its first position, as if stiffened; the same effect was seen upon galvanic stimulation, but these tests did not evoke reaction in all nerves. In some of the muscles prolonged contractions were produced, and these were quite remarkable. On faradic stimulation of the thenar muscles a groove slowly developed which was only sluggishly compensated for after withdrawing the stimulation. This groove was even more distinct upon galvanic irritation.

With a current of 5 milliampères a deep groove was produced in the deltoid; this persisted for from five to ten seconds after the removal of the electrode, and disappeared gradually. There was a similar reaction in the trapezius, in the biceps, in the extensor carpi radialis, in the antithenar, and in the interossei muscles. Here and there an undulating motion was perceptible, but, notwithstanding all my efforts, I was unable to produce true rhythmic waves over entire muscle groups.

When the tongue was stimulated by galvanism the contraction was sluggish, and disappeared gradually. Here also I was unable to produce waves.

Any one who has seen a similar case, or has read of these symptoms, will at once understand that we are dealing with a rare case of Thomsen's disease (myotonia congenita); this was even a greater rarity, for it was noted in a woman, whereas the condition is usually seen in men.

Here we note *heredity*—the mother appears to have had a similar affection. Here was a muscular development decidedly disproportionate with the otherwise graceful structure of the body; the history and the statements of the patient make it plain that it was difficult for her to produce movements, and the rigidity of the movements on sudden intention was demonstrated;

the patient had suffered since childhood from these conditions. We also succeeded in evoking the remarkable electrical reactions which are designated as myotonic, and this effect of mechanical irritation makes the diagnosis clear beyond all doubt.

I shall now discuss the *entire symptom-picture,* and will take this opportunity to explain other details of the case.

Our knowledge of the disease dates from Thomsen's noteworthy publication in 1876. We find that Bell (1832) and Leyden (1874) had previously reported cases which agreed perfectly with the symptom picture. Thomsen reported in his article that he had suffered from the affection, and that similar conditions had been traced in his family through four generations. Soon afterward Seeligmüller, Peters, Bernhardt, Strümpell, Moebius and Westphal also reported interesting cases. In 1886 and in 1889 Erb published his fundamental reports, and first demonstrated the electro-diagnostic phenomena of this disease, the mechanical irritability of the muscles, and their histologic relations, which he had carefully studied.

Microscopic results of an autopsy were detailed by Déjérine and Sottas, and Schiefferdecker and Schultze recently extended these researches; a number of authors have complemented the clinical picture, and have especially portrayed the myotonic symptom picture in combination with other nervous diseases. Briefly sketched, the symptom picture might be described as follows:

Males who are members of families in which the ascendants have suffered from similar conditions are most frequently affected; they are usually born with the affection; rarely do they acquire it later. The most prominent symptom noted is the colossal muscular development which is especially marked in the extremities; yet the muscular power is slight. The stiffness which appears upon intended movements, and which upon gross movements increases to spasm, particularly inconveniences the patient. On mechanical irritation, grooves form in the muscles and only slowly disappear. Strong electric stimulation with direct and indirect irritation often causes tonic evidences of contraction; faradic and galvanic irritation produce grooves in the muscles which only slowly disappear. In some cases galvanic waves have been observed.

The symptoms are chiefly noted in the muscles of the extremity, rarely in the trunk, and still more rarely in those muscles supplied by the cranial nerves. Sensation, the sensory sphere, and the reflexes are not involved.

ETIOLOGY

If we attempt to amplify this scheme we must first consider the *etiology.* We know of but one actual factor in this—heredity. There are reports of families four generations of whom have suffered from the affection, but the disease also attacks persons who do not come from myotonic families. It is interesting to note that other nervous affections have appeared in the ascendants, and sometimes also in the descendants, such as psychosis, neurosis, and alcoholism. This will recall the history of our patient.

We know that the malady is usually congenital; but few cases have been reported in which it appeared in later youth. We know the prevalence of

the affection in the male; briefly, there is remarkable, perhaps quite uniform, material which indicates that we are closely approaching the solution of this enigma. In some cases it has been maintained that fear or trauma had an etiologic effect. I believe these factors to be rather predisposing than etiologic.

SYMPTOMS

Among the conspicuous symptoms is hyper-development of the muscles. In our patient this peculiarity was evident, but the case could not be regarded as classic. In other cases I have seen over-development of the entire musculature. The muscles stood out plump and strong, and reminded us of the proportions of the Farnese Herakles.

The extremities are usually the chief seat of the affection, but the most varied locations have been reported. Thus, Oppenheim mentions a case in which the muscles of mastication and the muscles of the lower extremities were implicated while the arms were exempt. The motor power fails to correspond with the mass of the muscles: The patients are often only moderately strong or may even be regarded as weak. The *stiffness* of which they complain is even more disagreeable than the weakness. This becomes especially troublesome when they intend to perform a movement, the patients then feeling that they must overcome some resistance, and the muscles actually do resist the impulse of the will. If sitting, it is impossible for them to rise, if they are standing and desire to walk seconds pass before they can make the first step, which is slow and laborious, but after walking for one or two minutes movements often become quite normal. One of my patients reported that, when climbing a ladder as a school-boy, on the first attempt he always lagged behind the other boys. After he overcame this, he could reach the top sooner than the others. If, while he was sitting, he was told to rise he could do it only slowly and with effort. When he was ordered to step out, it required a few seconds for him to take the first step, after which he slowly proceeded forward as though dragging heavy chains after him. Gradually this condition so improved that after three or four minutes he could run with uncommon rapidity, and even succeeded in executing some quite agile jumps.

Besides this condition, which may be designated as *intention rigidity*, actual intention spasms, such as Seeligmüller described, are observed in almost all cases. Movements strongly intended often increase the spasm. If such a patient closes his hand tightly, and then tries to open it, this is absolutely impossible; after prolonged effort he succeeds in opening it slightly, then gradually more, but only spasmodically. Several minutes may pass before he can again extend his hand; similar conditions are usually observed in the lower extremity, often in the muscles of the trunk and the nape of the neck, in some cases also in the diaphragm. The muscles supplied by the cranial nerves are rarely implicated; probably the tongue is most frequently affected; the patients often complain of difficulty in moving this organ, and this symptom may very often—but by no means always—be the chief cause of the difficulty in speech. Some observers have noted myotonic disturbances of the muscles of the eye; for example, with strong intention a hindrance to the action of the bulbi. Oppenheim reported an interesting case of this in

one of his patients in whom the myotonic disturbance was distinct only in the orbicularis palpebrarum, while the other symptoms could be demonstrated in the remaining muscles of the eye. In isolated cases we also note disturbances in the muscles of mastication and of the face. Severe spasm is produced not only by marked intention; sometimes severe shock, such as a fall, may cause rigidity. Often such persons are for several minutes unable to rise, and the body is as stiff as if it were made of wood.

Excitement often decidedly increases the symptoms. In the morning and when the patients are alone, they are able to walk quite well and to work, but movements are decidedly hindered when they are under observation or are excited. As school-children they suffer greatly, also as young men during military service. Mental stimulation which is not exciting and pleasant influences often lessen the disturbance. In contrast with this, in our patient, excitement often had a beneficial effect. Hard physical labor, general somatic affections, the immoderate use of alcohol, are all deleterious factors. Cold has a particularly unfavorable effect, and in some cases this is the only factor which causes any disturbance.

Jensen made minute myographic studies into the nature of the muscular contraction, and determined that the first contraction of the myotonic muscle consumes a much longer time than normally, and that it is chiefly the coördinates of the decrescents which appear to be so markedly slowed. Accordingly the myotonic muscle very slowly relaxes, the process consuming more than 8 seconds, while the normal decrescent requires but about 0.2 of a second.

The curves show that it is the terminal portion of the process of relaxation which is chiefly slowed, this latter portion usually but not always appearing to be quite sharply defined in comparison with the first, which, however, also shows a distinctly abnormal slowing. These abnormalities in the course of the myotonic curves gradually lessen, and after about ten contractions there is a return to a normal condition. The reaction of the motor nerves and muscles to *mechanical* and *electric* stimulation has been described in the history of our case. We concur in the classical description of Erb. We were unable to demonstrate the waves noted by this author, but this symptom appears to be quite rare in Thomsen's disease, for later investigators were often unable to demonstrate it.

The mechanical irritability of the muscle is never increased, the faradic resistance to minimal contractions is normal, and no prolongation appears. With stronger and longer continued currents there is a *prolongation*, but brief stimulations do not produce it. Galvanic currents applied for a short time elicit prompt reaction from the nerve but no prolongation; labile currents undoubtedly evoke prolonged tonic contractions. There are no peculiarities in the form of the contraction.

In the muscles after mechanical stimulation Erb describes contractions similar to those we have mentioned. "Even the pressure of the tip of the finger upon the muscle, and still more so tapping with the percussion hammer, will produce sluggish tonic contractions in the muscle bundle, and, according to the position and size of the muscle, these appear as deep grooves, furrows, or rolls. The contractions are decidedly prolonged, and, according to the strength of the stimulation and the tendency of the muscle, last from 5 to 30 seconds."

All muscles respond readily to faradism; with brief currents which produce minimal contraction, or with a single opening contraction, there is no prolongation; with somewhat stronger currents the contractions may last for 20 seconds. In some cases, when individual muscles are included between the two electrodes, a peculiar, irregular wave of the entire muscle mass may now and then be observed (Petrone, Bernhardt, Erb). Direct *galvanic* contraction is occasionally increased. The anode is said to act stronger than the cathode. In our case the latter symptoms could not be determined; the contractions were peculiarly sluggish. This qualitative peculiarity was only manifest after strong currents, never after weaker ones. If the intensity was increased, a depression slowly appeared, remained for from 5 to 10 seconds, and disappeared gradually and sluggishly.

The phenomenon of rhythmic wave-like contractions with a stabile current (which could not be demonstrated in our case) is, according to Erb, best evoked if the electrode is not placed directly upon the muscle but upon the end of the tendon, or somewhat deeper. If the current is sufficiently intense, contraction first occurs, and is succeeded by an irregular, undulating movement of the muscle, and this soon merges into an ordered, regular wave movement. More or less extended waves of contraction follow more or less rapidly: They begin at the cathode and move toward the anode. The electric phenomena are sometimes labile, but are subject to variations.

This exhausts the list of the principal symptoms. It should, however, be mentioned that the tendon reflexes are occasionally diminished, sometimes increased. The sensory functions and those of special sense are normal. Ataxia has not been described. There is never marked mental deterioration, but the patients have a peculiarly anxious and shy expression. They are ashamed of their affection, never speak of it, and, owing to this peculiarity, often do not consult a physician until after the lapse of years.

It is readily understood that every possible nervous condition may be combined in these individuals who belong to neurotic families. A number of hysterical peculiarities were manifest in our patient.

It is interesting to note that *bladder* and *rectal disturbances* are never mentioned, although the external sphincters contain transverse-striped muscles, and the implication of almost all other transverse muscle groups has been reported. This intactness of the external sphincters is conspicuous also in myasthenia and in the tetany of adults, which reminds us of the peculiar fact, also referred to by Oppenheim, that these muscles often have the physiological action of smooth muscles and even absolutely resist curare (as A. Fröhlich and I first pointed out).

A number of other nervous symptoms may be due to the fact that the affection is combined with various organic nervous diseases. Chief among these is muscle atrophy. As Hoffmann strongly maintains, this affection seems to undergo a direct change into muscle atrophy. In his opinion, this is favored by the fact that in a few cases myotonia preceded the muscular atrophy, that in the case described by Nogués et Sirol the patient's father suffered from myotonia and his son suffered from myotonia and muscular atrophy, and that the three children reported by Pelizaeus also had myotonia but did not all have muscular atrophy. On the other hand it is not clear that muscular atrophy always precedes myotonia, but in all cases that have

come under observation myotonia was found at the first professional examination, also muscular atrophy, although usually as a *forme fruste*, i. e., to a mild extent and not noticeable to the patient. It was determined not only in the atrophied muscle alone, although most distinct in this, but was widely distributed in muscles of great volume and good power. Finally, in all the cases except Hoffmann's the localization of the atrophy differed more or less, it neither corresponded to a conspicuous spinal type nor did it belong to the well known myopathic varieties. Other isolated cases (Dana, Déléage, Bettmann, Hoffmann) seem to show that muscular atrophy accidentally occurred in patients with myotonia. We shall revert to these conditions in the differential diagnosis, and shall then more minutely discuss the question of tetany.

Combinations of this affection with tabes have been described by Hoffmann and Nalabandoff—but these may have been due to accidental conditions. Kaiser reports a peculiar case which shows a combination of myotonia and athetosis.

Oppenheim once saw the disease combined with a congenital muscular defect, at another time with a congenital defect in the formation of the fingers.

PATHOLOGIC ANATOMY

Pathologic anatomy indicates that we are dealing with a primary disease of the muscles; that is, the only autopsy, which was in the celebrated case reported by Déjérine and Sottas, gave entirely negative results in so far as the nervous system was concerned.

The first investigators also found negative conditions in the muscles, but Erb succeeded in demonstrating hypertrophy; a number of other authors confirmed this remarkable discovery, and contributed additional details, particularly Fischer, Déjérine and Sottas, Martius and Hansemann. The latest and most comprehensive study we owe to Schiefferdecker and Schultze. The foundation of all these cases is the following: *Hypertrophy of the primitive fibers*; increase of the sarcolemma nuclei. As to the most recent methods of investigation, a quotation from Schiefferdecker's report will best present these. Examination of the deltoid muscle from a person attacked by myotonia showed a marked distribution of the fibers; the sarcoplasma presented an extremely characteristic and remarkable change in that, after fixing in formol (Jores), it was seen to be more or less extensively permeated by small and peculiar nodules which have thus far been found in no other muscle, either of man or animals. This granular condition was most likely the expression of a specific disease of the sarcoplasma; the number of nuclei was much above the normal (128 to 290). The volume of the nucleus had been decidedly increased without its composition undergoing change; it is important to note that the bulk of the nucleus had remained unaltered. This observation corresponds with that made by Déjérine and Sottas that the first phenomenon of myotonia congenita is the hyperplasia of the nuclei, which is naturally attributed to disease of the sarcoplasma. The fibrillæ of this muscle show peculiar changes in which apparently the transverse fibers are chiefly implicated, but which probably depend upon disease of all the fibrillæ, and the cause of which is most likely to be found in disease of the sarcoplasma. This change in the fibrillæ seems to start at the margins of the fiber and

gradually proceeds toward the center, indicating a change in nutrition which proceeds from the periphery; it does not favor the supposition that, perhaps as the result of marked thickening of the fiber, the nutrition no longer suffices, since, otherwise, the first changes in the fibrillæ should take place in the center of the fiber. In regard to the clinical observation that myotonia undergoes a transition into progressive muscular atrophy, Frohmann makes the interesting report that he succeeded in demonstrating numerous atrophic fibers between the hypertrophic ones.

The *nature of the disease* is still obscure. Erb ascribed it to an anatomical (histologico-chemical) change in the transverse striped muscles. This did not solve the question whether we are dealing with pure myopathy or with a muscular change due to nervous trophic influences and disturbances. In favor of the first view is the negative necropsy finding in the nervous system, such as Déjérine and Sottas found nine years after the publication of Erb's book. As favoring the *possibility of nervous influences* the following might be adduced: 1. That there are no spasms in the primary muscular affection; 2. That those affected often belong to families in which degenerative nervous diseases prevail, the patients themselves suffering from all varieties of nervous affections (like the patient whose history I quoted).

A few authors attempted to solve the enigma by attributing the disease to an *anomaly of metabolism.* Moltschanow showed that in his case the amount of urea in the urine was decreased as well as chlorin and uric acid. Wersiloff found a great diminution of creatinin. Bechterew demonstrated in one case a decrease of urea, phosphoric acid and the chlorids, while the quantity of uric acid was at one time decreased, at another time conspicuously increased. The latter author formulated the hypothesis that myotonia is a peculiar derangement of the metabolism in which toxic products are set free in the organism and have a poisonous effect upon the muscular tissue.

DIAGNOSIS

In typical cases the *diagnosis* is very easy. The heredity, the congenital nature of the affection, the hypertrophy of the muscles, the peculiar electromechanical reactions, the intention spasms, are all so characteristic that doubt can scarcely arise. It must be mentioned, however, that the affection has sometimes developed only in late youth, and cases have been observed in which, although there was no muscular hypertrophy, all the other symptoms of the clinical picture could be demonstrated. The diagnosis becomes perplexing when myotonic symptoms are combined with *muscular atrophy.*

In 1888, in the *Zeitschrift für klinische Medicin* I reported for the first time a typical muscular atrophy with intention spasm, and periodical, true muscular hypertrophy (which could be miscroscopically demonstrated), also a sort of myotonic reaction; the spasms subsequently disappeared. When, years later, H. Schlesinger saw the case partial sensory paralyses had appeared; this author mentions in his monograph, "Syringomyelia," that in this disease myotonic symptoms not rarely develop in individual muscle groups, and he refers to a *myotonia syringomyelitica* which should be differentiated from true myotonia. A. Fuchs recently demonstrated a case with myotonic phenomena in a symptom-picture which revealed the characteristics

of amyotrophic lateral sclerosis. When we recall that Hoffmann accepts the view of the transition of Thomsen's disease into muscular atrophy, and if we bear in mind that localized muscular atrophy may be combined with myotonia, we can readily understand that there may be transitional stages between these symptoms; one investigator may in one case make a diagnosis of myotonia undergoing transition into muscular atrophy, while another investigator may diagnosticate a myotonic symptom-complex combined with *muscular atrophy.*

We have recently been considering another extremely interesting combination. This is *tetany with myotonic symptoms.* Schultze was the first to call attention to this peculiar association, later Kasparek, Bettmann, Hoffmann, v. Voss and I; recently the subject has been minutely studied by Schultze. In these cases we refer merely to myotonic symptoms in tetany, not, however, to a combination of the latter affection with Thomsen's disease, because neither clinical nor histologic investigation has ever revealed muscular hypertrophy. Tetany has been ascribed to various conditions. We know of cases which followed extirpation of goiter, also of gastrointestinal tetany, and the so-called idiopathic workman's tetany. Notwithstanding the fact that I saw many cases of tetany in Vienna, the symptoms described were only twice observed by me, and were then typical intention spasms. Other authors have reported the same. Occasionally there may be some rigidity on an attempt at movement. Schultze and others have demonstrated a mechanical myotonic reaction of individual muscles, particularly of the tongue; in other cases electric reactions resembling myotonia were evoked. In proof of the view that these are not *combined forms of true Thomsen's disease* is the circumstance that a decrease of the myotonic symptoms is sometimes observed simultaneously with a decrease in the symptom of tetany.

In a *differentio-diagnostic* respect it may be interesting to mention that *intention spasms* (without myotonic reaction) occur also in *other* diseases of the nervous system, usually in severe spinal affections. In these cases the spasms not only occurred in the muscles which were actively tense, but were also noted in distant groups; similar conditions have occasionally been observed in hysteria. In a patient convalescing from enteric fever Nothnagel once saw general spasms of the flexors and extensors upon flexion of the elbow. In examining the patient whose history I have related, spasms were sometimes observed not only in the muscles which act simultaneously, but also in the antagonists.

Some vague resemblance to intention spasm is found in spastic *occupation neuroses,* but minute investigation will at once lead to a correct diagnosis.

In concluding our differentio-diagnostic study, we must describe the conception of paramyotonia. Eulenburg designated by this name a disease which resembles Thomsen's disease, being a hereditary or congenital form of "muscular idiosyncrasy," which has been traced through six generations of one family. In the individuals affected, this anomalous action was evident throughout the entire muscular system, and produced marked phenomena of spasm and of rigidity in individual muscular regions, and in others an inhibition of movement. A spasmodic rigidity followed exposure to cold; in some muscles it disappeared rapidly, in others more slowly, to give place either to the normal condition or to a paresis-like immobility which persisted

for some time. No myotonic reaction could be demonstrated. Hlavacek reports a combination of paramyotonia with myotonia.

As far as life is concerned, the **prognosis** of the disease—I am now speaking of typical myotonia—is absolutely favorable. In genuine cases no cure has as yet been observed, but remissions and improvements are occasionally reported. As a rule, the progress of the disease is not rapid.

Therapy has as yet yielded no brilliant results. Systematic massage and gymnastics appear to form the best mode of treatment. In the patient whose history was related the former method had no marked effect. Some authors maintain the beneficial effects of hydrotherapy, of spa treatment, of carbonic acid springs. Galvanism and faradism have frequently been tried. In regard to internal treatment, iron, arsenic, strychnin and sodium iodid may be of use.

LOCALIZED SPASM

By E. REMAK, Berlin

For practical purposes spasms limited to individual muscles or muscle groups are best considered in one article, since only the most minute differential diagnosis will enable the physician to group the case that he observes under one or the other pathologic form.

Although, in accordance with an old custom, text-books still include limited muscular spasms among diseases of the peripheral nerves, it has been for some time recognized that they are only rarely due to this cause, and even then there must be a reflex stimulation and increased irritability of the nervous centers and tracts, either of the spinal cord or of the brain. Clinicoanatomical experience in organic diseases has led us to some conclusions as to the parts of the nervous system implicated in localized spasms, for they are accompanying symptoms of such diseases. Among these we rarely find neuritis, but frequently meningitis (opisthotonos), among spinal diseases those of the anterior gray substance (fibrillary contractions), next of the pyramidal lateral column tracts (spasms of spastic spinal paralysis and amyotrophic lateral sclerosis), of the posterior columns (athetoid contractions of tabes), among cerebral diseases those of the pons (contractures in the course of the facial nerves and trismus), and especially of the motor area of the cerebral cortex. The experimental and clinical findings of the last three decades which indicate the exact localization of the motor centers in the anterior central convolution, and their relations to definite muscular regions in cortical spasm or so-called Jacksonian (partial) epilepsy, have chiefly shown that they may begin and run their course locally, according to their point of origin. In these investigations of the point of origin of localized spasms in cases in which there was no organic disease, and also concerning the various forms of spasm produced, an attempt was made to locate the ganglion centers, etc., and to localize and describe the form of the partial spasm. An important guide was the distribution (whether unilateral or bilateral, whether throughout one or several nerve tracts), as well as the form of the spasm itself. The symptomatologic and minute classification lately proposed for the differentiation of the muscular spasms distributed throughout the body, and for the recognition of several special pathologic forms of quite different pathogenesis, namely, of myokymia, myoclonia, myotonia, tetany, chorea electrica, chorea rhythmica, and even of tic, is the result. All of these affections must, in the individual case, be considered as localized spasm, because they occasionally appear as limited spasms and remain so for some time, which is also true of the initial stage of tetanus, of some forms of

tremor, especially of paralysis agitans, and of chorea and athetosis in which involuntary movements are occasionally spasmodic without being for this reason strictly designated as localized spasms. The etiologic relations of the most common general neurosis, hysteria, are sometimes more intimate than those of the former neuroses. Aside from the fact that hysteria produces spasms and contractures of typically hysteric character, this "grande simulatrice" may also resemble other forms of localized spasm. Authors hold different opinions as to whether some of the general spasm neuroses (myoclonia, chorea electrica, chorea rhythmica, and tic) are merely special expressions or are forms of hysteria. Lately, however, the psychogenous (mental) origin of some localized forms of spasm other than hysterical has been urged by French authors, and their strict differentiation as tic from spasms due to organic or functional disturbance has been declared to be important, because in these psychical treatment alone is said to be effective.

DIFFERENTIATION OF FORMS

This review shows how necessary is an exact classification in every case of localized spasm—for prognosis as well as for treatment. Only when the obvious symptoms of localized spasm are recognized, does the diagnosis begin.

Muscular spasm may be broadly defined as an involuntary, therefore apparently spontaneous, muscular contraction which is not due to external, mechanical, electric, or thermic stimulation. Muscular spasms, such as *intention spasm* and *occupation spasm,* may be associated with voluntary movements. *Myotonia* is the spasmodic arrest of the voluntary contraction of the muscles (Thomsen's disease).

All muscular contractions do not necessarily lead to a shortening of the muscle, and need not even implicate the entire muscle. Distributed in naked persons who are cold, localized in progressive muscular atrophy which is of spinal origin, and in amyotrophic bulbar paralysis, the *fibrillary contractions* may involve only isolated bundles of muscles. Widely distributed and continuous, wave-like, fibrillary, muscular contraction, previously described by Kny, was designated by F. Schultze in 1895 as *myokymia.* Hoffmann (1896) and others have observed its local occurrence.

Spasms are strictly differentiated, according to the rapidity of their course, as *tonic* and *clonic.* Clonic spasms which last but a moment have been designated as *lightning-like*; for example, those in the chorea electrica of infancy, as Henoch called it, and in myoclonia (polyclonia) which are probably identical. Clonic contractions have not rarely appeared as fibrillary muscular contractions; for example, in so-called idiopathic facial spasm.

Contractions of restricted distribution, in more or less rapid rhythmical sequence, and affecting the antagonistic muscular groups, produce various *tremors* which are not included among muscular spasms; only their most severe forms lead to so-called *shaking palsy.*

When muscular contractions follow one another in such rapid succession that the muscle does not have time to regain its position of rest, these spasms are designated as clonic. When the contractions last for a considerable time we speak of them as *tonic,* and, if extremely violent, as *tetanic* spasms. Limited but painful tonic spasms, especially those so common in the calf of the

leg, are designated as *cramps*. Extensive and violent spasms of several muscle groups of a member are called *convulsions*; epileptic or hysterical spasms are types of these.

Permanent, tonic, muscular spasms lead to *contracture*. In so far as this is an accompanying phenomenon of organic diseases (for example, cerebral hemiplegia and spastic spinal paralysis), also of hypertonia and increased tendon phenomena, we are not here concerned with it, nor with the contractures of paralysis agitans, of catalepsy, etc. In a differentio-diagnostic respect the frequently localized *hysterical contractures* must be considered. Contractures which persist after severe peripheral paralysis, particularly of the muscles of the facial nerve which were formerly paralyzed, do not depend upon muscular spasm but upon interstitial retraction and induration. But to these, clonic contractions may be added. Usually these occur as *conjoined movements* of voluntary or of involuntary muscles, especially in the face and in the eyelid. Voluntary movements often produce a local attack of spasm; for example, mastication and speaking evoke attacks of idiopathic facial spasm. Some forms of spasm are caused only by definite coördinate movements (writer's cramp, etc.).

Between clonic and tonic spasms and contractures we observe transitional stages. Clonic spasms may show frequent alterations, first becoming tonic, then clonic. Tonic spasms sometimes lead to contractures.

None of these forms of spasm produces voluntary movements, but simpler ones which differ from the voluntary by being arrhythmic, sometimes rhythmic, occurring in succession, and often reminding us of reflex contractions. Even when peripheral irritation and organic diseases of the reflex tracts cannot be determined, we may assume that the localized spasms are due to an irritative condition at some point in the reflex arc, but it frequently remains doubtful whether the motor nuclei of the spinal cord, of the medulla oblongata, or of the pons must be considered the reflex center, or whether higher cerebral ganglia, even those of the cortex, are implicated. *Spasms* of *cortical* origin are characterized by marked contractions usually of slow rhythm, which repeatedly, often paroxysmally, pass gradually from one muscle group to another, and consist of clonic contractions of synergistic muscles (for example, the muscles of the arm, the extensors and flexors of the finger, and of the elbow, the abductors and adductors of the shoulder); these do not, however, give us the impression of being true coördinate movements. As the cortical spasms are perhaps due only to irritation of the so-called sensory spheres of the cerebral cortex, for which the not uncommon initial paresthesias may be clinically utilized, it is not impossible that the cortical spasms are also to be attributed to reflex cause.

Following the example of the French authors (Gilles de la Tourette, Charcot and Guinon, Brissaud and others), for more than twenty years *maladie des tics* or *tic convulsif* has been differentiated as a spasmodic affection which runs its course with apparently voluntary, systematic, coördinate, complicated, and often grotesque movements, the contortions appearing in the face as grimaces. It is probably quite correct to assume that, perhaps in consequence of some transitory peripheral irritation, these developed from habit, and owing to an absence of inhibition were repeated under constraint (automatically); at all events, they originate from psychomotor coördination centers.

If these psychogenous spasmodic forms, which may also be local, are designated as "tic," the nomenclature at once becomes perplexing, since the term "tic convulsif" now means something quite different from what it formerly signified. In contrast to tic douloureux (neuralgia of the fifth nerve) we mean by tic convulsif the common, unilateral, idiopathic, facial spasm. To prevent confusion Jolly, in 1892, proposed designating this as "*tic impulsif*." For the same reason, Marina had previously (1888) proposed that we distinguish *myospasia impulsiva* from *myospasia simplex*.

But in tic impulsif, during the periods between the attacks, coördination of voluntary movements is as little disturbed as in intermittent localized spasms. This enables us to differentiate all of the localized spasms, including the tics, and is in contrast to *chorea*, in most of the forms of which choreiform unrest (*folie musculaire*) persists in the periods between the attacks and disturbs the coördination of voluntary movement to a still greater extent. This is also true of posthemiplegic chorea occurring in organic hemiplegias and diplegias, especially of that subdivision described by Hammond in 1871 as *athetosis*, which is characterized by continuous motility, in the main increased by voluntary movements, and usually located in the ends of the extremities, especially the fingers. Idiopathic bilateral athetosis also becomes especially noticeable upon voluntary movement.

If neither chorea nor athetosis can be grouped with localized spasms, nevertheless these affections may be combined. In 1898 F. Schultze showed that contractions and lightning-like spasms of the individual muscles (monoclonia) are observed in chorea as well as combined with choreic movements. In 1888 I reported the case of a man, aged 39, who had suffered from a left-sided hemiplegia ever since he was a child of eighteen months, and who, besides a left-sided, posthemiplegic hemiathetosis, was also afflicted with persistent rhythmic, clonic contractions of the left platysma myoides occurring from 96 to 120 times per minute.

To the above rule that muscular unrest in chorea is increased by voluntary movement, *progressive* (and usually *hereditary*) Huntingdon's *chorea* is an exception. In this affection the choreic movements in the limbs are voluntary, and sometimes cease, that is, are inhibited, by voluntary movement, which may cause a superficial resemblance to tic impulsif. Since we are in this malady invariably dealing with a general disturbance, a neurosis, there is no necessity for a minute description.

On the other hand, *chorea rhythmica* must be grouped with localized spasms. French authors especially, perhaps erroneously, have designated as chorea rhythmica the rhythmical, forcible, and usually bilateral contractions of synergistic muscle groups. In this affection, probably because of hysteria, coördination of voluntary movements is quite undisturbed during the periods between the attacks. This is also true of Henoch's *chorea electrica*, the identity of which with polyclonia (paramyoclonus) was previously stated to be very likely, as well as that it may occur locally.

SYMPTOMATOLOGY

In most forms of localized spasm there are no pathognomonic symptoms except spasm. Aside from the local contractions reflexly due to neuralgia,

even *pain* is not usually an accompanying symptom, and it is prominent only in the well-known *cramps in the calves*, some forms of *myokymia*, and in attacks of *tetanus* and *tetany*. Localized clonic, and even tonic, spasms and contractures are generally painless; but the feeling of contraction may become annoying.

Spasms lasting for years, for example, of the facial nerve, may be unaccompanied by paralysis or muscular atrophy. But after long-continued and violent spasms, especially of the muscles at the nape of the neck and in other parts of the neck (spastic torticollis) there may be slight *hypertrophy* of the same muscles, especially of the sternocleidosmastoid and trapezius. A transitory *paralysis* or *paresis* after localized spasmodic attacks is characteristic of cortical spasms, and on repeated attacks cerebral monoparesis or monoplegia may result. Quite exceptionally, especially when peripheral cranial nerves have been subjected to pressure from basal tumors, it has been noted that peripheral paralysis, for example, of the facial, is preceded by the irritative symptom of localized contractions.

A typical combination of spasm (contracture) and paralysis of peripheral nature is shown by Rose's *head tetanus* which follows an infection of wounds of the face by tetanus bacilli, and first appears as trismus with unilateral paralysis in the region of the facial nerve (of the same side), and also with symptoms of contracture.

Changes in the mechanical and electrical irritability of nerves and muscles are rarely evident in localized spasms, even though of long continuance. A pathognomonic *increase of irritability* is noted only in *tetany*, the attacks of which are characterized by an obstetric position of the hands, most marked in the region of the ulnar nerve. I refer the reader to the article upon tetany as to the increase of mechanical, electrical, and particularly of galvanic, nerve irritability, even at the onset of the contractions (AnOC and CaOC, also AnOT).

According to all the nerve specialists of Berlin, tetany of adults is a rare affection in that vicinity, while in Heidelberg and Vienna, for example, it is common. It is therefore remarkable that *infantile tetany* (in which Escherich, Ganghofner, Mann, and Thiemich have also demonstrated an increase of galvanic nerve irritability which, according to Finkelstein and Japha may be associated with or may be without spasm of the glottis) should in Berlin so frequently be almost latent, and manifested only by a galvanic hyperirritability of the ulnar nerve and relieved by a change of food (discarding cow's milk). This condition, revealed only by an increase of the electric contractility, is unknown in the adult. On the contrary, according to my own and foreign researches it may be considered the rule that in localized clonic and tonic spasms persisting for years the electric contractility is normal; this may be shown, for example, in idiopathic unilateral facial spasm, by careful comparison with the healthy side. The same is true of so-called occupation spasms, particularly of writer's cramp. Only myotonia, which is characterized by the spasmodic continuance of every voluntary movement (mostly congenital) and increased mechanical irritability of the muscles with groove formation on percussion, as well as the contractions which persist with a sluggish course after faradic and galvanic irritation, have been shown to be myotonic reactions.

A *diminution* of faradic and galvanic *irritability* as well as *DeR* of the muscles is noted in localized spasms only when they complicate a *degenerative neuritis* or are dependent upon it. I saw this complication in 1889 in a case of occupation neuritis with milker's spasm, and I have mentioned a similar case of traumatic neuritis in my work on "Neuritis" (p. 94 et seq.). This decrease in contractility and the spontaneous contractions or constrained movements which appear with contracture in cases of peripheral facial paralysis that are not yet cured are typical instances of neuritic and muscular degeneration and regeneration. The same is true of the *athetoid spontaneous contractions of polyneuritis* described by Löwenfeld, E. Remak, Korsakow, and others, which I have explicitly described in my book (p. 380 et seq.).

When *sensory disturbances of the skin or of the articular sense* are perceptible with or after localized spasm, these do not depend upon the spasm, but upon the cause of the affection. If they follow cortical spasms, for instance, they may be due to organic disease.

When associated with neuritic contractions and spasms, they may be perceived in the diseased nerve areas, as in my case of milker's spasm. The majority of hysterical spasms and contractures run their course with sensory disturbances, especially hemianesthesia.

In localized spasm *reflex irritability* is often normal. It is sometimes so far altered that spasms may be readily evoked at definite pressure points. Occasionally, however, they may be prevented by this means, as in some forms of facial spasm known since the time of v. Graefe and R. Remak. In hysterical spasms these pressure points are also detected in the area of the spasms as well as in remote regions, for example, in the so-called "ovarie." Distinctly abnormal cutaneous reflexes, such as Babinski's toe reflex, may be recognized in spasms of the lower extremity which depend upon organic foundation (disease of the lateral columns).

In localized spasms the *tendon reflexes* are by no means invariably altered. Sometimes they are increased, especially in myoclonia, tic, and hysteria. Unilateral increase or decrease of the tendon phenomena on the same side as localized spasm almost always favors an organic disease. After cortical attacks a unilateral increase of the tendon reflexes, especially the foot phenomenon (dorsal clonus), may denote the development of monoparesis or hemiparesis.

ETIOLOGY

From this description it becomes evident that there is no uniform *etiology* of localized spasm. It is clear, however, that if in the so-called idiopathic forms we exclude a peripheral irritative factor or an organic nervous disease, we may frequently determine a certain spasmophilic, neuropathic predisposition. Not rarely nervous headache and severe hemicrania have preceded; emotion, care, and sorrow are sometimes incidental causes. Over-exertion and an improper method of carrying on their work will often lead to occupation spasms. The family history of patients suffering from myoclonia and tic frequently indicates a neuropathic predisposition.

COURSE

The *course* of localized spasm is far less readily outlined than that of peripheral paralysis; when definitely localized a further distribution is likely. For instance, if there is localized spasm with succeeding paresis, during a subsequent attack other regions may be involved in a form of cortical epilepsy due to focal disease, in encephalitis, in meningitis, or even in a beginning progressive paralysis. Trismus makes us dread an outbreak of tetanus, a tonic spasm of the ulnar nerve one of tetany. Myoclonia which is at first localized may develop into polyclonia; a localized unilateral tic may become a bilateral or general tic. A hysterical and localized spasm or a contracture, even if it ceases, frequently recurs or may implicate other regions. Even in the spasmodic attack strictly localized to a nerve region a favorable course can be expected only in those rare cases in which peripheral irritation is recognized and removed. Usually the spasm becomes chronic with remissions; I have known it to remain quiescent for decades. Although we cannot promise recovery in such cases, we may comfort the patient with the well-founded assurance that the further extension of his distressing affection is impossible.

TREATMENT

Provided no organic disease can be detected, the *treatment* of localized spasm must be directed to the cause; if an underlying disease be found, it must receive appropriate treatment. If, for example, a cortical disease has been diagnosticated, and this, owing to its nature, cannot be relieved by internal medication (iodin preparations, etc.), as soon as its exact seat has been determined the operation of trephining is indicated. If peripheral irritation cannot be relieved by absorbents, we must resort to surgical measures; for instance, extraction of a tooth, or, in cases of incarceration of sensory nerves, the pressure from the cicatrix or callous mass must be relieved by *neurolysis*. On the other hand, *stretching of a nerve*, particularly of a motor nerve, has not fulfilled the hopes at first entertained concerning it. There is relief so long as the paralysis lasts, but this often torments the patients more than the preceding spasm; when the paralysis disappears the spasm usually returns. Therefore, with spasm in a muscular area the function of which is not too important resection of the motor nerve is preferable. As, however, the motor innervation of spasmodic muscles is frequently supplied by several nerve branches, or even by nerve trunks, here also our treatment often fails. It is probably for this reason that Kocher and others have been so successful, especially in stubborn cases of spasm of the muscles of the nape of the neck, their method being the successive severing of the spasmodic muscles.

Before deciding upon operative measures, however, we should try both by systematic internal and external treatment to diminish the tendency to localized spasm. Among internal remedies the preparations of bromin, valerian, belladoma, arsenic, and tincture of gelsemium, have not, as a rule, been markedly beneficial. Curare, atropin, antipyrin, etc., have been tried subcutaneously. Of external methods electricity, usually the stabile galvanic current, on prolonged use has given the most satisfactory results. The best

method is to apply an anode having a diameter of 10 to 15 centimeters with a strength of current of from one to two milliampères either to the painful pressure point, to the pressure point which inhibits the contraction, or at the exit of the motor nerve. Although success is by no means certain, the frequency and intensity of the attacks will often be diminished. Other methods, such as applying the induced current to the antagonists not affected by the spasm, have fallen into disuse, and should at most only be resorted to for the relief of hysterical contractures when the suggestive element in electrotherapeutic treatment is likely to have an effect.

Brissaud and his pupils, Meige and Feindel, have proposed a special treatment for *tic impulsif*, namely, *psychomotor education*, which in the main is identical with Oppenheim's inhibitive treatment. It consists of practice in maintaining a position of rest; first, the patient is taught by the aid of a mirror to keep the affected area immovable, second to practise on command and before the mirror slow, correct movements of the muscles involved in the tic.

In the cure of occupation spasms, especially of writer's cramp, practice in new coördination movements, the method being alternated with gymnastics, massage, and electrotherapy, has proven most beneficial. At all events, these modes of treatment are more rational than hypnotic suggestion, success from which has been reported in isolated cases.

CLINICAL VARIETIES

In passing to the description of localized spasm in various muscular regions, we must refer to our previous description both for their symptomatologic and pathogenic classification as well as for their treatment. After examining the patient, and taking into account according to the rules of neuropathologic diagnosis, all the symptoms that have appeared, we must first decide the cardinal question, whether or not the spasm is the symptom of an organic disease (cerebral, bulbar, spinal, meningitic, or neuritic). Next we must consider whether it is reflex or idiopathic; in the latter case, whether it is fibrillary, clonic, or tonic, whether it indicates contracture, or is limited to a definite nerve region. If these points are decided in the negative, we must next ascertain whether tic impulsif is present, also whether the localized spasm represents its onset or that of general spasmodic neurosis in which hysteria especially plays a part. It is at once obvious that with rare exceptions occupation spasms implicate only the upper extremity.

Group I.—*Spasm of the muscles of the face* in its various forms is the most common of all localized spasms. Among my 244 cases of all kinds of localized spasm, in 109, therefore 44.6 per cent., the facial muscles were affected.

(1) Bernhardt in 1902 described as *myokymia of the facial nerve* a permanent spasm of the contracted left half of the face in a woman aged 27; this disappeared within 3 months. Newmark and Vitek have since reported analogous cases which were right-sided, and also terminated in recovery. In Newmark's case, however, the symptoms of multiple sclerosis soon afterward appeared, and this proved fatal. In 1886, among the early symptoms of multiple sclerosis in an engineer, then 21 years old, who was under my ob-

servation for ten years, I saw various muscular contractions in the forehead, eyelid, cheek, and lips of the left side without any sign of contracture.

(2) In a few cases of severe *trigeminal neuralgia* of the second branch I observed at the height of the attack undoubted *reflex clonic and tonic contractions* of the muscles of the eye and cheek with retraction of the mouth. Such localized spasmodic attacks from pain are, however, not always strictly limited to the region of the facial nerve. For instance, in a girl aged 5, who had toothache with inflammatory swelling of the gums, I afterward saw clonic contractions of the left angle of the mouth and of the muscles of the forehead. Simultaneously the eyes turned to the left and spasm occurred in the shoulder. On improvement in the gums, the attacks ceased.

(3) Painless, unilateral, "*idiopathic*" *spasm of the muscles of the face,* which has several times been mentioned (*tic convulsif* in the earlier sense) is the prototype of an attack strictly confined to a nerve region (the facial nerve) in which clonic and tonic spasms occur. I have reports of 46 cases among which there were 17 males and 29 females. The predominance of females has already been commented upon by Gowers and others. In my cases of both sexes spasm was more common upon the left side (11 males, 18 females). My youngest patient was a boy aged 9, my oldest a woman, aged 66, who had suffered from the attack for six months. In several of the older patients, however, the affection had begun 20 years or more previously.

The contractions, as a rule, first appear in the muscles of the eyelid as a unilateral blepharospasm, which is the most common form of partial facial spasm. Later they involve the muscles of the cheek (the zygomatic, the levator anguli oris, etc.) and subsequently those of the forehead and the chin. In fully developed cases earlier authors as well as I have observed contractions in the muscles that move the lobes of the ear, also in the platysma myoides, and in one case a patient complained at the height of the attack of a grinding sensation in the ear which I attributed to a contraction of the stapedius muscle. But the spasm never extends beyond the region of the facial nerve, and is always limited to the side first attacked.

The duration of the individual attacks varies from a very brief time to 15 minutes or longer. When prolonged, the clonic contractions at the acme of the attack pass into tonic spasms, the face during the interval becoming perfectly passive. Nevertheless, in a few instances in which the attacks were frequent I have observed a slight increase of the nasolabial fold and a narrowing of the palpebral fissure. The contractions are very irregular. I have already stated that they are frequently produced by chewing, swallowing, and speaking. After it has begun, *true tic convulsif* (idiopathic facial spasm) *cannot be controlled by any exercise of the will.*

Since the spasm never attacks the muscles of the tongue, the neck, or the shoulders, as do cortical spasms, it is likely that the irritative process which causes idiopathic facial spasm runs its course in the peripheral neuron of the facial tract alone. Some necropsy reports, for instance, those of F. Schultze, have shown that direct irritation of the facial nerve by an aneurysm of the vertebral artery may produce facial spasm. But a reflex irritation in the tract of the trigeminal nerve is usually much more likely, because, with the exception of the previously mentioned rare cases of basal tumors pressing on the facial, in which a rapid but transient spasm introduces a succeeding

paralysis, such an irritation has never been observed after idiopathic facial spasm which persisted for years. In a case of endocranial tumor of the anterior cranial fossa which had injured the first branch of the trigeminal nerve, Oppenheim saw facial spasm on the same side.

Only very rarely do peripheral irritations (conjunctivitis, irritation of a tooth, affection of the ear, etc.) prove clinically to be of etiologic importance. The pressure points in the face and in the transverse processes of the cervical vertebræ which v. Graefe, R. Remak, and others described, and from which attacks may be produced or aborted, I have scarcely ever found to be active, in spite of much labor expended upon repeated tests. It has already been stated that nervous headache and attacks of migraine occasionally precede facial spasm; but hemicrania does not always occur upon the same side as the facial spasm.

What has been previously stated of the treatment of localized spasm particularly applies to true facial spasm. Actual cure only exceptionally occurs; more often there is merely improvement; exacerbations and relapses are common.

(4) *Post-paralytic spasm of the muscles of the face* is not infrequently an accompanying phenomenon of severe or moderately severe peripheral facial paralysis which has not yet quite yielded. While the face is at rest there is usually a slight *contracture,* probably due to interstitial myositis of the formerly paralyzed half of the face, which reveals itself by a narrowing of the palpebral fissure and the marked prominence of the nasolabial fold. The voluntary and involuntary movements, which to some extent reappear, cannot be evoked in the various portions of the face without implicating the other muscles, but on closing the eye the nasolabial fold is increased by the tonic and conjoint action of the zygomatic muscles, etc., and in puckering the mouth (as in whistling) the eye is more nearly closed. In such cases we not infrequently observe more or less conspicuous and apparently spontaneous contractions, particularly of the muscles of the cheek, occasionally of the chin, whose synchronism with the physiologic wink of the eyelid I have several times demonstrated (1881 and 1898) and have observed to be invariable. *When the patient voluntarily suppresses the closing of the lid these contractions cease.* I have therefore regarded these spontaneous contractions as clonic, constrained movements due to the physiologic closure of the eyelid. The irritation which Hitzig and others assume to be conveyed to the nucleus of the facial nerve seems reasonable, and in the cases observed by R. Remak, Hitzig, Bernhardt, and Müller is undoubtedly true of the post-paralytic facial spasm which also implicated the non-paralyzed half of the face, sometimes to a marked extent.

Treatment for these conditions is of but little avail. The contracture of the cheek is best combated by stretching and galvanization with the anode. I also advise that the patient practise the movement of individual muscles before a mirror, and that he suppress conjoined movements as far as possible. Emotional movements of the face must be refrained from in order to prevent distortions.

(5) *Tic* (impulsif) and

(6) *Myoclonia* of the facial muscles can be differentiated only with great difficulty, and some authors, for instance, Meige and Feindel, group them

together. If we consider as tic those combined movements which resemble voluntary ones, and as myoclonia the clonic, lightning-like, individual contractions of muscles, we find by experience that both forms of contraction actually occur in the face side by side.

Of 43 cases in which the face was solely or chiefly implicated, 26 cases occurred in early life (4 to 20 years of age) and 30 cases were females. The high ratio of 69 per cent. of females probably depends upon hysteria which must often be taken into account. A woman whose case I reported came to me in 1894 on account of hysterical stuttering, but she also presented tic in the muscles of the face, for, owing to almost rhythmical contractions of the occipitofrontalis, transverse folds formed in the forehead, the ears were occasionally moved, sometimes also the muscles which raise the upper lip, and the platysma myoides contracted. Certainly these cases are most common in neuropathically predisposed individuals. Fright and trauma (especially of the head) were in a few cases the accidental cause, and tic of the face, especially nictitatio and blepharospasmus duplex, has been observed by myself and others as an accompanying phenomenon of traumatic neurosis. Whether onanism, which is frequently observed in children, may be regarded as the etiologic factor, or whether this is simultaneously due to a nervous predisposition, cannot at present be decided.

It is characteristic of tic as well as of myoclonia of the muscles of the face that the contractions are not limited to one side but are usually bilateral, are not always strictly confined to the region of the facial nerve but occasionally implicate the external muscles of the eye, the tongue, and the muscles of the vocal cord, as may be proven by directing the patient to make slight sounds. Sometimes they also implicate the muscles of the neck and the nape of the neck. In cases of apparent myoclonia of the face, after the patient removes his clothes we now and then note lightning-like contractions of the trunk and extremities which had previously been overlooked (polyclonia, chorea electrica).

Unlike idiopathic facial spasm the patient may occasionally suppress these contractions at will, as is shown by the fact that during the examination there are no contractions, but these are frequent when the patient believes himself to be unobserved. A relatively common localization of facial tic, sometimes due to photophobia, is a frequent closure of the eye (nictitatio) or a conspicuous blepharospasmus duplex, occasionally an involuntary lateral rotation of the eyes. In addition to tic of the eyelids, Meige and Feindel differentiate mimical tic, tic of the nose (snuffling tic), tic of the lips (sucking tic), and tic of the chin. But they quite correctly lay stress upon the frequent combination of these tics with each other, with clonic contractions of the fronto-occipitalis, the muscles of the chin, and the platysma myoides. As I observed in the case of a girl aged 16, contractions of the fronto-occipitalis may be so severe as to shake the hat. Clonic and tonic spasms limited to the platysma myoides or to the muscles of the chin, I have several times observed to be bilateral, once only unilateral, and in rhythmical sequence, and have also seen these as individual attacks in hysterical women. In a case which was under my care for ten years, these paroxysmal contractions of the platysma were a partial phenomenon of tabes in conjunction with hysteria. The rhythmical platysmic contractions occasionally mark a transi-

tion into the previously mentioned chorea rhythmica (hysterica), and they also occur with rhythmic bilateral contractions of the muscles of the shoulder and arm.

The course of myoclonia and of tic is irregular, and varies inasmuch as when the contractions cease in one area they may appear in another or may become general. Nevertheless the symptoms may sometimes disappear, but relapses are common.

In *treatment*, especially of the young, the removal of deleterious or psychical sources of irritation is indicated, possibly also a stay in a sanatorium. Soothing measures, for example, warm baths, are generally advisable. Cold applications are contraindicated. In some cases there have been apparent results from the adminstration of moderate doses of arsenic and simultaneous and systematic galvanization of the nape of the neck by the anode (3 or 4 milliampères); in 1881 I found this treatment beneficial in a few cases which were diagnosticated as Henoch's chorea electrica, but what its therapeutic value is I cannot state. Lately we have been inclined to expect more from psychotherapy, but as this necessitates both the good will and cooperation of the patient, like the inhibitive therapy previously described (psychomotor education), to carry it out in children demands the utmost patience on the part of the physician.

(7) *Hysterical contracture* of the facial muscles may be introduced by clonic contractions, but may appear without this, and may persist unilaterally or bilaterally in a more or less unchanged condition. The diagnosis of hysteria should be made only after every other form of spasm has been excluded, especially that due to organic cerebral disease (for example, of the pons) as well as post-paralytic and tetanic contracture. Hysterical contracture of the muscles which close the eye has been several times described. This also may be unilateral and, according to Parinaud and Charcot, Nonne and Beselin, Wilbrand and Sänger, may resemble ptosis (*ptosis spastica pseudòparalytica*)—a point which I cannot here discuss. A unilateral and usually slight hysterical contracture of the lower portion of the face is a partial phenomenon of typical hysterical *hemispasmus glossolabialis*, to which I shall refer when describing spasm of the tongue. In this condition, as Charcot, Brissaud and Marie have shown, and as I stated in 1892, unilateral contracture resembles paresis of the lower branches of the facial nerve which supply the mouth on account of the deepening of the nasolabial fold and the contractions of the lips. A light passed in front of the apparently paretic side of the mouth is not blown out, as in true paresis, upon this side, but after it has passed the median line and is on the side of the contracture.

An *isolated, unilateral, hysterical contracture* of the lower half of the face without contracture of the tongue has so rarely been described (except by Ollivier, Bourneville, Binswanger) that I will mention a typical case of my own.

A woman, aged 21, who for more than 6 months had been subject to hysterical crying, had attacks in which, after several contractions of the left cheek, the mouth and the left nasolabial fold were markedly drawn to the left, and remained so for one to three days. Upon admission to the hospital she presented a peculiar appearance. While the upper portion of the face down to the middle of the nose was quite symmetrical, the left ala nasi was drawn to the left, and the left angle of the mouth to the left and downward. the left nasolabial fold and the fold of the chin being

strongly marked. In speaking and on opening the mouth there was no change. The tongue was protruded in the median line, and was freely moved. There was no difference in the faradic contractility of either branch of the facial nerve. Faradization of the muscles of the right side brought the mouth to its correct position, but it subsequently returned to the previous one. Nevertheless, the deviation of the face was corrected after faradization had been repeated several times in the course of a few days.

(8) We can regard as *occupation spasms* only those exceedingly rare cases of spasm of the facial muscles which (analogous to true writer's cramp) are produced by definite movements of the face, and solely by these. To this group belongs a case reported by Zenner as *auctioneer's spasm,* in which, at first upon repetition of a number, subsequently in ordinary speech, spastic contractions appeared in different muscles of the face, particularly the orbicularis oris. In the case of a *trumpeter* observed by Oppenheim, who felt a contraction in the orbicularis oris as soon as he placed his instrument to his mouth, true spasm could not be determined. Bernhardt mentions a case of *reading spasm* which Duchenne described as occurring in a young man: As soon as he began to read, a contracture of the frontalis elevated the eyebrows, and the eyelids were spasmodically closed. On the other hand, Toby Cohn reported as *watchmaker's tic* a continuous, typical, clonic-tonic spasm of the entire left half of the face supplied by the facial nerve, this occurring in a watchmaker whose work had for years required him to press a magnifying glass to his eye, and who had developed a spasm of the muscles supplying the left eye, a true blepharospasm.

Before concluding the subject of localized facial spasm I must mention that since Hitzig's celebrated case of traumatic abscess in the right anterior central convolution, in which the focal symptoms began with clonic spasm in the region of the left facial nerve, particularly about the mouth and nose, but in which also the tongue showed transitory signs of paralysis, many similar cases of *cortical facial spasm* have been reported. Although the attacks may appear during perfect health, in which case they are of short duration, nevertheless the signs may point to a severe cerebral affection (tumor, etc.). A correct diagnosis is of the utmost importance. For example, a tuberculous man, 60 years of age, consulted me because in the preceding four weeks he had had five attacks, of at most two minutes' duration, in which the mouth was retracted to the left, the lower jaw became rigid and somewhat retracted, the tongue was immovable so that he could not speak, yet consciousness was not lost. The objective examination of the nerves gave absolutely negative results; not even paresis of the left branch of the facial supplying the mouth could be determined, and this is of pathognomonic significance. For a time after the administration of a mixture of iodin and bromin the attacks lessened in severity; six months later the house physician informed me of the lethal outcome of the case. The attacks had been intensified, and left-sided hemiplegia had finally occurred.

In the initial stage of these cases the diagnosis is frequently difficult, for, as the following case shows, *hysteria* may simulate the picture of cortical spasm.

A girl, 13 years old, the child of nervous parents, was brought to me because for about eight months preceding and at irregular intervals she had had numerous attacks, lasting about five minutes and accompanied by a feeling of anxiety, during

which the muscles of the left cheek and of the eye moved to and fro. The tongue was drawn to the left side of the mouth so that she could not speak, she saw a play of colors, and her left hand felt numb. No contractions were observed in the head or arm, and consciousness was retained. The objective findings were entirely negative. Under psychical and faradic treatment the attacks ceased, and four months later there had been no recurrence of the attacks.

Group II.—Except for fibrillary contractions, localized *spasm of the tongue* is very rare, while, on the contrary, the tongue is frequently implicated in general spasmodic neuroses, chorea, epilepsy, and tic.

(1) The continuous, wavy, fibrillary contractions which are observed in the atrophic tongue in amyotrophic bulbar paralysis, and bilaterally and unilaterally in hemiatrophy of the tongue due to traumatic or neuritic hypoglossal paralysis, may be designated as *myokymia of the tongue.* These contractions correspond to the so-called paralytic oscillations which have been studied by physiologists. They must not be confounded with the tremor of the quiet or protruded tongue which is frequently observed in progressive paralysis, in alcoholism, in melancholia, etc.

(2) A few cases have been described of *reflex spasm of the tongue* due to painful affections of the mouth, as from caries of the teeth (Mitchell), after extraction of a tooth (Féré), or in trigeminal neuralgia which implicates the lingual nerve (Romberg). Reflex irritation may also originate in the nasal cavity (Wendt) or in even more distant nerve areas. Gallerani and Pacinotti described a case of spasm of the tongue, of the lips, and of the pharynx, which was manifest in the speech chiefly as aphthongia, and gradually disappeared after the excision of a small piece of porcelain from the occipital nerve. It appears that reflex spasm of the tongue is even less strictly localized than the idiopathic form.

(3) *Idiopathic spasm of the tongue,* according to the observations of O. Berger, Dochmann, E. Remak, Erlenmeyer, Bernhardt, Hirt, Personali, Lange and Sänger, appears without obvious cause, occasionally in nocturnal attacks, usually with bilateral, and sometimes clonic, contractions which are more marked upon one side, and which may be preceded by a sensory aura such as paresthesia of the tongue. In 1883, I presented before the Berlin Medical Society the case of a man, aged 33, whose tongue, resting upon the floor of the mouth, by a decrease and flattening of its surface was moved 45 to 50 times in the minute, while corresponding rhythmical contractions of the muscles of the tongue could be externally felt between the lower jaw and the hyoid bone. The tip of the tongue never struck against the teeth. The contractions became stronger when the tongue was depressed with an instrument; then it became markedly arched, especially toward the left side. The tongue was protruded rhythmically, and even forcibly. The contractions were increased, and invariably produced, by mastication. In this case the tongue was frequently caught between the teeth or pressed against the palate by a tonic spasm which prevented deglutition. In the more violent attacks produced by mastication, the spasm subsequently implicated the lower half of the left side of the face, and caused severe tonic and clonic contractions of the muscles of the cheek and the area surrounding the mouth, while the upper branches of the facial nerve were not involved. A paresis of the left branch of the facial supplying the mouth, which was previously demonstrated,

was more profound after these major attacks. This affection, which had developed within four weeks, declined markedly within eight days under the administration of potassium iodid and potassium bromid, and the daily anodal galvanization of the inframaxillary region; it has never reappeared. There was no paresis of the facial nerve when I examined the man two years later. In my case, and also in Erlenmeyer's, the spasm implicated the region of the right facial nerve. In Hirt's case spasm of the muscles of mastication always preceded spasm of the tongue. While in my case the attacks were chiefly produced by the act of mastication (Romberg's *masticatory lingual spasm*), in other instances nothing of this kind was noted, and in Lange's case the tonic spasm of the tongue ceased when the patient began to chew, so that during the day he constantly chewed something in order to keep his tongue in his mouth. Closer analysis of the reported cases confirms the view that the pathologic picture of idiopathic spasm of the tongue is not so uniformly and strictly confined to the region of the hypoglossal nerve as is, for example, idiopathic facial spasm; therefore it seems unlikely that the pathogenesis of idiopathic spasms of the tongue is invariably the same. If they are in any sense true spasms it is doubtful whether they are due to affection of the nucleus of the hypoglossal or of the cortical region of the tongue. In either case we can understand the transference of the effect to the tract of the lower facial and the motor trigeminal nerves. As paresis of the facial nerve could be demonstrated in my case, I assumed a partial cortical epilepsy.

Whether the treatment I employed brought about a cure cannot be stated with certainty, but, at all events, in other cases (for example, that of Berger) galvanic treatment has been effective. Personali saw good results from antipyrin in increasing doses of from 0.8 to 4.6 grams per day. Sänger found iron and arsenic to be beneficial.

(4) According to Meige and Feindel, *lingual tic* (tic impulsif of the tongue) rarely occurs alone. They characterize as *licking tic* the tic movements in which the tongue moistens too profusely the free border of the lips; as *chewer's tic* those in which the tongue rolls about between the teeth and the inner surface of the cheeks. On account of their psychogenous origin, the cases described by Erb, Seppilli, and Oppenheim probably belong to the tics of the tongue, cases of spasm of the tongue accompanied by hallucinations and other psychical anomalies.

(5) *Hysterical spasm of the tongue* is noted as one of the phenomena of *hemispasmus glosso-labialis,* but also on protuding the tongue and without a simultaneous facial contracture as a unilateral tonic spasm in this affection. Not rarely it appears with hemianesthesia, usually of the other side. In the typical case of hemispasm of the right side, which I analyzed in 1892, the tongue lay straight upon the floor of the mouth or, perhaps, deviated slightly to the right. With irregular rotary movements it was pushed strongly to the left, then in a convex arch to the right side, so that it was held almost entirely in the right half of the mouth, and deviated more than it ever does in hemiatrophy or in cerebral hemiglossoplegia. In this deviating position the patient was able to move the tongue up and down, but to the left only to the median line. As soon as I attempted to move the organ to the left the muscles on the right side of the tongue offered strong resistance. When this was overcome, and the tongue was brought to the left angle of the mouth,

it did not deviate as soon as the pressure was relaxed, but only after considerable time and as if by voluntary movement. Even when the tongue was returned to its normal position by faradic stimulation of its left half, on interruption of the current it did not deviate until after some time had elapsed.

Hysterical contractions of the tongue may accompany other contractions of the face, of the neck, etc. For instance, I saw severe attacks in a case of hysteria virilis occurring in a man, aged 28, who presented right-sided hemiparesis and hemianesthesia, and in whom persistent, almost rhythmical, and painful contractions of the right-sided muscles of the cheek and forehead distorted the face. His tongue was protruded paroxysmally, the contractions of the face increasing, and extending to the right platysma myoides and to the right arm.

(6) *Articulatory lingual spasm* which only appears on an attempt to speak, and at first prevents speech, which Fleury (1865) designated *aphthongia,* was observed by Fleury, Vallin, Mossdorf, Ganghofner, and Steinert, usually in excitable young people from mental emotion (fright, sorrow, anxiety). Upon attempting to speak there is a *tonic intention spasm* of the muscles of the tongue which usually forces the tip of the tongue against the teeth and its root against the palate. Even this form of lingual spasm is rarely limited to the region of the hypoglossal nerve, but usually implicates the lower muscles of the face, the platysma myoides, the sternocleidomastoid, the abdominal muscles, and, by causing the patient to make a fist (Steinert) or by contraction of a lower extremity (Ganghofner), even the muscles of the extremities. These authors have warned us not to confound this condition with severe stuttering, in which extensive constrained movements may also occur. In the treatment of these spasms, it is advisable to resort to psychical influences, and, judging by the favorable results of Mossdorf, to anodal galvanization of the upper part of the nape of the neck.

(7) *Other occupation spasms of the tongue* have been only rarely reported, by v. Strümpell in a clarionet player, and by Turner in a cornetist. When he played with the band, the latter was unable to move his tongue for the production of certain notes, while at home he could produce them at will.

Group III.—In passing we may mention localized spasms of *the palate* which usually run their course as rhythmical contractions. They have been noted as the consequence of organic diseases, bilaterally in tumors of the cerebellum (Oppenheim), and unilaterally in aneurysm of the vertebral artery (Oppenheim and Siemerling). They are more common when purely functional (hysterical) in origin (Williams, Scheich, Goldflam, Meyerson, Peyser, Avellis, Bernhardt and others). The various forms of spasm of the glottis need no special mention.

Group IV.—Localized *spasm of the muscles of mastication* mainly affects the muscles innervated by one nerve, the motor branch of the fifth; these muscles are the temporal, masseter, pterygoid, mylohyoid and digastric. Authors usually differentiate only clonic and tonic (trismus) forms of this spasm. Meige and Feindel, however, described a tic of the lower jaw (biting and chewing tic) and a psychogenous trismus.

(1) Chattering of the teeth from exposure to cold and from chill in fever is due to clonic rhythmic *contractions of the muscles of mastication.*

Similar clonic contractions limited to the muscles of mastication now and then occur in hysteria. Clonic spasms of the muscles of mastication as a partial phenomenon of general spasms are more common. It is well known that these cause epileptics to bite the tongue, and in some cases of paralysis agitans they produce rhythmical horizontal contractions of the lower jaw (pterygoid muscles).

(2) A *tonic spasm of the muscles of mastication* (*trismus*) can be assumed only after excluding the fact that the teeth are locked because of an ankylosis or pseudo-ankylosis of the joints of the jaw. The palpable demonstration of tonic tension of the masseter and temporal muscles is decisive. This may readily be due to some painful irritation affecting the third branch of the fifth nerve as well as by painful affections of the teeth, and is especially prone to occur in children from the cutting of teeth. We must always bear in mind, however, that trismus may be the first symptom of tetanus, and a simultaneous paralysis of the facial nerve is an indication of head tetanus. The preceding injury to the face may have been so slight as to have been forgotten. For example, in 1883 I saw a case which was later described by P. Güterbock, a fatal case in a man aged 34. As the lockjaw was apparently preceded by bilateral pain in the region of the jaw, I erroneously assumed a reflex origin in the oral cavity, and sent the patient to a surgeon for examination under anesthesia. In a case of head tetanus in a boy aged 9, diagnosticated by me in 1901 and described by Jolly, a diagnosis of meningitis was made at his home. As a matter of fact, at the onset of the affection a diagnosis may be extremely difficult, especially if, as P. Richer, v. Eiselsberg, Jolly, and Binswanger have observed, there is also *hysterical trismus* which resembles beginning tetanus. Such cases may be identified with the *psychogenous trismus* described by French authors, which may also be a symptom of marked psychosis. The diagnosis is much less obscure when trismus occurs as a symptom of organic cerebral disease (meningitis, acute bulbar paralysis, tumor of the pons or cortical disease), because the accompanying symptoms of irritation as well as the general signs point to the true nature of the disease. In conclusion, the rigidity of the muscles of the jaw in mastication, which is peculiar to Thomsen's disease (myotonia), may also be mentioned.

(3) According to Meige and Feindel, *tic impulsif of the muscles of mastication* (*biting and chewing tic*) occurs in predisposed individuals in consequence of an erosion of the lips or of a slight periostitis of the jaw, and is shown by the fact that mastication produces sensation in the affected area. On the other hand, constrained tic movements in the lower jaw may become so severe that the lips are bitten (cheilophagia). Onychophagy which, as is well known, is a sign of degeneration, may also be regarded as biting tic.

Group V.—Next to spasm of the muscles of the face, I have found localized *spasms of the muscles of the neck and nape of the neck* to be the most common form (in about 50 cases they formed 20 per cent.). I do not group among these painful and chronic torticollis (caput obstipum or torticollis rheumaticus) in which there is extreme intolerance even of passive motion of the stiff, cervical vertebral column, this depending chiefly upon inflammatory, and partly upon reflex, contraction of the sternocleidomastoids, especially of one, in consequence of cold to the nape of the neck or inflammation of the

cervical vertebræ. This will be described, as well as congenital torticollis which is usually due to a shortening of the muscles. If we confine ourselves wholly to the clonic-tonic spasms of the muscles of the neck and nape, a sharp differentiation of the various forms becomes very difficult, much more so than in the forms of spasm previously described.

(1) *Nodding, salaam,* or *greeting* spasms (spasmus nutans) occur in early childhood, usually in the period of dentition, and are characterized by a pagoda-like twitching or nodding of the head which occurs several times in a minute and depends upon clonic, and usually rhythmic, contractions of the deep muscles of the neck (splenius, biventer, recti et obliqui capitis), sometimes also of both sternocleidomastoids. These contractions are not infrequently associated with nystagmus, which is most apt to occur when the head is firmly held or, as I have noticed, when the child while looking at an object periodically interrupts the movement of the head. This form of spasm, as I recently had an opportunity of observing in the case of twins, lessens after some time, but it may also be the first symptom of a grave cerebral affection.

(2) In predisposed children six years of age and upwards *myoclonia* and *tic* are sometimes, although not exclusively, localized bilaterally in the *muscles* of the *neck* and the *nape.* I have records of 13 such cases occurring chiefly in females (10). In irregular sequence, here and there, brief individual contractions of the superficial muscles of the neck were noted, especially of the platysma myoides (myoclonia). Or the head was rapidly thrown backward or shaken (tic) by brief and usually symmetrical, combined, spasmodic contractions of the muscles of the nape. In a case of Henoch's chorea electrica which I demonstrated in 1881, that of a girl aged 10, the head was forced backward 60 times in a minute by rhythmical contractions of the muscles of the neck and palpable contractions of the splenii capitis, while spasms of the platysma were also visible; as a rule nictitation and tic-like contractions of the muscles of the face reveal the nature of the affection. This rarely persists to advanced age. But a spinster, aged 52, had from earliest childhood, and particularly when walking, been subject to brief posterior movements of the head and, simultaneously, bilateral and visible contractions of the platysma. Rhythmical spasm of the platysma in hysteria has already been mentioned; this cutaneous muscle is supplied by the facial nerve.

(3) Clonic and tonic *rotary spasms of the neck* (*torticollis spasticus*) I have observed in 24 cases ranging in age from 13 to 66 years (11 males and 13 females). In only two cases did it appear before the twentieth year; in the overwhelming majority it was after the thirtieth. As a rule the occiput (region of the ear) on the same side as the spasm is forcibly and paroxysmally drawn downward, the face is turned to the opposite side, and the chin is elevated, while contractions of the sternocleidomastoid are evident both to inspection and palpation. As the trapezius of the same side frequently and simultaneously becomes tense and draws the head backward, raises the shoulder, and draws the shoulder-blade toward the vertebral column, we were formerly inclined (according to the traditional teaching that localized spasms originate from individual peripheral nerves) to explain rotary spasm simply as a *spasm of the spinal accessory.* It did not, however, escape the notice of vigilant observers that the muscles of the nape of the neck

which are supplied by the upper cervical nerves, such as the longissimus capitis, the splenius capitis, and the transversus spinalis of the same side, are implicated in the spasm; that, especially with persistent intermittent spasms, contractions of the antagonistic muscles at the other side of the neck are also alternately noted. These combined spasms of the muscles of the neck are explained by Seeligmüller and others as arising because, in consequence of a spasm of the spinal accessory, the antagonists of the corresponding muscles of the other side make a constant effort to keep the head in an erect position, and are thus themselves contorted by spasms. From my own observations I have come to the conclusion that a pure spasm of the spinal accessory is very rare, although I am willing to admit with Bernhardt that it does occur. In fact, close study of paralysis of the trapezius has shown that the cervical nerves are certainly implicated in the innervation of this muscle, and that when the trapezius is more decidedly involved in the spasm it is a mistake to consider merely a strict localization in the region of the spinal accessory. This is of practical importance, inasmuch as it explains the numerous failures after division of the spinal accessory in cases of so-called spinal accessory spasm.

But in most cases not only the limitation of rotary spasm of the neck to the region of the spinal accessory but also the assumption of a true spasm has been doubted. Brissaud and his pupils, Bompaire, Meige and Feindel, regard it as a *psychogenous tic* (*torticollis mental*). Neuropathically predisposed or psychically influenced persons, from an insignificant irritation in the region of the nape of the neck or from their occupations, are said to contract a habit of turning the head which subsequently develops into a constrained, psychogenous tic (impulsif). As proof of the tic nature of the spasm, it is maintained that when the attention of the patient is distracted the spasm sometimes ceases, and that they can sometimes suppress the contractions, as I proved in several cases, by making slight pressure with the finger against the chin, the contractions being previously so violent that the investigator was unable to check them by any means. Nevertheless Meige and Feindel hold that the differential diagnosis between spasm and tic of the neck is not easy, and they base their diagnosis of tic essentially upon the demonstration of psychical anomalies which are peculiar to all tic patients (peculiarity in or change of disposition, childish mental state).

From the investigation of my own clinical cases I believe it to be absolutely impossible to differentiate cases of true spasm and psychogenous tic, and I certainly believe that there are many transitional stages between them.

As rotary spasm of the neck is a most conspicuous and distressing affection which not only renders the patient unable to work but is apt to occur during walking, etc., and therefore interferes with all social life, the moody, disagreeable nature of these patients is sufficiently explained. But Oppenheim and others, as well as I, have observed marked psychical disturbances (hallucinations, etc.). A man, aged 25, in whom persistent torticollis spasticus had been observed, was arrested a few years later for exhibitionism. Scotoma is frequently noted. A woman, aged 47, who was under my observation for seven years, had left-sided spasm of the spinal accessory which improved and left but the merest trace; but within a year she was disturbed by continuous photopsia though the eyes were in a normal condition. On

several occasions simultaneously a bilateral blepharospasm revealed the tic nature of the affection. In 1881 I examined a woman, aged 52, who had peculiar rotary movements of the eyes. This case, which had been successfully treated by my father in 1862, was of etiologic interest because the patient had for 13 years been employed at reeling wool which compelled her constantly to move her head from one side to the other. This occupation etiology, lately urged by Walton, appeared also in six others of my cases (in a balancer, a piano-teacher, two locksmiths, a velvet smoother, and in a statistician whose work was the comparison of rows of figures). In all of these cases spasm was introduced by disagreeable sensations in the nape of the neck (usually, but by no means always, pressure points could be demonstrated in the transverse processes of the cervical vertebræ) and this suggested a reflex genesis. Nevertheless, nothing conclusive can be stated in regard to the starting-point and the pathogenesis of rotary spasm of the neck. Oppenheim believes it likely that this spasm originates in the kinesthetic centers for the muscles of the neck in the cortex of the brain.

A markedly tenacious *course* and the inefficacy of all antispasmodic therapy is almost an invariable rule in this form of spasm. Nevertheless, under rest and galvanic treatment of the nape of the neck (especially of the transverse processes), persisted in for a year and longer, in four cases I saw such an improvement that there was but a slightly oblique position of the head. Among these was one case that had been under observation for eight and a half years. But the permanence of the recovery is always doubtful, and here psychical influences play a great rôle. A woman, aged 28, was attacked at the age of 16 with right-sided spasm of the spinal accessory, but between the ages of 21 and 25 and while she was engaged to be married she was entirely exempt. After the breaking of her engagement, a relapse occurred and the spasm persisted for three years. A patient, aged 66 (according to a history taken by my father who made a diagnosis of alternating spasm of the spinal accessory), who for thirty years had followed his occupation as a school-teacher, suffered a severe relapse after a profound mental shock.

Psychomotor education, which for this form of spasm is especially lauded by Meige and Feindel, may be employed alone or as an auxiliary to other forms of treatment, particularly galvanism. Hasebrock lately brought about improvement in the position of the head by the use of elastic rubber bands fastened to strips of plaster, which aided the action of the healthy antagonists.

According to de Quervain's compilation of the results of Kocher's operations, one method which consisted in the severing of all the muscles implicated in the spasm should be considered in especially severe cases. Kocher attaches great importance to subsequent gymnastic exercises. An engineer, aged 40, suffering from severe spasm which was at first left-sided and then bilateral, and who at that time was unsuccessfully treated by me, was operated upon by Kocher eleven months later, and six years afterward I had an opportunity of confirming the absolute success of the operation. The functional disturbance was slight, and the deep transverse cicatrix on the nape of the neck did not greatly disfigure him.

(4) Aside from the previously mentioned painful and partly reflex *caput obstipum*, bilateral *contracture of the muscles of the nape of the neck* which

appears as stiffness of the nape of the neck or is markedly developed as opisthotonos, is a symptom of tetanus or organic disease of the posterior cranial fossa (tumor, meningitis, etc.). In 1890 I demonstrated in a tuberculous patient slight contracture of the muscles of the neck and more decided contracture of the muscles of the shoulder and upper arm with excessively increased tendon reflexes in this region; the autopsy, conducted by Goldscheider, revealed a myelomeningitis cervicalis anterior which appeared to have irritated the anterior cervical roots.

In paralysis of the serratus complicated with partial paralysis of the trapezius, *unilateral contracture* of the single muscle of the nape of the neck which passes to the shoulder-blade (levator anguli scapulæ) and of the rhomboidei causes the so-called rocking position of the scapula. In some cases reported by A. Eulenburg, Nové-Josserand, Bernhardt, P. Manasse, and others, an acquired high position of the scapula was attributed to permanent and idiopathic tonic contraction of the levator anguli scapulæ and of the rhomboideus. In a case reported in 1903 by P. Manasse, in which all other methods of treatment were for a long time unsuccessful, repeated division of the contracted muscles led to recovery. It is noteworthy that during the operation, and while the patient was under anesthesia, clonic contractions of the muscles occurred.

Group VI.—Of all the localized spasms of the *upper extremity* the most numerous are *occupation spasms*, particularly *writer's cramp.* As *occupation neuroses* (of which *occupation spasm* is a common form) will be described in a separate chapter, I shall not discuss them here; I reported my own experiences in an article in the "Real-Encyklopädie" in 1894. I shall merely remark that while, as a rule, writer's cramp is noted exclusively during the act of writing, yet when the affection is severe it may last longer than this, and may exceptionally present itself also as *intention spasm* during other occupations. For instance, in a book-keeper whose hand showed spasmodic dorsal flexion, and deviated toward the ulnar side on every attempt to write, the same spasm was produced whenever he held small objects in his hand (for example, playing cards), and this partial and tonic *radial spasm* was frequently visible when he held nothing. L. Laquer reported a substantive tonic radial spasm in connection with writer's spasm. Another patient, under my observation with writing spasm, was seized with a *flexor spasm* of the right wrist-joint and fingers on every attempt at writing; this contracted the hand into a perfect ball shape. The spasm, like that of Gowers's case which chiefly affected the region of the median nerve and amounted to a flexor spasm, also occurred when, on making an effort to grasp anything, the thumb was placed in apposition to the fingers. When the spasm was more markedly developed, not only were the fingers spasmodically contracted but the hand also showed spasmodic volar flexion, and the elbow-joint was flexed. The view of a cerebral origin for such forms of spasm is confirmed by the history in the following case of symmetric irradiation of occupation spasm. An official, who had suffered for 15 years with severe writer's spasm and painful stiffening of the joints of the right hand, and who for 4 years had written with his left hand, complained that for 2 years of this latter period the right thumb by a constrained movement had been forced under the other fingers. He could only resist this pain-

ful spasm by grasping some object with the right hand while writing with the left.

Peripheral irritative factors may produce, presumably by way of the reflexes, secondary occupation spasms of the upper extremity similar to my previously mentioned case of milker's spasm in a milk-maid, who, after prolonged milking, at first suffered from nocturnal painful spasms of the fingers, but in the second week after abandoning her occupation and while under observation had painful drawing sensations lasting for 15 minutes; these began in the fingers and passed to the shoulder when flexor spasms of the first three fingers (*spasms of the median nerve*) occurred, and also actual pain. In this case there was bilateral neuritis of the median nerve with a corresponding disturbance of sensation in this region, also in the area of the right superficial radial nerve, as shown by the reaction of degeneration. As other cases of occupation neuritis are not usually accompanied by such local spasms, perhaps these depend upon a special "spasmophilic" predisposition based on hysteria. This may also explain why localized attacks of spasm, rhythmical, clonic, tonic contractions, or tonic spasms, are so much more rarely observed after over-exertion, no matter of what nature, than so-called occupation paralyses. After exhaustive gymnastics Hochhaus saw in the case of a boy, aged 9, contractions of the right triceps and supinator longus which occurred 40 to 50 times per minute. I saw in a girl, aged 10, rhythmical contractions of the right biceps and supinator longus after climbing. In a workman who had frequently lifted heavy loads of iron Bernhardt (1901) observed clonic contractions in the left pronator teres which recurred about 150 times a minute. Brissaud (1902) saw in cork-cutters a permanent spastic retraction of the last two fingers of the left hand, such workmen being compelled to keep these fingers in one position.

After trauma, but only with a special predisposition (with or without external wounds), localized (hystero-traumatic?) spasms of the arm of varying extent may be observed. The painful muscular contractions which occasionally are felt in stumps after amputation are familiar to us from the descriptions of Romberg, S. Weir Mitchell, and others. Injuries without an external wound or cicatrix are often sufficient to produce these. A patient, aged 18, who sustained a fall upon the right elbow-joint, suffered for two years from contractions in the right thumb as soon as he placed it in a position of medium apposition. A girl, aged 9, a few weeks after a distortion of the right wrist was asked to write; immediately afterward her right hand contracted into a fist and could not be opened for twelve hours. A boy, aged 9, three days after being struck by his brother a blow on the right arm with a stick, had permanent rhythmical contractions of this arm which persisted for two years, and then suddenly ceased, leaving a tremor which appeared only when he tried to write. A girl, aged 18, had daily four or five attacks of clonic contractions in the right hand which, closed to a fist, assumed a position of dorsal flexion, the elbow-joint being flexed and the upper arm spasmodically adducted; neither the head nor the facial nerve was implicated in this spasm. It was noticed that the spasm could be produced at will by pressure upon a painful spot on the back of the forearm. There was gradual improvement after anodal galvanization of this area. This was probably hysterical brachial hemispasm, and was attributed

to the pressure of an exostosis. In children especially, even without traumatic etiology, probably owing to hysteria and particularly after emotional disturbance (fright and the like), various forms of spasm of the upper extremity occur which, because of their apparently psychogenous development and the variations in their mode of appearance, are difficult to describe systematically; they frequently undergo a transition into hysterical contracture, chorea rhythmica, chorea hysterica, and tic impulsif. A girl, aged 11, was under treatment two years for a grapho-tremor; there was a contracture of the left arm accompanied by pain in the left elbow-joint, and this resembled a cerebral spastic hemiplegia running its course simultaneously with almost rhythmical contractions of the adductors of the shoulder. Under the administration of arsenic and the use of galvanism partial recovery occurred in three months, the tremor persisting. A boy, aged 11, after being frightened by a locomotive, was subject to contractions which spasmodically adducted the arm to the thorax, the hands approximating to each other. Occasionally contractions were noted in the legs when elevated or the patient was forced to stamp if sitting. The head was occasionally rotated to the side. Improvement followed the employment of valerian and the faradic brush. A very hysterical girl, aged 13, had attacks in which the right arm, and occasionally the foot, was drawn up to the head. Pressure in the ovarian region evoked spasmodic contractions of the hands. In adults also rhythmical and extensive spasms of the arm are observed after accidents. To this category belongs a case described by A. Eulenburg in 1897 as fright neurosis, in which hemianesthesia and rhythmical contractions of the left pectoralis major, the clavicular portion of the deltoid, and individual bundles of the serratus were observed in a patient who had suffered from an accident.

The differentiation of such cases of pure functional spasms of the arm from other forms is most important, but is not always easy. I can merely touch upon the affections to be considered, which are as follows:

(1) The forms of true epilepsy which begin with a sensory and motor aura in the upper extremity and are chiefly due to irritation from a cicatrix, usually after injury to the finger; 2. Cortical spasms of the arm which are ushered in with rhythmical clonic contractions, do not extend beyond the arm, do not necessarily produce unconsciousness, and are usually followed by more or less temporary paralysis; 3. Attacks of tetany which, especially in the region of the ulnar nerve, are usually bilateral, but can only be demonstrated as such when the characteristic electro-diagnostic symptoms are evident, because spasm of the ulnar nerve and contractures may be due to other causes. For instance, in a girl aged 11, after an attack of indigestion with vomiting, I saw hysterical intention spasms in the region of the left ulnar nerve upon trying to grasp any object or when extending her arm backward. There was marked analgesia of the arm. The attacks disappeared upon application of the faradic brush. Another girl, aged 11, suffered for three months, at first only on writing, from a transitory and finally permanent contracture in the right ulnar region; this was cured by labile galvanization of the ulnar nerve. In a man, aged 39, who for several years had slept in the open air in Texas, I observed a gradual and permanent but painless spastic contracture in the left ulnar region, especially of the flexor carpi ulnaris, yet there was no disturbance of sensation or of the mechanical or

electric contractility. In the morning the contracture was only slight, but it increased during the day until it resembled ulnar tetany. The benefit from galvanization was here only transitory.

I need not refer to the various forms of trembling which occasionally increase to a shaking spasm, particularly in paralysis agitans, chorea, athetosis, tetanus, and myotonia, and which are most common in the upper extremity. That tic impulsif also affects the upper extremity has been mentioned. Meige and Feindel described a *scratching tic,* also the irresistible impulse to pick the nails of the fingers and toes.

Group VII.—Localized spasm of the transverse striped *muscles of the trunk* is noted in the muscles of the chest, of the diaphragm, and of the abdomen.

Spasm of the muscles of the chest is comparatively rare, is usually bilateral, and is probably due to hysteria. In a lady, aged 24, who was very hysterical, I saw a rhythmical succession of paroxysmal contractions of the pectoralis and the inward rotators of the arm which occurred about 70 times a minute, and did not cease when the arm was raised nor in walking. Such cases have also been called chorea rhythmica hysterica.

Singultus (hiccough), which is often very disagreeable, is due to *clonic spasm of the diaphragm.* Violent contractions of the diaphragm without a simultaneous dilatation of the glottis cause the inspiratory murmur. The affection is relieved by derivatives and blisters, by faradization of the scrobiculus cordis, by the administration of antispasmodics, and by diverting the attention of the patient from his condition. Prolonged clonic spasm of the diaphragm is comparatively rare. A lady, aged 65, who had marked kyphoscoliosis, suffered for three years, apparently after influenza, from pain in the region of the epigastrium, and rhythmical contractions of the entire upper part of the body which were evidently contractions of the diaphragm (48 to 54 in a minute); Litten's diaphragm phenomenon was also observed as well as constrained movements of the erector trunci and right quadrati lumborum. Under anodal galvanization of the nape of the neck and back, this tachypnea was decreased from 36 to 30, and the contractions gradually ceased.

Tonic spasm of the diaphragm, usually of hysterical nature but also occurring in tetanus and tetany, was described by Duchenne and others as a very distressing affection, for the vaulting of the epigastrium and hypochondrium may greatly increase the dyspnea. Powerful rubefacients applied to the lower thoracic region and hot baths are useful in this condition.

Isolated spasms of the *abdominal muscles* seldom occur except in tetanic attacks, epileptic spasms, and in polyclonia, and in all of these hysteria is probably the chief etiologic factor. A man, aged 31, who had been subject to headache, suffered for four weeks from clonic contractions of the abdominal muscles accompanied by pain in the scrobiculus cordis and anorexia: when he was in the recumbent posture these contractions became paroxysmal and arrythmic, and involved both recti and obliqui so markedly that the whole trunk trembled. They could invariably be produced by pressure upon the epigastrium. Neither respiration nor the diaphragm were implicated. A conspicuous, girdle-like, cutaneous anesthesia extending from the scrobiculus cordis to below the navel indicated the hysterical nature of the spasms.

Under faradism of the skin and galvanic treatment the patient recovered in three weeks.

Even unilateral contractions of the abdominal muscles have been described by Duchenne and Bernhardt.

Group VIII.—Localized spasms in the *lower extremities* are more or less rare, but they occur in the same forms as in other regions.

(1) *Myokymia* was described by Kny and Fr. Schultze as most prone to affect the muscles of the calf, and in a few instances painful spasms of the muscles of the calf were periodically observed. In a case of J. Hoffmann's they were localized in the region of the sciatic, and Bernhardt and I observed them in cases of bilateral or unilateral sciatica. It is noteworthy that in all of these instances the tendo Achillis reflex was absent, therefore neuritis probably existed.

That myokymia in the lower extremity may develop chiefly from overexertion, and may gradually disappear after rest, was taught me by the following case:

A chemist, aged 24, had suffered ever since he was 2 years of age from infantile spinal paralysis of the right leg (chiefly of the crural region, but also of the sartorius and of the tibialis and peroneal regions with the exception of the tibialis anticus) and there was decided shortening of the leg; from long standing in the laboratory and excessive use of the leg there was considerable hypertrophy, and simultaneously a persistent, painless, muscular contraction of the left leg. On standing, and when the muscles of the left leg were tense, the entire musculature formed waves, and when in the recumbent posture instantaneous and wavy contractions of varying intensity appeared in the muscles of the calf, the thigh, and also on the extensor surface. The electric contractility of the left side was normal; the galvanic contractions were lightning-like. Galvanism and a course of treatment at Wildbad had a beneficial effect. But even after the patient changed his occupation the contractions continued for a long time, and when I saw him nine years later he stated that they had disappeared only three years previously.

(2) *Clonic contractions* in the region of the crural and obturator nerve are apparently of reflex origin, and are occasionally produced by irritative factors in the cavity of the pelvis. In 1874 I saw a girl who, after a gunshot wound of the pelvis from which the bullet had not been extracted, suffered from persistent clonic contractions in the muscles of the thigh of the same side.

To this category probably belong the contractions noted in diseases of the sexual organs, of the bladder, and of the rectum, as well as in inflammations of the hip-joint. In a case of hernia described by Bernhardt in 1901, three weeks after a bilateral radical-operation clonic contractions occurring about 120 times per minute were noted in the adductor muscles of the right thigh.

A. Eulenburg, Fr. Schultze, and Niebergall have described analogous cases following exertion, long standing, or marching; clonic and tonic spasms of the ilio-psoas and rectus femoris, without assignable cause, have been reported in neurasthenics (Klemperer, Koch, and others).

Occasionally, as I observed in a boy aged 10, the lightning-like contractions of myoclonia are chiefly confined bilaterally to the muscles of the thigh, but the contractions may also be sometimes noted in the abdominal muscles.

When paroxysmal and *unilateral* clonic contractions of the thigh appear, it should always be borne in mind that they may be due to *cortical epilepsy.*

In a case which I described in 1899, and in which Oppenheim found at the autopsy a left-sided tumor of the cortex of the brain, I saw rhythmical contractions of the right extensor quadriceps which took place at the rate of 20 within 10 seconds. The muscles of the calf were also involved in the spasms. The attacks were at first confined to the right leg, so that the patient dragged the leg for one or two days after an attack. In subsequent attacks, the contractions were again in the right thigh, but now attacked also the right half of the abdomen and the right shoulder, the head was drawn to the right, the shoulder elevated, and well developed cortical spasms were produced. In a case of cortical epilepsy after trauma in a boy, aged 11, I saw contractions of the left thigh (extensor quadriceps) which occurred rhythmically but not very frequently in the abdominal muscles, the sternocleidomastoid, the platysma, the muscles of the shoulder and the pectoralis. Immediately afterward there was flaccid paralysis of the left ankle and of the muscles which moved the toes. This form of cortical spasm in the leg is by no means common.

In a case reported by me in 1902, I made a diagnosis of cortical epilepsy on account of paresis of the right leg, the foot reflex, and Babinski's sign, although the attacks invariably began with contractions in the great toe of the right side, next in the leg, and occasionally also in the thigh, usually radiating no further. While, in such cases, hemiparesis and monoparesis are preceded by clonic contractions, these are much less frequently observed as a post-hemiplegic phenomenon. A case of right sided hemiplegia which Bernhardt reported in 1894 is especially noteworthy because in its course a true plantar clonus of the foot appeared, and when the foot was in the equino varus position the muscles of the calf and the tibialis posticus contracted more than 200 times per minute. This plantar clonus was regarded as a variety of so-called post-hemiplegic chorea and athetosis. But only occasionally do slight clonic contractions of the extensors of the toes produce the so-called athetoid spontaneous movements which are observed in polyneuritis, and sometimes also in tabes. We find in literature reports of idiopathic and functional rhythmical contractions of some of the peroneal muscles, usually in young persons (Jobert, Bernhardt).

(3) The very common and painful *cramp in the calf of the leg*, which is of brief duration, and usually occurs after exhaustion, but also under normal circumstances and, as a rule, at night, is a *tonic spasm*, and is caused by any severe or unusual movement (plantar flexion), generally during sleep, and is limited to one side. The calf becomes as hard as a board, is very painful upon pressure, and for a considerable time after. Whether varices play a rôle in its development is very doubtful. But it is certain that an abnormal blood mixture is a predisposing cause, because spasm of the calf of the leg is observed in chorea, in enteric fever, in diarrhea, in diabetes, in alcoholism [and gout], also after pregnancy in which, perhaps as a symptom of stasis, it is particularly common. Local pain favors the peripheral origin of these cramps.

Much more rare are tonic, idiopathic, and extensive muscular spasms of the leg which chiefly occur in walking. A locksmith, aged 31, who came under my observation, suffered for six months when walking or when carrying loads from attacks of clonic spasm in the right tibial region which began

with brief contractions of the flexors of the toes. In the attacks which I saw, the tendo Achillis was contracted to its maximum so that passive dorsal flexion of the foot was absolutely impossible. Besides this tonic spasm I saw brief contractions of the flexors of the toes, and as the spasm in the calf of the leg ceased after anodal galvanization these were also noted in the peroneal region. The attacks lasted almost three hours. The tendon reflexes were unchanged, but the mechanical irritability of the muscles seemed to be increased without increase of the electrical contractility. Under rest and galvanization for two months, the attacks became less numerous and of shorter duration. This case may be grouped with occupation spasms of the lower extremities such as Duchenne saw in a lathe worker, Eulenburg in silversmiths, Stuertz in tanners, and Schultze in dancers in consequence of toe-stepping.

These cases of tonic spasm which appear on walking or similar movements must be differentiated from those described by Bamberger, Guttmann, Frey, and others as so-called *saltatory reflex spasm.* In this affection, when the foot is placed upon the floor, clonic contractions of the lower extremity are produced and cause the whole body to make a jumping movement. As the tendon reflexes in such cases are always greatly increased, Erb holds that an extraordinarily increased reflex irritability of the spinal cord is the cause. Oppenheim regards the disease as a rare form of hysteria. As a rule, the prognosis is favorable.

(4) Several forms of *tic impulsif* of the lower extremities (walking and jumping tic) have been described by Meige and Feindel; for instance, cases in which the foot is turned toward the other leg, cases of stamping tic, knee flexor tic, and jumping tic. They admit that tic of the lower extremity alone is very rarely met with.

A girl, aged about 7 years, who had been treated by me for hysterical chorea, was again brought to me at the age of 14 because for several years she had hopped with the right foot while walking, and this movement had been constantly growing more frequent. After a few normal steps her gait would be interrupted by a kind of polka step with the right foot, this having the appearance of a voluntary movement. Faradization relieved it within eight days. But a year later she returned with marked tic impulsif, shaking of the head, bilateral nictitatio, etc.

This varied realm of localized spasm has been discussed largely from the standpoint of my own experience rather than from the reports in literature. A bibliography of the subject will be found in the second part of Bernhardt's article, "Diseases of the Peripheral Nerves," in the second edition of Nothnagel's collection which appeared in 1904, as well as in Binswanger's "Hysteria" (1904) in the same work, and in Meige and Feindel's "Tic, Its Nature and Treatment."

THE PRESENT STATUS OF GRAVES' DISEASE (EXOPHTHALMIC GOITER. BASEDOW'S DISEASE)

By A. EULENBURG, Berlin

When the Merseburg physician, Karl von Basedow, about 1840 published in Casper's Wochenschrift his article, "Exophthalmos from Hypertrophy of the Cellular Tissue in the Orbital Cavity," he could scarcely have dreamed that he had immortalized his name—still less so that he had left to the world the difficult task of ascertaining the cause of the disease which was named after him.

The nosographic priority and the perpetuation of the name will probably be upheld by German physicians. For although the celebrated Dublin clinician, Robert James Graves, had in 1835 recognized and described a disease which in England and America is called after him "Graves' disease," this consisting in a complication of goiter and exophthalmos (exophthalmic goiter), and the younger Charles Parry of Bath had reported in 1825 five cases of "enlargement of the thyreoid gland combined with enlargement or palpitation of the heart" (only one of which cases showed exophthalmos), Basedow undoubtedly deserves the credit of having first called attention to the *triad of symptoms* consisting of palpitation (tachycardia), enlargement of the thyreoid, and exophthalmos, and of having raised this syndrome from a mere coincidence to the dignity of a nosologic entity, to a clinical therapeutico-pathologic conception. Naturally he gave us merely a clinical foundation; the structure, which as we shall see is yet far from complete, is very different from anything the author of the first article (and of a second which appeared eight years later) could possibly have foreseen. Although we are still laboring over some of the problems of this remarkable *symptom triad* which he and many subsequent observers and investigators worked upon, yet in the course of time they have become more clear, and our insight into their nature, our recognition of the internal connection and the common root of the pathologic structure, has been greatly facilitated and is almost perfect. This at once becomes obvious if we cast a retrospective glance over the views concerning Basedow's (Graves') disease which in the course of time have been gradually evolved. This shows that, upon the whole, there are but three fundamental views, and these have been periodically remodelled into different forms which determine or dominate our clinical conception, as well as our theoretical and practical views of Basedow's disease. Under a few speculative heads these have been massed into theories to which collective designations have been applied, hence we have at present *hematogenous, neurogenous* and *thyreogenous* theories of Basedow's disease. It is very instructive to note how

these points of view, according to the difference in theory, all culminate in the predominant opinions which, even to-day, are incomplete and inconclusive—a circumstance which at first appears to indicate that each of them contains a kernel of truth.

SYMPTOMATOLOGY

Before discussing these questions, however, it is advisable clearly to define our conception and the nature of Basedow's disease, for our present position concerning actively discussed theoretic as well as practico-therapeutic questions will naturally depend upon our conception of Graves' disease. As is well known, the original and apparently precise limitation of the symptom triad—*cardiac palpitation* (or, better, tachycardia), *goiter* and *exophthalmos* —in the course of time was subject to many changes, which sometimes tended to complete or transform, sometimes to limit or narrow, the pathologic picture. Above all, a series of valuable and clinically remarkable differentiodiagnostic symptoms were subsequently included as common although not invariable accompanying phenomena of these three cardinal symptoms. Among these were the eye phenomena known as Graefe's, Stellwag's and Moebius's symptoms; the muscular weakness, Marie's tremor of the members, the decrease of galvanic resistance determined by Vigouroux and Charcot (which Martius, Kahler and myself designated as a rapid loss of power of resistance and a diminution of the relative minimum of resistance); the symptoms of rapid metabolism, the sweating, the gastric crises, the copious watery diarrhea, the sensations of heat, the abnormal pigmentation and discoloration of the skin, leukoplakia, alopecia, amenorrhea and dysmenorrhea, polyuria, albuminuria and alimentary glycosuria which Chvostek first recognized as very common (not so regarded by H. Strauss), the typical emaciation and cachexia; in a word, that entire group of symptoms indicative of neurasthenia and hysteria, and consisting of nervous psychical disturbances (occasionally developing to complete psychoses!), which became so predominant in the pathologic picture that some authors on this account regarded Graves' disease as in the main merely a localized and peculiarly modified form of hysteria. On the other hand it did not escape the attention of the earliest observers of the disease that one or even two of the three "cardinal symptoms" may for a time or permanently be absent—in cases in which, otherwise, the clinical course was typical of Graves' disease. Exophthalmos is most often absent, more rarely goiter and tachycardia—in very rare cases exophthalmos alone is found, but it then manifests the characteristics of the exophthalmos of Graves' disease (so-called "exophthalmus cachectique," after Fischer). Naturally it must be observed whether the absence of individual and leading symptoms is only transitory, whether it is observed in an early stage of the disease, or whether it persists throughout the entire course of the affection so that the pathological picture is incomplete ("abortive" or "rudimentary" form). It has become the custom, following Trousseau, to designate these cases by the term this author invented, "maladie dite fruste par l'absence du goitre et de l'exophthalmie," and these cases were later designated by Charcot as the "*forme fruste*" of Graves' disease. The expression "fruste" (which is derived from coins whose impress is indistinct from use) does not

actually apply to the cases of rudimentary development. It is better adapted to those in which, in the course of the disease, spontaneously or under therapeutic influence, the chief symptoms either disappear or improve and thus obscure the typical character of the affection. At all events this teaching of a "forme fruste" has led to many errors, and, in particular, cases have been designated as Graves' disease and treated by operation in which there is grave doubt as to the diagnosis. Also under the modern appellations of "secondary" or "symptomatic" Graves' disease, in contrast to primary or genuine, "iodin thyreoidism" and "iodin Graves' disease," etc., many a suspicious case may have been treated.

DIAGNOSIS

How shall we meet these difficulties in diagnosis? We must start from the fact that there may be either constant, specific, or pathognomonic symptoms of Graves' disease—that, therefore, the diagnosis can never be based upon the presence or the absence of a single symptom; on the contrary, when the cases are at all doubtful the diagnosis should be based upon the general habitus, or the entire course of the disease. Compensation for the three cardinal symptoms by others perhaps less important may be of value in determining the signs of the disease. If, for instance, there is no trace of exophthalmos but very conspicuous psychical (neurasthenic or hysterical) symptoms and secretory trophic disturbances, such as sweating, diarrhea, amenorrhea, emaciation, alimentary glycosuria, edema, etc., or the distinctly developed symptom of a galvanic loss of resistance, we can hardly doubt that the case is one of Graves' disease; if, on the other hand, neuro-psychical and secretory trophic anomalies common in this affection are absolutely and permanently absent, even though we note the concurrence of the three cardinal symptoms, and especially if one of these be absent, the diagnosis cannot be regarded as positive.

It is evident that the diagnosis does not depend so much upon the mere presence of the most important symptoms of Graves' disease as upon the characteristic manner of their appearance—a circumstance which in modern casuistry is not always sufficiently appreciated. The mere presence of *goiter* is not enough but it must show the specific composition of the goiter of Graves' disease, must be of a soft, elastic consistence, must be almost uniformly distributed, and accompanied by thrills, pulsation, and a loud, roaring, often systolically increased murmur—briefly, by all the peculiarities which from the onset *clinically* distinguish the *hyperemic, vascular* goiters due to hypertrophy and hyperplasia, attended by degeneration of the thyreoid gland, from cystic degeneration and malignant tumors. I do not here consider the question in how far such differentiations are justified from the standpoint of the pathologic anatomist—which, from the recent findings of Lubarsch, Renant, Goffroy, Greenfield and others, may be doubted, inasmuch as the goiter of true Graves' disease is to some extent a simple vascular goiter undergoing the hyperplastic change of the thyreoid, and to some extent an "autonomous neoplasm" (Lubarsch) with the formation of irregular, greatly dilated cavities which usually contain no colloid and very little or no granular and coagulated fluid. Nor does the mere demonstration of exophthalmos

suffice, but this must be bilateral and uncombined with dilatation of the pupils, while the coincidence of certain other disturbances of the eye, of Graefe's, Stellwag's or Moebius's sign, as well as persistent nutritive disturbances of the bulbus, may under some circumstances help to verify the diagnosis. [Unilateral thyreoid enlargement and unilateral exophthalmos are not rare in undoubted cases.—Ed.] Finally, tachycardia and cardiac palpitation in themselves are not sufficient, but we may expect with some degree of regularity not only a paroxysmal but a persistent and almost habitual appearance of this cardiac neurosis which may exist with a normal or but slightly altered blood pressure,[1] with or without anemic murmurs or other anomalies in the vascular apparatus, and with the signs of consequent cardiac insufficiency, dilatation, etc. The other symptoms, which, like the tremor, the mental state, etc., reveal to the eye of the practised observer much that is characteristic, are analogous, and in doubtful, atypical, and rudimentary cases often aid materially in the diagnosis.

THEORIES CONCERNING GRAVES' DISEASE

With these preliminary remarks we turn to the consideration of the three principal theories in a historical sense, the earliest of which is the "hematogenous theory" which emanated from Basedow himself, and corresponded to the humoral pathological view of that period, that the disease was due to a faulty, anemic, and chlorotic admixture of the blood (accurately depicted in his second report in Casper's *Wochenschrift*, 1848). Subsequently many authors concurred in this opinion or have held similar ones (Helfft, Lubarsch, Cooper, L. Gros, Prael, Fischer, Hiffelshein, Mackenzie, Taylor, Warburton Begbie). Among the arguments in its favor was the overwhelming frequency of the disease among females, its usual appearance during youth, its very frequent association with menstrual disorders, particularly with amenorrhea, the favorable influence of pregnancy in isolated cases, and later reports of its frequent coincidence with enteroptosis (Schwerdt). Other observations, such as the hereditary tendency of the disease, its often acute development after trauma, severe somatic and psychical shock, and the like, might with equal justice point to a neurotic origin of the affection. Still others, such as its combination with severe intestinal affections which has recently been much emphasized, with jaundice, etc., might indicate an autointoxication from the intestinal tract changing the secretory activity of the thyreoid, which we shall later consider. At all events, the arguments which favor the hematogenous theory are opposed by many others, and that an anemic, chlorotic, altered blood mixture is the etiologic cause is at least exceedingly doubtful. Aside from the facts that the disease may occur in men (although decidedly less often), that it is met with in children and in women during the climacterium, that pregnancy, as a rule, has an unfavorable rather than a benign effect upon the disease, this hypothesis does not even explain the three chief symptoms, since exophthalmos does not appear in ordinary chlorosis, goiter is rarely present, and when it is it differs decidedly from the goiter of Graves' disease, and tachycardia is neither markedly developed nor so persistent.

[1] Compare "Die Untersuchungen von J. Donath," *Zeitschrift f. klin. Med.*, Bd. 48, Heft 1 und 2.

The other symptoms of Basedow's disease show no intimate relation to the ordinary symptoms of chlorosis and chlorotic anemia. Moreover, Graves' disease attacks those who are neither anemic nor chlorotic, and examinations of the blood carefully and frequently made according to modern and exact methods, the estimation of the hemoglobin, the counting of the blood-corpuscles, etc., have given varying and contradictory results. Hence, according to the earlier views, an anemic and chlorotic state of the blood cannot be regarded as the true cause, the specific pathogenic agent; there must still be an unknown quantity. On the other hand, it shows that the hematogenous theory cannot be completely set aside, but that it must be regarded as a *factor* in the newer conception of a thyreogenous autointoxication, and must be included within this.

The *neurogenous* theory may, in a certain sense, be attributed to the earlier authors, particularly to the Driburg physician, Brück, who in Casper's *Wochenschrift* (in 1840 and also in 1848) also called attention to the similarity of these nervous disturbances to hysteria, and even disignated the exophthalmos in Basedow's disease as "buphthalmos hystericus." But, instead of following further this true and fruitful conception, which only a few attempted, authors lost themselves in byways, such as the one-sided and exaggerated pursuit of animal experiments which then and long afterward afforded abundant opportunities for application to human pathology. Although Benedikt more than thirty years ago declared very ingeniously that nothing was so dangerous as "to find a narrow physiologic bridge just wide enough to stand upon," yet, for a long time many authors considered the sympathetic bridge, others the vagus bridge, and still others the bridges including the medulla, to be broad and firm enough to support weighty and cumbersome theoretic loads. As a matter of fact, in a retrospective survey after this lapse of time, we can scarcely suppress the feeling that the greatly lauded advance of experimental physiology at that period occasionally exerted a most unfavorable influence, pathologic facts frequently being distorted into one-sided and erroneous conclusions which brought many disagreeable disappointments to their enthusiastic adherents and sympathizers for which physiology was not to blame! Thus was evolved, not a single neurogenous theory of Graves' disease but, on the contrary, a consecutive series of theories which disproved one another, and which we may designate as the sympathetic theory, the vagus theory, the spinal, bulbar, and cerebral theories, last of all the constitutional, neuropathic, or neurosis theory. I shall not explicitly present these "theories," most of which, like other speculations, terminate in unsolvable contradictions, but shall limit myself to a rapid enumeration of their fundamental principles (some of which are accepted even to-day).

With the exception of the unfortunate view of Koeben (1855) which was based upon the irritation of the sympathetic by the pressure of a preexisting goiter, the *sympathetic theory* depended upon Claude Bernard's celebrated experiment upon the cervical sympathetic. Aran (1860) and Trousseau (1862) were pioneers in the study of the symptoms of Basedow's disease. but conflicting opinions arose because some of the symptoms appeared to be due to experimental irritation, others to the division of the sympathetic of the neck; for instance, tachycardia and exophthalmos were produced by irrita-

tion, arterial dilatation and goiter by section. Many ingenious attempts were made to solve these contradictions by proving either that tachycardia and exophthalmos were symptoms of paralysis, or, inversely, that goiter was the result of irritation (from stimulation of the vasodilator nerves, according to Benedikt and Ch. Abadie). All this reasoning was more or less artificial, and these forced hypotheses led to nothing convincing. Geigel (1866) tried another method, for, instead of utilizing the sympathetic border column in explanation, he took the vasomotor and oculo-pupillary centers of the sympathetic of the neck in the cervical and upper thoracic cord, and postulated a paralytic condition in the one, and an irritation in the other of these centers, which, however, by no means solved the difficulty but merely transferred it to another point. Others, in opposition to clinical proofs, assumed irritative and paralytic conditions in succession (Horner and Nicati) or they used the vagus to explain the tachycardia resulting from paralysis of the regulatory pneumogastric fibers, as was previously done by Friedreich in paralysis of the vasomotors of the coronary arteries running in the sympathetic. When Filehne's experiments were made known (1879), and were confirmed and supplemented by Durdufi, and subsequently by Bienfait and Troitzki, everything seemed for a time to favor the *bulbar* theory, especially of the areas of the fourth ventricle and corpora restiforme, injury to which caused an increase in the pulse rate (from exclusion of the regulatory pneumogastric center) as well as moderate and often unilateral exophthalmos and—in isolated cases—hyperemia of the thyreoid. A few positive autopsy findings (Mendel, Leube, later Kedzior and Zanietowski,[1] Dana[2]—to which numerous negative ones were opposed, as was the case with the preceding sympathetic and pneumogastric hypotheses—appeared to confirm the *bulbar* theory, which for a considerable time found some noted advocates (such as Brissaud at the Bordeaux Congress, 1895). But the inconstancy of the results of investigation, the inequality of the points of attack found by different experimenters, and the want of analogy with most of the symptoms of Graves' disease, led to reflection; for example, it was noted that true goiter never developed in the animals experimented upon!

The results of this theory did not come up to the expectations, and repeated attempts to trace the origin of the disease to the cerebrum, and perhaps even to localize it in the cortex, also failed. Careful observation of the clinical facts and their consideration made it certain from the onset that all attempts at localization within the central nervous system must necessarily fail to explain the manifold symptom-complex of exophthalmic goiter; hence, as was clinically justifiable, nothing remained but the *constitutional neuropathic or neurosis theory* mentioned at the beginning of this article, and in various quarters this was accepted—lately with special force by Buschan. It cannot be denied that there is much to favor this theory; among other considerations the previously mentioned etiologic factors, the hereditary and family occurrence of Graves' disease, its acute development after trauma and

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[1] *Neurol. Centralbl.*, 1901, Nr. 10, p. 438, "Atrophie des linken Corpus restiforme. Bei Lebzeiten rechtseitige Strumectomie und Ligatur der linken Schilddrüsenarterien."

[2] *New York Med. Journ.*, June 14, 1903, "Changes in the Bulbar Nuclei, especially of the Pneumogastric."

severe psychical shock, especially after fright, its coincidence with other constitutional neuropathies, with hysteria, neurasthenia, hemicrania, epilepsy, chorea, paralysis agitans, etc., and even with conspicuous functional psychoses (melancholia, mania). Nevertheless, many of these conditions can be otherwise explained; certainly the neurosis theory does not embody the "entire and full" truth; like all other neurotic hypotheses it leaves unsettled the question as to the origin of the disturbance in the mechanism of the nerve, the exciting and specific cause of the disease, and the pathogenic agent to which the nervous system succumbs. Here, as in the hematogenous theory, we are confronted by an unknown X whose satisfactory solution appears to be promised by the third theory, the *thyreogenous.*

This theory, in so far as it attempts to deduce the other symptoms of the disease from a previous and *primary local affection of the thyreoid gland,* is not of recent date. In the form of the mechanical thyreoid gland theory it was first proposed by Koeben in 1855, then in the sixties by French authors (especially Piorry and Cros); according to this view, tachycardia and exophthalmos are pressure phenomena due to the enlargement of the thyreoid gland, but the obvious and just objections to this theory of course made it untenable. What, however, may be justly alleged against this mechanical theory of the thyreoid gland nowise applies to the later chemico-toxic (autointoxication) theory we exclusively hold at this time. On the contrary, this is absolutely the product of morphologic, physiologic and clinico-therapeutic facts which, as is well known, have recently led to a complete change of opinion regarding the function and importance of the thyreoid gland in the normal organism as well as its functional anomalies in certain severe and general cachexias. The important relations of these functional anomalies to the pathologic conditions of cachexia strumipriva and myxedema were made clear in 1883 by the pioneer labors of Kocher and others, and, what is especially fortunate and invites further research in this realm, these experiments yielded valuable therapeutic results in the form of organotherapy. These facts in regard to cachexia strumipriva and myxedema were the basis of the new chemical thyreoid gland theory of Graves' disease. Gauthier (1886) appears to have been the first to emphasize that the disturbed chemism of the thyreoid gland in preexisting goiter is an active factor in the development of Basedow's disease, especially the cachexia; but this was purely incidental and led to no decisive conclusions in a pathologic respect. On the other hand Moebius, who designated Basedow's disease as an "*intoxication of the organism from the pathologic activity of the thyreoid gland,*" in laying the foundations of his view called attention to the contrast which appeared to exist anatomically as well as symptomatologico-clinically between myxedema (as well as cachexia strumipriva) and Graves' disease. In the one case there is aplasia and atrophy of the thyreoid gland, in the other enlargement and hyperplasia; in the one case there is slowing of the pulse, narrowing of the palpebral fissure, an impairment of the psychical functions, an increase in weight, coldness and thickening of the skin; on the other hand there is tachycardia, exophthalmos, psychical excitement, general emaciation, loss of structure, and increase of the cutaneous temperature. This might be further complemented by the differences in regard to the excretion of phosphoric acid proven by Wilhelm Scholz (working under Kraus) between myxedema

and Graves' disease. Nevertheless such a diagrammatic comparison is always somewhat artificial, inasmuch as the almost invariable and cardinal symptoms of one disease are offset by the inconstant and secondary symptoms of the other. But, aside from this, there are many other difficulties that preclude a satisfactory understanding of Graves' disease upon the basis of this theory which appears so plausible.

In cachexia strumipriva and in myxedema it may be regarded as certain that atrophy or total absence of the gland and the consequent absence of the glandular secretion bear a direct causal relation to the symptoms of the disease, presumably because the absence of the product of the normal gland, which is necessary for the integrity of all the vital functions, or a certain auto-intoxication produced by the intermediary metabolism of the body, changes or neutralizes these symptoms. The latter view, according to which the thyreoid gland neutralizes the action of toxic products, is regarded by some authors (F. Blum) to have a better foundation than the experimental theory, while the former is clinically more convenient, but is objectionable because the giving off of a glandular secretion to the circulation has not yet been proven. In the study of Basedow's disease it is certainly very difficult to understand the symptoms while we hold to the fact of a hyperemia (and subsequently hyperplasia) of the thyreoid gland and a correspondingly increased glandular secretion. We cannot at once determine how an increase in function and an increased secretion of the thyreoid gland cause "hyperthyreosis," and have such a damaging effect upon the organism as to produce the severe cachexia peculiar to Graves' disease; for, in contrast to the influences which produce cachexia strumipriva and myxedema, nothing comes into question but an increased formation of those specific products of secretion which are said to promote health, or to have an antitoxic effect, or to increase an antitoxic effect. The similarity between presumable intoxication from excessive doses of thyreoid gland preparations and the symptoms of Basedow's disease is too vague. A sharp contrast between Basedow's disease and myxedema, anatomically as well as clinically, appears to be contestable since resemblances and differences between these conditions, as well as transitions into each other, may be recognized; even the secondary development of myxedema, with or after preceding Basedow's disease, has been observed in a few cases (Kowalewski, Sollier, Baldwin, Babinski, v. Jaksch, Hirschl, Faure, Ulrich and myself). On the other hand, this circumstance points to the fact that apparently complicated relations exist between the two forms of the disease. At all events, we do not attain our goal with the mere assumption of an increase in function and a quantitatively increased secretion of the thyreoid gland in Basedow's disease, but must also assume a *qualitatively* changed secretion, and resort to other hypotheses to explain it. Obviously an anomalous *nervous influence* upon the thyreoid gland should be held responsible for this, similar to certain anomalies of secretion of the salivary glands (for example, the paralytic saliva of the submaxillary gland after division of the chorda) which has been experimentally proven. This view is contradicted by Hürthle's investigations into the thyreoid gland, which demonstrate that experimental stimulation of the nerves in animals—faradic irritation of the isolated nerves of the gland or of the pneumogastric and sympathetic trunks in the thoracic cavity—exerts no appreciable influence upon

the activity of the gland. These results can hardly be regarded as final; on the contrary, later experiments favor the view that in the pathogenesis of the altered glandular function—in so far as the cardio-vascular symptoms are concerned they demonstrate the influence of an activity of certain nerve tracts (according to E. Cyon)—the accelerating fibers running in the sympathetic, those from the vagus, and the regulatory fibers from the depressor especially come into consideration. The "thyreogenous" theory in this respect calls for further neurogenous confirmation and completion.

But the experiments of Hürthle offer the possibility of another explanation. According to these, the physiologic as well as the pathologic production of a secretion in the thyreoid gland, of a definite composition of the blood flowing to and circulating in the gland, depends upon the absence or deficiency of certain products in the blood. From this the idea may have originated that in Graves' disease certain *primary changes in the composition of the blood,* the nature of which is at present unknown, produce a glandular stimulation, acting upon the glandular function, the secretion, the growth of the follicles, upon the degeneration in the cellular processes, and thus produces the peculiar, specific, and pathogenic intoxication of the secretion. Quantitative as well as qualitative changes in the composition of the blood of the thyreoid gland probably here act together. The *quantitative* effect is shown at once in the marked arterial congestion and hyperemia of the gland. But a simultaneous qualitative change in the composition of the blood favors certain facts; not only, as has been previously stated, that the blood mixture resembles chlorosis, but also that the appearance of the affection combined with acute infectious diseases or other debilitating conditions, such as hemorrhages, marked loss of fluids, etc., as well as its connection with severe gastro-intestinal disturbances and jaundice, which has lately been emphasized by various authors, favor this view. The last-named conditions remind us of Hürthle's experiments, according to which the tying of the biliary channels and the jaundice thereby produced always changes the activity of the thyreoid gland, and especially increases colloid formation as shown by the abundance of colloid cells and of colloid substance within the lymph spaces and in the epithelial cells. Hürthle maintains that there is no other explanation than the following: The constituents which pass into the blood in biliary stasis produce an irritation of the gland which stimulates it to the increased production of colloid.

That such an increased colloid formation may be combined with an increased excretion of a toxic product, a toxalbumin—the so-called thyreoproteid of Notkin—seems not unlikely from Notkin's researches regarding this substance. Usually thyreoproteid, which originates from the blood by a normal enzyme of the gland—through the "collective substance" (Blum) in which the iodizing process of the thyreoid gland plays a particular rôle—is transformed and rendered permanently innocuous. Why this does not occur in Graves' disease, or to only a decreased extent, may be seen from Hürthle's investigation as well as from some confirmatory reports of French authors. Besides the change in the production of the secretion, an altered and increased discharge of the secretion from the gland and its direct transference to the circulation by venous absorption, avoiding the lymph tract, appears here to play a part. Whether the colloid substance collected in the dilated

intercellular spaces leaves the gland by the efferent lymph vessels or is in part directly absorbed by the veins of the thyreoid gland is still questionable —according to Hürthle's investigations the latter view is by no means improbable. If we consider that, as Renaut has shown, pathologic changes often destroy to a great extent the lymph tracts of the gland and that, like the arteries, the capillaries and veins of the gland are also increased and dilated (the veins even more than the arteries, Dinkler states)—this further confirms such view. We may therefore with some justification conclude that *in Graves' disease the secretion formed in abnormal amount and of altered composition (which, under normal conditions, exerts a partial antitoxic influence), rapidly and without passing into the lymph tract at once enters the circulation and thus produces an increased toxic action.*

Here, as I have previously indicated, the "hematogenous" and the recent "thyreogenous" views of exophthalmic goiter meet, and one appears to be the indispensable complement of the other, although the finer threads of communication cannot at present be positively recognized. That *the glandular secretion is not only quantitatively but also qualitatively abnormal* owing to the altered blood composition is not only shown by the results of experiment but there are *clinical* points of support for this view. I refer to Baumann's proof of the *decreased amount of iodin* in the goiter of Graves' disease. The experiments of Chevalier, who in injecting urine into rabbits obtained a threefold greater toxic action with that from patients with Graves' disease than with normal urine, as well as the demonstration by Boinet, Silbert, and Bienfait of several active toxic ptomains in the urine of Graves' disease, may be utilized in the same clinical demonstration.

Finally, the question must be propounded, Upon which constituents of the organism, which organs or systems, does the toxic material generated by the thyreoid gland exert its pathologic effect? Despite the fatty atrophy of the voluntary muscles lately demonstrated in autopsies by Askanazy, there is hardly sufficient evidence to enable us to recognize a specific muscle poison, as Lemke attempted to do when explaining the cardiac palpitation called "tremor cordis" and exophthalmos. This view is opposed by the circumstance that in the development of exophthalmos the exterior muscles of the eye are not involved, for, as has been demonstrated in total ophthalmoplegia externa, the position of the bulbus in the orbit, as regards the frontal plane is entirely irrelevant (A. Fürst). On the contrary, the organ immediately attacked by the poison must be regarded as the *nervous system,* hence we must assign to *neurogenous* theories the place which they properly occupy in the pathogenesis of Graves' disease. In the thyreoid gland which functions normally we probably have an effective protective organ for the central nervous system. When this antitoxic protective action is absent or diminished it is quite proper to assume that the intoxicating effect of the product exported by the gland implicates more or less severely all parts of the nervous system but chiefly the nerve centers, especially those in the cortical regions which are endowed with higher psychical activity, and thus produces the picture of a neurasthenic-hysteric neurosis or neuro-psychosis. Notkin has attempted to prove that the previously mentioned "thyreoproteid" has first a stimulating and then a paralyzing effect upon the central nervous system. The *first* cause in the pathogenesis of Graves' disease would therefore be the abnormal quan-

titative and qualitative composition of the blood circulating in the thyreoid gland; the *second,* the abnormal secretion by the thyreoid gland of a specific, pathogenic, intoxicating, glandular product; the *third,* the neurosis or neuro-psychosis depending upon autointoxication and subsequently upon cachexia which is so frequently met with in severe forms of exophthalmic goiter combined with a more or less advanced local affection of the thyreoid gland.

TREATMENT

These theoretic views are merely provisional, but in applying them to practice (in this case *therapeutic practice*), which is the best test of every theory, a comprehensive and unprejudiced observation of the natural course of the disease and a varied treatment are the necessary prerequisites, but these conditions are not often fulfilled. Those who love theory, even in therapeutics, are prone to regard everything from the point of view of this theory, and, *vice versa,* any one inclined to a one-sided and uncritical therapeutic method will readily find a theory to adjust to his method. In the treatment of Graves' disease we must guard against such an over-estimation of methods based upon theoretic conceptions. On the other hand, the comparatively brief history of this disease convinces us that each of the previously described theories has affected the therapy, and has enriched it with more or less valuable curative measures some of which at least continue to be of use.

The *hematogenous theory,* according to which we first of all endeavor to improve the pathologically altered blood and the general constitution, suggests the employment of those drugs known since remote times to be "generally roborant" in anemia and chlorosis, such as preparations of iron and arsenic, and including mineral waters containing these substances, as well as quinin, caffein, ergotin, strychnin, etc.; naturally the "heart tonics," such as digitalis and strophanthus, have been less employed because they did not, as a rule, have the desired effect of slowing the heart. However, according to the clearness of the indication for these remedies, we will often be unable to do without digitalis or strophanthus, given periodically and usually with benefit, in the distressing and long-continued attacks of violent cardiac palpitation, as well as in advanced cases of severe cardiac insufficiency with venous stasis, dropsy, and insufficient diuresis. Lately sodium phosphate and sodium salicylate have been especially advised by French authors.

Much more beneficial than most drugs are the *dietetic and physical methods* of treatment which are employed for the same general reasons—to increase the constitutional energy and to change the amount and the composition of the blood. I need only refer to the numerous cases in which astonishing results have often been attained by certain *dietetic cures* (dry diet, vegetarian diet; under some circumstances over-nutrition), also by climatic treatment (prolonged residence even during the winter at high altitudes although not exceeding 900 meters), as well as by different *balneologic* methods. Winternitz, a past master in this realm, expressed himself a few years ago concerning the excellent results of a rational hydrotherapy, strictly individualizing. As is well known, applications to the heart or nape of the neck of water at a temperature of 50° F., or ablutions of the same temperature, often act as a substitute for digitalis. The general effect of these

curative methods can be explained according to very different points of view, therefore adjusted to different theories, but in many cases, fortunately, there can be no doubt of their efficacy or of the cures resulting from them. It is well to remember this, because of the too eager and one-sided operative treatment now so much in vogue.

Neurogenous theories have particularly enriched the therapy of Basedow's disease by the recent addition of *electrotherapy* and *psychotherapy*, methods whose importance and relative usefulness cannot be denied, although in this realm a correct estimation of what has actually been accomplished can be arrived at only with great difficulty. Although by some authorities it was stated a few years ago that electrotherapy in general did not fulfil the expectations that had been created, from my own experience of many years I can admit this only with certain limitations. I shall, however, not enter into this discussion, nor shall I analyze critically the different methods of applying electricity, the question of faradization and galvanization, of the peripheral and central, the local and general application of currents, of franklinization and arsonvalization, or the various forms of hydro-electric baths such as monopolar and dipolar, the alternating current bath, the four cell bath, etc., as well as the "electromagnetic systems" which have lately been proposed. This is, as the old Colonel in Fontane's "Effie Briest" said when difficult and dangerous questions arose, "a wide field," designed for individual habits and hobbies, an extensive playground for all time. Under some circumstances, however, these methods do not deserve the condemnation which has been meted out to them; skilfully and wisely used, and in a palliative, symptomatic, and even curative respect, they may achieve obvious results. Of problematic value, however, is another fruit of the neurogenous theory, particularly of the earlier sympathetic view promulgated about a decade ago (1895) by Jaboulay, and endorsed by Jonnesco and others, "*cervical sympathectomy*"; that is, the resection of the cervical sympathetic, either of the upper and middle cervical ganglion with the nerves connecting them, or also of the lower cervical nodes, therefore the extirpation of the entire cervical sympathetic column. The operation which is undoubtedly based upon incomplete and absolutely false theoretic deductions has not produced the expected results in practice, but has repeatedly resulted in death, the cause of which, as of the sudden deaths after removal of goiter, remains in doubt (such a case, which occurred a few hours after a "successful" operation, has lately been reported by Deshusses [1]). *Stretching* of the cervical sympathetic, practiced by Jaboulay, is not free from danger.

In considering what we owe to the modern application of the *thyreogenous* theory (chemico-toxic or autotoxic) to treatment, two things must be borne in mind: First, the different applications in this greatly disputed realm of diseases of *organotherapy* which changes even from day to day; second, *local operative treatment of the primarily diseased gland* by a partial removal of the goiter, "exothyreopexy," and by tying the arteries of the gland. Another method of treatment, much employed a number of years ago, and which gave such surprising results in myxedema and cachexia strumipriva, consisted in the internal administration of older preparations of the thyreoid

[1] *Journal des sciences médicales de Lille*, 1902, Nr. 52.

gland; following the assumption of a mere hypersecretion of the thyreoid gland, this appeared irrational or contradictory, while, under the assumption of a simultaneous and qualitative change in secretion it appeared to a certain extent justified, especially in the form of *iodothyrin*, and by the proof of a decrease of iodin in the thyreoid in Graves' disease was placed upon a firmer theoretic foundation. However, most of those who employed "iodothyrin" and originally expected great things of it, must have desisted from its use, at least in Graves' disease, as the expectations which had been raised were in no sense realized. The same is no less true of the proposed treatment with other glandular preparations, with thymus extract, ovarin, spermin, bile, etc., as well as with "*protylin*" (Roche), a non-toxic, phosphorus and albumin preparation artificially produced and much employed of late (phosphorus in a completely oxidized form included in the albumin molecule); according to Kocher this was said to be a specific in hyperthyreosis, the value of which in Basedow's disease, according to my experience, is scarcely greater than that of any good nutritive preparation. The last few years have given us various organotherapeutic specialties which, contrasted with the old thyreoidin, etc., are not employed as a substitute for the absent enzyme of the gland, but as a sort of specific *antitoxin, antithyreoidin.* First to appear, and not without the necessary advertising, were preparations under the name of Basedowsan, rhodagen, etc., which were very expensive, but of little benefit in Basedow's disease. Then, upon the advice of Lantz, the milk of thyreoidectomized goats, in which the newly formed protective substance was said to be excreted, was administered to patients with Graves' disease. Recently, under the advice of Moebius, one of the great pharmaceutical firms in Germany has produced a thyreoid serum which is called "*antithyreoidin Moebius.*" It is the blood serum of thyreoidectomized animals (sheep) from which, about six weeks prior to the first venesection, the thyreoid gland had been extirpated. The preparation introduced into commerce is mixed with a 0.5 per cent. carbolic acid solution, and shows an apparently perfect stability; it is sold in sealed glass tubes containing 10 c.c.; the price is $1.50. As the most common mode of administration is gradually to increase the dose, and consequently quite large doses are given (4.5 three times daily!), the contents of a bottle do not last very long and the method becomes expensive. This would be of little importance if the results were certain and the benefit greater than with other non-specific curative methods. But in a number of selected cases treated in this way for a long time, some mild and early cases, some severe ones in later stages, I have not been convinced of any reliable, certain, or specific effect from this antithyreoidin. In 3 of 6 cases I saw a remarkable decrease in the size of the goiter (in a recent and but slightly developed case it almost disappeared), but the other symptoms of the disease, especially the cardiovascular disturbances, were influenced but slightly or not at all. In one case these temporarily decreased but there was later a decided relapse; in another case there was an increase of tachycardia with marked irregularity of the cardiac action and aggravated dyspnea. In the other cases there was absolutely no result. As to the mode of administration, the remedy should be given internally in gradually increasing doses, beginning with 0.5 (therefore 10 drops) three times daily; according to the advice of Schultze's,[1] which

[1] *Münchener med. Wochenschr.*, 1902, Nr. 20.

I found valuable, it is best taken in raspberry syrup or sweet wine. As a rule, I have increased the dose by 5 drops three times daily every third day, until it has reached 2.0 (40 drops) three times daily, after which I decreased it in the same way, and after a short or long discontinuance I have for some time given medium doses (1.0 three times daily). I have seen no disagreeable effects from this method, but, unfortunately, neither have I seen the curative results reported by different authors (for example, Rosenfeld[1]).

From this point of view, therefore, thyreogenous treatment is not a satisfactory therapeutic resource. Frank and impartial consideration of the *operative treatment of goiter*, much resorted to for about fifteen years according to the previously described methods, by no means confirms the brilliant expectations which it raised, which were greatly exaggerated by its surgical adherents, a few of whom were inclined to regard this local operation as a sovereign cure for Graves' disease, and to relegate the affection entirely to the domain of surgery. As upon former occasions, I must again express my doubts of the correctness of this view which is utterly opposed to the true condition of affairs, and depends largely upon a lack of knowledge of the nature of the disease as well as upon exaggerated reports of operative results in earlier literature. After an experience of forty years with more than 600 patients suffering from Graves' disease (I now see at least 30 to 40 new cases every year), I maintain that in the overwhelming majority of cases operative treatment may be dispensed with, that without it and by a well chosen and individualized mode of treatment we may secure at least as favorable results, and that its employment should be accordingly limited to the few rare cases in which there are extremely severe and threatening local symptoms. If we review the surgical cases of the last decade, we find but a few isolated ones which will withstand criticism, in which actual cure or even marked improvement continued after years of observation, and such amelioration frequently related only to the subjective condition, while the predominant objective symptoms, such as tachycardia, exophthalmos, Graefe's sign, etc., were influenced slightly or not at all. We also meet cases which showed no improvement; with those in which the preferred partial thyreoidectomy had to be repeated on account of a subsequent growth, and was then usually without result, so that the question has arisen whether in such cases it is not better to extirpate the entire gland and to compensate for this defect by the lifelong administration of thyreoid gland; finally, we meet with cases by no means rare in which a fatal result immediately followed the operation, the cause often being stated to be "obscure." Of the three methods of operation, thyreoidectomy and ligation of the arteries of the thyreoid gland still show an immediate mortality of 15 to 20 per cent., while Poncet's "exothyreopexy" (exposing and taking out a lobe of the gland which, drawn through the cutaneous wound, is expected to atrophy) has been abandoned as being too dangerous. In the practice of eminent surgeons I have observed a few such sudden deaths after partial thyreoidectomy in youthful persons in whom the affection was by no means severe or hopeless, and I must admit that these cases made a very deep impression upon my mind, as the first law of our professional art is "ne noceamus." Of course I am far from

[1] *Allgem. med. Centralzeitung*, 1903, Nr. 8.—In a case reported by Adam, successfully treated, the cardinal symptom, tachycardia, was absent.

inclined to deduce general conclusions from such unfortunate and isolated occurrences. It must, however, be remembered that the patient does not succumb to Graves' disease unless there are complications; that even cases with apparently most severe cachectic symptoms often improve under hydrotherapy, climatic treatment, and the other previously mentioned methods, and are kept for years in an endurable condition; and that operations dangerous to life should be undertaken only when the patients have sufficient power of resistance and when the severe local phenomena produced by the goiter render the operation imperative. I therefore still consider justified the opinion which I expressed a few years ago concerning this question: Instead of seeking in Basedow's disease a present or future field for surgery, or even of regarding it as a debatable borderland, we should rest assured that for the present we are upon the certain ground of internal medicine, and only under most absolute necessity should we swerve to the surgical side.

An important question and one often difficult to decide is sometimes presented to the physician in discussing the relations of Graves' disease (especially in women) to marriage, and its physical consequences—therefore, the question whether women with Basedow's disease should or may marry. Before answering this question we should consider that in Basedow's disease we are dealing with a severe, often hereditary, degenerative predisposition (neuropsychic constitutional anomaly) as a rule affecting young women, not only attended with great debility but simultaneously with serious nervopsychical disturbances which to a limited extent only are susceptible of cure, and are therefore often permanent. We should especially bear in mind that pregnancy, the puerperal period, and lactation have in many cases a decided and generally unfavorable influence upon the course of Basedow's disease, either because the outbreak of the disease is directly due to these factors or because an already existing affection is decidedly aggravated—especially by pregnancy. It is true a few cases have been reported in which, immediately after a natural, normal, or artificially terminated pregnancy, the remission and even the disappearance of the symptoms was observed. How this influence of pregnancy and the puerperal period becomes operative, whether by transmission from the thyreoid gland to the sexual organs, these being apparently in intimate relation, or by direct autotoxic or reflex action (analogous to the chorea and the tetany of pregnancy) is not certainly known. That in numerous cases there is such an influence is empirically certain, as well as the not infrequent *direct transmission of Graves' disease to the offspring*, the prevailing affection to the females, generally from the mother to the female and exceptionally to male descendants, also the already alluded to combination of Graves' disease with other neuroses and neuropsychoses, as well as with severe vasomotor trophic disturbances and autointoxications (myxedema, scleroderma, Addison's disease, diabetes mellitus and diabetes insipidus). It has been determined by many individual observations that in the families of patients with Basedow's disease not only functional mental diseases and neuroses but also diseases of the heart and constitutional affections, tuberculosis, cancer, diabetes, etc., prevail, and the repeated occurrence of Basedow's disease in one and the same family (Buschan) has been noted. I have under observation a family in which the mother and two daughters suffer from Graves' disease; these two daughters, as well as two other daugh-

ters, are chloroanemic in a high degree, and show the signs of a conspicuous, hereditary, and increased neuropsychic predisposition; in one this amounts to a functional psychosis (melancholia).

If all these experiences are considered, and also the circumstance that the individual symptoms of Graves' disease, the goiter, and especially the exophthalmos when highly developed, awaken a certain physical repugnance, and thereby militate against any idea of marriage—we can scarcely escape the conviction that if a woman has suffered for some time from pronounced Basedow's disease it is a *decided contraindication to marriage*, and from a professional standpoint at least this must be accepted as a rule. Naturally, when the physician's advice is asked, he should consider the special circumstances of each individual case; on the one hand the degree of severity and the duration of the disease, and, on the other, the demonstrable etiologic factors, the influence of heredity and predisposition, of accidental and occasional damage, which may be decisive. When cases arise after marriage, or independently of it, or have been aggravated by marriage, by comparing the indications of a special case with the general laws herein laid down, we often find the treatment a grateful task which is crowned with success.

SEXUAL NEURASTHENIA

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DEFINITION AND HISTORY

NOTHING would appear to be more simple than to define and describe the pathological entity of "*neurasthenia,*" or, at least, to give a comprehensive portrayal, free from uncertainty, of the salient features of its clinical aspect. We are constantly using this term; it is the common property of the physician and of the layman who comes to us with the spontaneous announcement that he is a "neurasthenic"—not even rarely with the additional statement that he is a "*sexual neurasthenic,*" that he suffers from "*sexual neurasthenia.*" Yet, although it is hardly conceivable, and notwithstanding all researches and studies in this field, even to-day no neuropathologist would willingly accept the task of "defining" neurasthenia as an ontologico-pathologic conception in such a way as to be unobjectionable to himself, and both clear and comprehensive. The expression of the New York specialist for nervous diseases, George Beard (who died in 1883), is neither more nor less than the Hellenization of a term used in professional and belletristic literature in Germany for 150 years; moreover, the meaning of the word is debatable, or at least obscure, as it embraces and brings into prominence in a much too partial manner "*asthenia,*" loss of strength, and *weakness* as the empiric and fundamental symptoms of the "neurasthenic" clinical picture—and this appears even more clearly in the synonym employed by Beard, "*nervous exhaustion.*" Contrasted with this, but in just as one-sided a manner, the symptom of increased irritability was formerly given undue prominence by the use of the terms "*nervous erethism,*" "*nervosism.*" To say nothing of other numerous unsuitable designations, it is much wiser to use the earlier, common, and more applicable expression, "*irritable nervous debility,*" which embraces and correctly designates both of the component factors which make up the picture of neurasthenia, *excessive irritability* and *excessive debility or exhaustion* (*weakness*). Under any circumstances, if we desire such comprehensive knowledge of individual symptoms as will permit a clinical recognition of neurasthenia, we must base our study on these two inseparably connected and, to a certain extent, correlated conceptions. Moreover it is evident that in the neurasthenic we are dealing not merely with "nerve disturbances" in the restricted and ordinary sense of the term, but just as much or even more with disturbances of the psychical elementary processes and their effect upon the entire psychical life—not merely with "neuropathic" but also with "psychopathic" conditions, of all grades of severity and of varying importance. In consonance with these preliminaries I should at present like to modify our conception of neurasthenia and portray it as *a widely distributed,*

extremely chronic neuro-psychosis which is chiefly characterized clinically by pathologic disturbances in the equilibrium of innervation and by changing relations between the sensory and motor as well as between the higher cerebral (psychic) neuron systems which bring about association, these pathological disturbances being nosologically revealed by *extreme irritability of the sensory and psycho-sensory neuron systems* and *excessive exhaustion of the motor and psycho-motor (intracentral) neuron systems.* Like all other attempts at definition this naturally offers no actual explanation; still less does it suggest a comprehensive and exhaustive "theory" as to the nature of neurasthenia —it constitutes merely a brief description or is a compilation of its characteristic and chief symptoms. In studying the condition more closely it appears that the *wave of irritation* from sensory impressions in neurasthenics is in a certain sense deepened, and experience teaches that this is true not only for the wave of perceptions of the special senses but also for the subjective feeling chiefly expressed by uneasiness, for the "wave of pain," or, more generally speaking, the "wave of uneasy impressions." In fact it is characteristic of the neurasthenic that this sensation of uneasiness, the "negative impression of sensation" (Ziehen) appears after relatively slight sensory irritations and is marked by disproportionately slight resistance; according to experience this is usually evoked by irritation from other organs, irritations arising from the sensations of their own bodies, which, entering into the consciousness as intensified and long-continued sensations of uneasiness, lead to manifold *sensations of fear and compulsion,* to deeply impressed *conceptions of fear and compulsion,* to the numerous and peculiar "phobias" of neurasthenics. If the excessive exhaustion of the motor neuron is added to the extreme irritability of the sensory neuron, these constitute another origin for the development of the pathologic sensation of aversion. For even mere physiologic fatigue, and still more so the pathological condition of "over-fatigue" or "exhaustion," is accompanied by negative sensory impressions, by conspicuous sensations of discomfort, the "sensation of exhaustion" of Benedikt—and the sensory reaction of the exhausted cells and neuron groups to stimulation, according to the law of exhausted or destroyed nerves, is far more extensive and intense, i. e., painful, than the stimulation of normal, non-exhausted cells and neuron groups. Such a deviation from normal reaction permits us to conclude what causes are necessary to produce a pathologic change in the neuron and in its functional and chief constituents, the nerve-cells — a somewhat specific *"neurasthenic" change of the nerve-cells*—which is of course by no means so clear as the neuralgic and spasmogenic changes (hysterogenic, epileptogenic) gradually explained by a technical investigation which has advanced further and further, and has already given us such great results in the development and employment of staining methods, so that these changes can no longer be regarded as obscure. Next the "*functional neuroses*" and neuro-psychoses which we at this time still regard as neurasthenia, or at least so designate them, will be differentiated from those *organogenetic* and *histogenetic* diseases of the nervous system which present demonstrable material changes in structure, but which are not specially related, and which evidently differ qualitatively as well as quantitatively. Until this goal is reached we must be content to explain the nature of the presupposed "neurasthenic change" by means of the same un-

satisfactory theories which are applied to the elementary processes involved in the mechanism of physiologic nerve activity, at least in a hypothetical form. Obviously, according to our ordinary explanation of functional deviations from the normal, certain specific alterations of *chemism*, of *composition*, of *metabolism of the nerve cells*, the nature of which is still unknown, are decisive. These, reduced to the simplest formula, may be designated as "*a disproportion between consumption and restitution*," as a disturbance of equilibrium between degeneration and regeneration, between mal-assimilation and assimilation, in the protoplasmatic, intracellular metabolism of the nerve-cells. A habitually deficient, or congenitally defective nutrition, an accumulation of functionally damaged products of decomposition in consequence of their insufficient evacuation, we may regard (according to the "*hypothesis of exhaustion*" proposed by Weigert and Edinger) as the basis of the pathologically changed cell activity, and combined with this is the possibility of other general damage by "autointoxication." It is at least a probable, and perhaps subjectively a more satisfactory, explanation of the neurasthenic changes to hypothecate another origin; namely, from *permanent variations in equilibrium or an alteration in the force generated* (*energy production*) *in the nerve-cells.* This involves in particular the "*energetic*" theory of changes in the internal relations of labor which was formulated and published by O. Rosenbach, the energy formation and tension within the smallest labor elements, the "energists" of the nerve cells. This abnormal irritability and exhaustion which, as we may speedily convince ourselves, form the typical symptoms in neurasthenia can then be referred to a too slight "essential" and "non-essential" labor activity of the nervous inhibitive apparatus which is intended to regulate the protoplasmic processes and other forms of reaction; therefore, with relatively slight irritation, a disproportionately severe metabolism of tension material, of thermic and oxygenic energy, takes place. This may be temporarily compensated for by increased action on the part of the vascular system, but, if continued, will inevitably produce the symptoms of "fatigue" and "prostration." For a better understanding of this theory it must be remarked that the form of activity which, in Rosenbach's sense, is to be regarded as "essential" is that depending upon the production of tension from energy, and the "non-essential" is that depending upon great motor activity, that directed to the displacement of masses, whether within or without the organism. The conception of "*fatigue*" would correspond to a consumption of movement energy ("kinetic energy") in so far as the equilibrium of tension is not yet essentially altered; "*exhaustion*" represents a consumption of movement energy with marked diminution of the supply of tension energy which, however, after sufficient rest may again be formed, in which condition a certain amount of energy already formed and held in reserve is drawn upon; finally we have the condition of "*asthenia*" (*insufficiency*) in which it is impossible to produce fresh movement energy, or in which that already existent is insufficient for the essential cell labor required for the displacement of masses. We see that these hypotheses of exhaustion essentially agree, and, on the basis of a theory developed upon a conception of energy, simultaneously result in a finer development and richer confirmation of the hypotheses of exhaustion. Those for whom distinctions of this kind are too subtle may concur with Rudolf Arndt in regarding

neurasthenia as an expression of nerve activity based on *the laws of dying or exhausted cells* (the so-called Ritter-Valli theory), or he may assume with Féré a "decrease of the activity and vibration of the nerve molecule," or, according to the latest theories and tenets, as pathologic "*changes of the tracts*" and "*inhibitions*" of the neurons and neuron groups, the changes in the "tracts" being supposed to be chiefly in the sensory, the "inhibitions" principally in the motor neuron groups; or we may assume that a considerable number of them are increased. These theories add very little to our knowledge; but, like other biologic and pathologic hypotheses, they subjectively bring to us a sensation of relief in that we feel our ignorance less keenly, and, with a few smooth changes, or with symbolic transcriptions and theories, we dismiss these conditions.

No matter what the mysterious nature of the "*neurasthenic change*" may be we have at all events not the slightest difficulty in understanding that in the individual case these changes need not be evenly distributed throughout all neurons and neuron systems, but that either certain neuron systems are especially implicated or that individual neuron systems (either because of a lessened predisposition or a more rapid functional employment, or from these causes combined) are soonest and most markedly subjected to the "neurasthenic change." From a clinical standpoint it therefore appears reasonable to select from an enormous mass the most prominent phenomena which distinguish the clinical picture or give it typical color, and to classify or group these; this, indeed, has been done for some time. Therefore we speak of a *cerebral* and *spinal* form of neurasthenia (*encephalasthenia* and *myelasthenia*) or, to specialize still further, of a *cardiac*, a *vasomotor*, a *gastrointestinal* (*dyspeptic*) and, finally, of a *genital* (*sexual*) form of neurasthenia. The last named, "**sexual neurasthenia,**" which we shall exclusively discuss in the following article, has received a certain historic justification and sanction from the fact that Beard devoted his last years to a special study of the subject, and left an unfinished book which was widely read and occasioned much comment. The differentiation of this variety has, however, sufficient vindication from its uncommon frequency, from its symptomatic and etiologic factors, and, above all, from the extraordinary importance of its prophylaxis and therapy.

What we are to understand by "sexual neurasthenia," and how we may differentiate it clinically from other localized forms of neurasthenia, is clear from the preceding. Here we have a collective designation originating in, and corresponding essentially to, practical requirements, and the extensive prevalence and severity of this affection demand for it our attention; it is *a neurasthenia with local genital symptoms* (*either exclusively predominating or only periodically*), i. e., *with symptoms of "irritable weakness" and of increased irritation and exhaustion in the course of the genital nerve apparatus.* On further study of this disease we note what is confirmed by universal experience, that the pathologic disturbances of the equilibrium of innervation are nosologically revealed by *excessive irritability of the sensory and psychosensory systems, by excessive exhaustion of the motor and psycho-motor systems belonging thereto, by predominant sensations of disinclination*, and, in consequence of these, by general reactions upon the entire sensory system and the imagination.

We shall, therefore, first discuss the *general and special symptomatology*, then its *etiological* relations, and, finally, the *prophylaxis* and *treatment* of sexual neurasthenia in so far as this is practicable within the scope of a single chapter and from an individual point of view.

GENERAL SYMPTOMATOLOGY

The fundamental symptoms of neurasthenia are found in sexual neurasthenia, intimately related to the processes of sexual life; and the corresponding local color is given in the form of *psychical* ("*psycho-sexual*") disturbances as well as, in a more restricted sense, by nervous (somatic nervous) disturbances and changes. The latter are usually noted in the area of the genital nervous apparatus with its widely distributed centers and tracts of projection, its cerebrospinal, intraspinal, spino-sympathetic, spino-peripheral and sympathetico-peripheral neurons.

Among the chief *psychical* (*psycho-sexual*) symptoms we find primarily *anomalies of sexual sensation* which, following the general law of neurasthenic disturbances in sensation, are manifested by sensations of aversion, of fatigue, and of pain. The "wave of irritation" in this sphere of sexual sensations is also depressed, and thus, upon relatively slight irritation, disproportionately distributed and long-continued reaction appears, the "negative sensory phenomena" which dominate the sensations of pain and aversion—therefore "*psycho-sexual hyperesthesias* and *paresthesias*." Among the symptoms produced is a repugnance to natural sexual intercourse, its rare occurrence or complete discontinuance, aversion which may increase even to psychical pain, and terminate in sensations of fear and the "phobias" peculiar to neurasthenics. If these derangements involve more deeply the play of the imagination and become firmly rooted, normal sexual conceptions are weakened, are subject to greater inhibitions, with their disappearance sexual coldness and dislike develop, and ordinary sexual impulses may be completely lacking; on the other hand, many resort to improper associations by means of which new or previously excluded fantastic ideas develop in the sexual realm of the imagination, and an uncontrolled fancy allows these to fructify into pathologically perverse images and impulses. Here may be found the strongest roots and the cause of *onanism*, which is practised by the neurasthenic not only prior to normal sexual intercourse but afterward, even in the married state, and at all ages, and is preferred to ordinary sexual intercourse because usually not immediately conducive to sensations of aversion and fear, and because it presents to the fancy ever changing and desirable imaginary conditions.

Onanism is, therefore, not only (as is ordinarily assumed) the most common cause of sexual neurasthenia but, perhaps just as often, the symptom and sequel of sexual neurasthenia developing from pathologic sensations of repugnance experienced in normal sexual intercourse; at all events this circumstance favors the return of many neurasthenics and non-neurasthenics to these "premiers amours." To the same extent, and for the same reason, there is a tendency to *various anomalous forms of sexual intercourse* (in marriage and outside the bond) because these—not yet associated with distressing fears—appeal to the imagination as desirable, and lead to the most curious

and most severe *psycho-sexual abnormalities and perversions.* Here are concealed the deeply embedded roots from which springs the sexual neurasthenia of individuals already predisposed to pathological, psycho-sexual, and dangerous habits, to *sadism, flagellation, masochism, fetichism, exhibitionism, pederastia,* and other perversions of the sexual instinct; finally, to *sexual inversion* in the form of acquired homosexual desires of "uranism" and tribadism.

Those who reject normal sexual intercourse as antipathic, or who find this act produces distressing sensations of aversion and fear, and those whose psychical and mental predisposition so inclines them, constantly search for new and artificial means of satisfaction; they may find these, at least temporarily, in pederastia and irrumation, in active and passive flagellation, in coprophagy and "ideal" coitus, in all modes that a creative fancy can suggest—and these are numerous.

It must, however, suffice if we merely indicate the paths leading to the mysterious and magic realm of "*sexual psychopathy,*" which from this distance is viewed with wholesome awe. We deny ourselves a visit to this region, and return at once to the more certain ground of the *somatic-nervous,* pathologic disturbances.

Here we first consider the various *sensory disturbances* in the region of the genital nervous apparatus which are due to excessive irritability and appear as sensations of aversion and pain, hyperesthesia, paresthesia, and dysesthesia of the genital projection tracts. On account of the intimate relationship of the nerve tracts in the uropoietic and genital apparatus, especially in the male, sensations in the lower parts of the urinary and genital passages (including their associate organs) cannot be distinguished; hence we find in addition to the hyperesthesias and dysesthesias immediately associated with sexual intercourse painful sensations also in the urethra and bladder (*urethralgia* and *cystalgia*), spontaneous neuralgic pains and pain on micturition (*dysuria*), as well as pathologic reflexes originating from the sensory nerves of the bladder and urethra in the form of *cystospasms* and *urethrospasms* which are usually painful, often a sense of pressure during the discharge of urine (*strangury, tenesmus vesicæ*) and spastic *ischuria* due to spasm of the smooth internal sphincter of the bladder and of the transverse striped compressor urethræ. These conditions may be combined or may alternate with one another. The path of these reflexes is through the center of the bladder innervation which is supposed to be located in an indefinite area between the center for erection, the center for ejaculation, and the center for the rectum, and closely adjacent to these in the lowest portion (sacral) of the spinal cord and above the terminal part designated the conus medullaris. This opinion has lately been combated by L. R. Müller[1] for weighty clinical and experimental reasons. The voidance of urine is probably brought about by the *sympathetic* nervous system, by the hypogastric nerves, and the nervus erigens. The spinal tracts which conduct it communicate to the centripetal spinal fibers of the brain by a reflex action the bladder's condition of fulness. While the detrusor is innervated by the nervus erigens which originates from

[1] L. R. Müller (Erlangen), "Klinische und experimentelle Studien über die Innervation der Blase, des Mastdarmes und des Genitalapparates." *Deutsche Zeitschr. f. Nervenheilkunde,* Bd. XXI, 1901.

the three first sacral roots (Rehfisch [1]) of the hypogastric plexus, the tonus of the internal sphincter is maintained and the closure of the bladder is effected by the inferior mesenteric ganglion and the hypogastric nerves arising therefrom. According to Müller, the reflexes which are here produced do not occur in the spinal cord but below the conus medullaris in the sympathetic ganglion nodule of the pelvis, especially in the inferior mesenteric ganglion and in the ganglia of the hypogastric plexus where the spinal nerves terminate as pre-cellular fibers, and from which the post-cellular nervi of the hypogastric nerves and of the nervous erigentes pass to the bladder. We observe that the process of *erection* also originates outside the spinal cord, namely, in the sympathetic ganglion nodules on the floor of the pelvis which, by means of the nervi erigentes, supply the vessels of the corpora cavernosa. The impulse for the first act of *ejaculation,* the opening of the seminal vesicles, is also conveyed chiefly through this ganglion, while the second act, the discharge of the semen, is produced by the reflex contraction of the transverse striped ischio-cavernosus and the bulbo-cavernosus muscles in which act the spinal cord is also implicated. The ganglion cells for these muscles are apparently situated in the lowermost parts of the spinal cord (conus medullaris).

Of course, for our fundamental consideration nothing of importance is changed by this transposition of the most important urogenital reflexes from the sacral cord to the pelvic ganglia of the sympathetic. But it compels us to reject the contradictory and untenable theory of a so-called "*lumbar cord neurosis*" which has for some time been criticised, not only from a clinical but also from an anatomico-physiologic standpoint. It would be well if this misleading expression were eliminated from our vocabulary!

If we adhere to this theory, particularly in studying the *motor secretory* disturbances in the course of the genital nervous apparatus in sexual neurasthenia, we must consider its various divisions—which, however, are also characterized as "stages."

The most important of these disturbances are *pathologic pollutions* and *erections,* and the *various forms of spermatorrhea and prostatorrhea;* above all, genital debility, the *neurasthenic forms of impotence* (which in numerous cases constitute the chief symptom or, often for a long time, the only one). These manifestations, because of their great practical importance—especially pathologic pollutions and impotence—call for a somewhat comprehensive and special symptomatologic description, as they frequently necessitate special treatment and local therapeutic measures.

SPECIAL SYMPTOMATOLOGY

(a) PATHOLOGIC POLLUTIONS

The expression "pollution" means nothing more than a discharge of semen apart from the act of cohabitation but, as a rule, accompanied by sexual irritation (in the female there is a corresponding discharge from Cowper's and Bartholin's glands). The question arises whether and under

[1] E. Rehfisch, "Ueber die Innervation der Harnblase." *Virchow's Archiv,* Bd. 161, 1900.

what circumstances—particularly in men—these pollutions are to be regarded as pathologic, and whether there are normal pollutions which may be regarded as physiologic. The great majority of authors undoubtedly regard the nocturnal emissions which occur in otherwise normal and mature males, apparently without special cause and at long intervals, as physiologic and normal. Hence males in whom, after the age of puberty, such emissions do not from time to time occur—and to my knowledge there are not a few of these—would resemble mature females who do not menstruate, and who are therefore abnormal. I do not concur in this opinion, but, on the contrary, believe that an emission is in a certain sense abnormal and due to anomalous or increased irritation, although it is unnecessary to at once regard it as pathologic; for instance, it may be like cough or vomiting, which are not "normal," yet, on the other hand, are not necessarily symptoms of disease. As is well known these depend upon individual conditions, upon the mode of life and the nutrition; for example, upon the use of alcohol at evening, upon the position of the genitalia (dorsal decubitus), and upon other accidental and occasional circumstances. However, apart from this question and in answer to those who in a certain sense regard emissions as normal, I will state that there must be a more or less definite limit to this normality, which depends upon where we draw the line and what we regard as the criteria of the normal. The opinion is almost unanimous that no law for this exists, and that it must therefore be based upon a chain of circumstances. The age of the individual, the frequency and the time of the emissions, their composition, particularly when combined with erection and orgasm, and the local and general symptoms which follow the act, are important in the differentiation of each individual case.

The occurrence of emissions before the development of puberty and before puberty is established must be regarded as *pathologic*, especially when these pollutions do not occur as isolated instances but are so frequent as to be almost regular nocturnal occurrences; this is often the case in boys predisposed to neurasthenia, whose imagination is early excited by erotic pictures, and who are early addicted to masturbation. I was recently consulted on account of a boy, aged 15, who suffered almost nightly, at least four or five times a week, from repeated seminal emissions, often accompanied by erotic dreams; he was anemic, poorly nourished, and presented the usual symptoms of so-called "school nervousness," especially the characteristic headaches (which continued after excision of the tonsil), digestive disturbances, etc., and who had also long suffered from enuresis nocturna. Cases of this kind, by no means rare, might tempt us to propose for the clinical picture, characteristic to a certain extent, the term *infantile,* or better, *pre-juvenile sexual neurasthenia.* Aside from the early age at which these pollutions occur, their frequency must be regarded as pathologic, as is also the case in adults if these emissions are repeated several times a week, or for a few days in succession, or even several times during the same night; the pathologic nature is all the more obvious if these patients practise sexual intercourse sufficiently and regularly in or out of wedlock. Seminal emissions which occur during the day and not at night, with or without obvious cause, or upon slight mechanical or psychical irritation, are undoubtedly of morbid character. These "*pollutiones diurnæ*" are of much more serious import than the nocturnal, for

instance, pollutions which occur without erection or orgasm, without a feeling of lust, in which therefore the emission or the loss of semen takes place from a flaccid, non-erect organ. This condition is often associated with hyperesthesia and pain in the urethra and bladder, with the previously mentioned reflex spasms of the detrusor and sphincters, the symptoms of strangury, of tenesmus (the so-called "irritable bladder"); or it is combined with diminishing sexual power or complete impotence, and is then to be regarded as a sign of exhaustion and by no means as an irritative phenomenon, especially if, at the same time, the production of semen is reduced, and only a few drops are exuded from the urethra, not actually ejected. The insufficient quantity of semen discharged from the seminal vesicles into the posterior urethra does not distend the pars prostatica sufficiently to produce an orgasm, or the reflex contraction of the ischio-bulbocavernosi muscles by the ejaculatory center in the lowest portions of the spinal cord. In this process, therefore, the center has no function; the semen exudes drop by drop from the urethra, but is not ejected, resembling the condition in disease of the conus medullaris which functionally deranges or permanently destroys this center. Finally we must consider the result of these emissions upon the organism and upon the general constitutional condition of the patient. If this retroaction is extremely severe and long continued, manifesting itself in general shock or lassitude, in debility which is almost complete exhaustion, or in any marked and extremely unpleasant local sensations invariably persisting for a few days, their pathological dignity should not be underestimated, provided that by abnormal frequency, by qualitative deviations, etc., they do not result in the previously mentioned exceptional condition. There has been much discussion as to the nature of this deleterious retroaction of emission upon the general health, and two opinions which are diametrically opposed to each other have been brought into prominence. According to the earlier view, which has recently again been advanced by Donner, the seminal fluid is supposed to contain a valuable substance which can be replaced only with the greatest difficulty; therefore the loss of more or less of this fluid entails serious consequences to the organism. More recent theories attach but slight importance to the seminal losses. The destructive factor manifests itself by functional hyperirritation which primarily and immediately acts upon the affected lumbar cord, also upon near and distant neuron systems, this irritation being conveyed to the entire nervous system, transitorily producing a kind of nervous shock. It is self-evident that the first view, according to which the seminal loss is looked upon as the most serious factor, can scarcely be maintained; for, if this conception were true, the amount of the losses would be of great significance, while, on the contrary, the resultant excessive exhaustion not rarely experienced after very slight loss of seminal fluid and also the "shock theory" necessitate a certain limitation or extension. The consequences which have been described are also occasionally observed in cases where no orgasm has occurred with its attendant effects upon the spinal ejaculatory centers. On the contrary, the seminal fluid which has passed into the pars prostatica is exuded without any phenomena of a centripetally conveyed stimulation. Apparently the implication of the nervous mechanism as shown by the appearance or absence of these consequences is of as much importance as the preceding general condition of the nervous system, *the*

degree of neurasthenic change already existent which, in the individual case, is the most decisive and important feature of the pathologic process.

A condition *in women* analogous to that of pathologic emissions in men is observed in the frequent occurrence of *pollutions*, the so-called *vulvo-vaginal crises* and *clitoral crises.* The secretion is mainly from Bartholin's or Duverney's glands situated at the mouth of the vagina, but it is usually mixed with secretion from the vaginal glands, often also with mucus from the cervix. A touch upon the mouth of Bartholin's glands at the entrance of the vagina, the "erogenous zone," as well as upon the clitoris and other erogenous parts of the female, often produces a copious secretion from the previously mentioned glands, such as chiefly takes place in masturbators, but which also occurs in not a few women during the ordinary act of cohabitation although this is of varying intensity. In the female, the discharge of a more or less abundant, pale, tough, mucoid secretion after lascivious dreams, or even spontaneously, is an unerring sign of sexual neurasthenia, and under similar circumstances is analogous to the pathologic pollutions of male neurasthenics; we hear less of these, and they are not sufficiently understood even by physicians; therefore, when they occur in physical virginity from otherwise normal genital organs they are not correctly diagnosticated.

(*b*) SPERMATORRHEA AND PROSTATORRHEA

According to Fürbringer, "*spermatorrhea*" is a very suitable designation for the emissions independent of seminal discharges, those which occur independently of erection and orgasm as well as without ejaculation, particularly in conjunction with the evacuation of feces and urine—so-called *defecation* and *micturition spermatorrhea.* We are still in doubt as to the origin and importance of these processes. Is this an increased production and spasm of the seminal vesicles supplied with smooth muscular fibers, therefore an "*irritative symptom*"?—or, on the contrary, is it a symptom of "*weakness,*" due to insufficiency or paresis of the ductus ejaculatorius?—or, as must be assumed in many cases, do mechanical factors play a rôle, especially does abdominal pressure either predispose to or directly favor it? In sexual neurasthenia, "micturition spermatorrhea," the discharge of semen with and after urination or the admixture of semen with the latter ("spermaturia") is comparatively frequent, particularly in those cases in which a local gonorrhea is the cause of the condition, and in which spermatozoa are occasionally found embedded in the "Tripperfäden" (see Etiology). Excluding simultaneous micturition the conditions are different in true "defecation spermatorrhea," which in itself is met with much more rarely; here there appears to be either a purely mechanical expression of the seminal vesicles—or, as has been assumed, by a central radiation process the stimulation produced reflexly is said to pass from the center of the rectum in the lowest portion of the spinal cord to the neighboring center for seminal discharge. This view in its present form can scarcely be adhered to if the results recently reported by L. Müller are proven true, according to which the center for defecation as well as that for evacuation of the bladder is not situated in the lowest portion of the spinal cord, but, on the contrary, in the sympathetic ganglion of the pelvis and the rectum so far as the internal sphincter ani is innervated

by fibers of the sympathetic nervous system which originate partly in the hemorrhoidal plexus and partly in the inferior mesenteric plexus. The centripetal communicating branches of these plexuses, for the rectum as well as for the bladder, have merely the function of transmitting the rectum's condition of fulness and the necessity of evacuation. Medullary spinal fibers (through the inferior hemorrhoidal nerve) pass only to the external sphincter ani and the skin in the region of the anus, and conduct the contraction of the sphincter, designated as the "*anal reflex*," the center of which is localized in the conus medullaris. As the anal reflex in neurasthenics is usually increased (Rossolimo [1]) and combined with generally increased cutaneous reflexes—it seems plausible that, not from the reflex of discharge but from the anal reflex at the *termination of defecation*, the seminal glands may discharge into the urethra; on the contrary, as we know, the discharge of semen as such appears to be independent of the spinal cord, and the ejaculation of the semen only is controlled by a center situated in the conus medullaris. It is probable, therefore, that the reflex processes combined in micturition and defecation spermatorrhea should be regarded as occurring *below the spinal cord in the sympathetic ganglia of the pelvis.*

The discharge designated as "*prostatorrhea*," or prostate secretion (usually pathologically changed) without admixture of semen, is decidedly rare in sexual neurasthenia, and probably occurs only in cases due to gonorrheal infection, and accompanied by gonorrheal local disease. In these cases there is either a swelling of the gland from parenchymatous prostatitis or a gonorrheal posterior urethritis, with increased irritability of the prostate, in which the glandular secretion either mechanically (by abdominal pressure) or by a spasmodic contraction of the smooth musculature of the prostate, is discharged into the urethra. The normal prostatic secretion is of thin, fluid, milky, turbid consistence, while in inflammatory prostatic disease it is thicker, and of muco-purulent character; it has the peculiar, so-called seminal odor; on treatment with a one per cent. ammonium phosphate solution it shows the well known spermin crystals; besides amyloid granules it usually contains cylindrical cells of the prostate which indicate fatty degeneration, also round cells and blood-corpuscles in varying quantity; it is frequently observed in the urine in the form of threads, resembling gonorrheal threads, which are especially profuse toward the end of micturition. Aside from its semeiotic importance in differential and local diagnosis, this secretion is not so interesting as other findings in the urine and the urethral secretions which indicate a continuous and local effect from gonorrhea in the course of sexual neurasthenia.

(c) PATHOLOGIC ERECTIONS. "PRIAPISM"

The "center for erection," as a rule, has been located in the lowest portion of the cord near the "ejaculatory center"—a view in opposition to which is the circumstance that in transverse disease of the cervical and thoracic cords, which are clinically associated with irritative symptoms, pathologically increased erection (priapism) is frequently observed; but this condition is rare in isolated focal disease of the lumbar and sacral cord and of

[1] Rossolimo, "Der Analreflex, seine Physiologie und Pathologie." *Neurolog. Centralblatt*, 1891, Nr. 9.

the conus medullaris. After these regions are destroyed erection is still possible.

L. R. Müller has lately published, in an article repeatedly quoted, the experimental proof that the *center of erection in animals* (*dogs*) *cannot be located in the spinal cord* since, after the removal of the entire sacral cord and the greater portion of the lumbar cord, dogs always exhibited intense erection when confronted with bitches in heat, while these erections did not occur after section of the spinal cord in the lower cervical region. Therefore, the tracts which pass from the brain to the center of erection must be situated relatively high, and certainly pass out of the cord in the upper lumbar region. Besides from stimulation (psychical) centrifugally transmitted from the cerebral tracts, erection may be produced reflexly from the periphery in many other ways; by mechanical irritation (friction) of the penis—as in masturbators—and from the stimulation of the secretion which has accumulated in large amounts in the sexual glands (seminal vesicles, prostate gland). This reflex, like the bladder and rectal reflexes and that for the discharge of semen, also occurs below the spinal cord in the *sympathetic ganglia on the floor of the pelvis.* The centripetally conducting tracts of the reflex arc in question pass from the dorsal nerve of the penis, the terminal branch of the common pudendic nerve, to the conus medullaris, thence by communicating branches which have not yet been definitely determined to the previously mentioned ganglia which, on their part, bring about the centrifugal innervation of the corpora cavernosa by the erigens nerve which passes out of the hypogastric plexus.

If in sexual neurasthenia pathologically increased erections occur during the day as well as at night, usually combined with feelings of lust, oftener, however, with sensations of disgust, even of actual pain, which last for hours (priapism), these symptoms of psychogenous origin are in part the expression of abnormal and excessive psychical stimulation which, in the form of erotic pictures and conceptions, passes from the brain to the spinal tracts of the center of erection—or they are due to *reflex stimulations* which pass from the periphery along the paths of centripetal conduction which have been mentioned.

These symptoms are chiefly found in *masturbators* in whom central psychical and the periphero-mechanical irritations are combined in various ways. Naturally, increased erections are relieved by masturbation. Erections are frequent in those men in whom sexual neurasthenia is due to local genital diseases, especially of a *gonorrheic* nature, or to local irritation developed in the posterior portions of the urethra whereby, periodically at least, reflex stimulation of the center of erection produces greatly prolonged erections which form the most common symptom in the initial stages of gonorrheal posterior urethritis. Cases of this kind are usually accompanied by other irritative symptoms previously described, by cystalgia and urethralgia, by reflex cystospasmus (spasm of the detrusor vesicæ) or urethrospasm (spastic ischuria from spasm of the compressor of the urethra), more frequently even with simultaneous or alternating spasm of the sphincters of the bladder and urethra, in the latter case presenting the well known picture of *tenesmus vesicæ, strangury.* If prolonged, other symptoms of increased reflex irritation as well as pathologic erections decrease or disappear, partly because the peripheral sources of irritation gradually pass away, partly and chiefly be-

cause the irritability of the *center of erection decreases more and more,* and a condition of neurasthenic impotence is introduced and dominates the later stages of the affection.

(d) NEURASTHENIC IMPOTENCE

"Impotence"—employed in its widest sense, which is to be immediately discussed—if not the most frequent, is practically the most important symptom of sexual neurasthenia; in the eyes of the patient it is the dominant symptom, and the one which, chiefly or exclusively, compels one-third to one-half of all sexual neurasthenics to seek professional aid; this, too, is the realm probably selected for literary publications—not always above suspicion, forming the subject of monographs by specialists, particularly from a therapeutic aspect.

When patients, among whom neurasthenics are at once detected, complain of increasing impotence for which they seek relief, our first object must be to ascertain from the history whether they are actually suffering from "impotence" or simply from a "decrease in power" in the scientific sense in which we employ this expression. As is well known, we differentiate between "potentia cœundi" and "potentia generandi"; the laity understand the expression only in the first sense, therefore sterility due to aspermatism they do not designate as impotence—and as physicians we also usually understand by this only the *incapacity to carry out coitus in consequence of disturbed mechanism of the genital functions,* as well as of erection and ejaculation. But the laity by no means understand the differences and limitations of these conditions because of which deficient or *absent libido* is satisfied in *perverse channels,* which causes a suspension of the exercise of the sexual functions; these conditions, fundamentally so far asunder, are often confounded, but by careful questioning we soon learn that the supposed impotence consists in a decreased or perverted sexual desire, or more or less pathologic aberrations of this desire, without demonstrable disturbance in power. Only well educated persons who are trained to think can be taught to appreciate this difference between deficient or absent libido and a loss in power. In the same sense a person may refrain from eating because of a want of appetite or because the state of his digestive organs does not permit him to ingest food and digest it. As a rule, not much can be expected from explanations of this kind; nevertheless, we must not fail to make them, for many of these patients with supposed impotence and a dread of *disease of the spinal cord,* "*tabophobia*"—particularly those who have masturbated for some time—are brought to us, and because psychical influence is a powerful force in combating the pathologic symptoms belonging thereto.

Hence, among sexual neurasthenics we find quite a number who are chiefly affected by deficient libido. They are more or less potent — have "power" but do not "desire"; on the contrary there are some who neither desire nor have the power (these are relatively the least miserable); finally there are great numbers of those who have "desire" but very deficient power, and this is finally lost. These suffer from *impotence* in the restricted clinical and practical therapeutic sense—the mechanism of erection and ejaculation being quantitatively affected, or qualitatively changed, or the power appearing to be entirely lost. But here, as is evident from our diagram of the anatomico-

physiologic structure of these organs, very different modes of origin and forms of impotence are conceivable and possible.

In studying this diagram briefly from our present standpoint, we must differentiate three centers which control the extent and function of the male genital organs, namely: 1. *The center for erection*; 2. *The center for the discharge of the seminal fluid*; 3. *The ejaculatory center.* Of these three centers the first two have their seat in the sympathetic ganglia upon the floor of the pelvis (inferior mesenteric ganglion and hypogastric plexus, especially in the plexus seminalis, prostaticus and deferentialis); the third, however, is situated in the lowest portion of the spinal cord (conus medullaris). Erection and discharge from the seminal glands are, therefore, *sympathetic* reflexes, while ejaculation is a spinal reflex. The centrifugal nerve for the purely vasomotor process of erection is the erigens nerve which supplies the corpora cavernosa (this nerve being a virtual continuation of the first three anterior sacral nerve roots which pass to the hypogastric plexus). With sufficient stimulation from the center of erection the reflex is conducted to the center for seminal discharge, the smooth muscle fibers of the seminal ducts, the seminal vesicles, and the prostate contract peristaltically, and the discharge of semen takes place into the posterior urethra, the pars prostatica distends, this being normally accompanied by an orgasm and with irradiation of the reflex to the spinal ejaculatory center. The centripetally conducting tracts which produce these reflexes follow, at least in part, the course of the dorsal nerve of the penis (terminal branch of the common pudendic nerve) to the conus, thence through communicating branches pass to the ganglia on the floor of the pelvis and on upward into the tract of the three upper posterior sacral roots to the spinal cord. The function of the ascending centripetal tracts from the spinal cord to the brain is to convey to the central organ the sensory irritation affecting the erogenous zones as well as the filling and discharge of the seminal glands and the sensations produced thereby. Their cortical terminations and "transcortical" sensory continuations are as little known as the "transcortical" motor beginnings and the course of the centrifugal genital tracts starting from the brain, which apparently increase and also inhibit irritative conditions throughout the spinal cord, conveying them from the spinal cord to the previously mentioned sympathetic ganglia where, like the spinal nerves of the bladder, they terminate in these ganglia as precellular fibers.

According to our diagram, starting from the periphery and passing to the center, we may and must theoretically differentiate the following forms of impotence; i. e., the suspension of the male genital functions:

(1) *Peripheral sensory impotence* due to functional disturbance of the urogenital centripetal tracts which pass to the sympathetic reflex ganglia.

(2) *Peripheral motor (vasomotor) impotence* from functional disturbance in the motor (vasomotor) innervation tracts of the corpora cavernosa—i. e., in the erigens nerve—of the smooth musculature of the spermatic duct, the seminal vesicles and the prostate gland, also of the bulbi and ischiocavernosi muscles.

(3) *Sympathetic ganglion impotence* caused by a disturbance in function of the sympathetic ganglia of the floor of the pelvis which control the reflexes of erection and seminal discharge.

(4) *Spinal ganglion impotence* from disturbance of function of the ejaculatory center situated in the lowest portion of the spinal cord (conus).

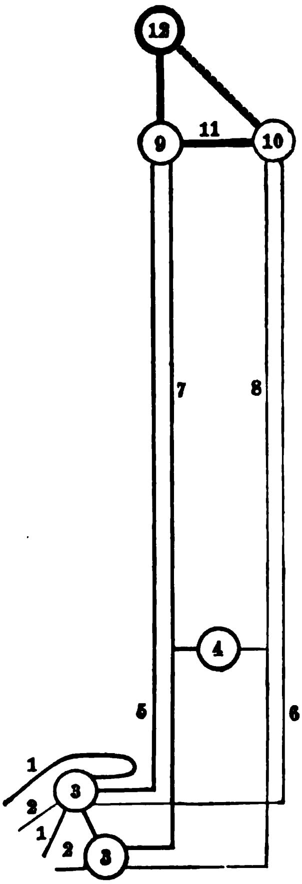

Fig. 195.

(5) *Spinal sensory conduction impotence* from functional disturbance in the spinal portion of the ascending genital centripetal tracts.

(6) *Spinal motor conduction impotence* from functional disturbance of the spinal portion of the descending genital centrifugal tracts.

(7) *Cerebral sensory* and

(8) *Cerebral motor conduction impotence* due to analogous functional disturbances in the intracerebral portions of the corresponding genital tracts.

(9) *Cortical sensory* and

(10) *Cortical motor impotence* from disturbances of function in the cerebro-cortical terminations of the affected tracts.

(11) *Intercentral impotence* caused by disturbance in the function of the communicating tracts of the sensory and motor cortical terminations.

(12) *Transcortical*[1] *impotence* through functional disturbance in the cortical centers beyond those previously mentioned and combined with them, these being higher psychosexual centers, the centers for memory and for sexual conceptions.

We are chiefly interested in the forms enumerated in sections 1 to 4 because these are usually combined or they occur successively and play the leading rôle in neurasthenic impotence. In 1, peripheral stimulations are not conveyed with sufficient strength to the reflex ganglia. In 2, the reflexes produced in the ganglia are not sufficiently strong. In 3 and 4, the reflex transmission to the ganglion itself is markedly inhibited; in 3, in the sympathetic ganglia for the reflexes of erection and seminal discharge, and in 4, in the spinal center for ejaculation. If, therefore, *the fourth form exists without the third, erections and the discharge of the seminal fluid may take place in the pars prostatica, the semen, however, at most is only exuded but no longer ejected. If, on the other hand, the third form exists without the fourth, it is a question whether the decrease in power exists only in the sympathetic center for seminal discharge or in the center for erection, or in both simultaneously. In the former case erections may occur without seminal discharge; in the second, seminal discharge without erection (a discharge of the semen from a flaccid, non-erect organ); in the third case the erections*

[1] A designation for which we have analogous terms in "transcortical," sensory, and motor aphasia, which have lately become common.

are feeble, weak, or not sufficiently prolonged and finally cease, the seminal discharges being scant and eventually absent entirely. In the fully developed cases belonging to this category there is, of course, no question of ejaculation, since no orgasm or sufficient erection and extension of the pars prostatica takes place. Before reaching these terminal stages which indicate decided exhaustion in the genital reflex tracts and centers, in many cases a stage precedes which is characterized by signs of *excessive irritability in the sensory (adduction) tracts of the genital reflex arcs with a more or less developed exhaustion in the motor tracts (abduction).* For instance, while the erections are usually weak and of insufficient duration, and the quantity of the discharge from the gland is decreased, the wave of irritation is still comparatively deep so that ordinary peripheral irritation by the accumulation of blood in the corpora cavernosa is no longer necessary, and the irritation is centripetally conveyed toward the spinal ejaculatory center and upward with abnormally decreased resistance, so that the periodic variations in the function of the three genital centers which follow one another are decidedly lessened, and the entire mechanism functions much more rapidly.

While emission usually occurs only from the erect penis, the friction of which in the vagina produces a sensation of lust, and the irritation of the glandular secretion effused into the pars prostatica originates and increases the ejaculation which culminates in an orgasm—under circumstances described the irritation which leads to erection is sufficient, after the resulting emission, to cause a discharge from the seminal glands; simultaneously with this, the ejaculation of the semen takes place through reflex contractions of the ischio- and bulbo-cavernosi muscles. Therefore in the preliminary stage prior to impotence, the familiar and greatly dreaded *early evacuation of semen occurs, "ejaculatio præcox."*

Combined with the forms which have been minutely described in the preceding, many others are observed in sexual neurasthenia. "Irritable weakness" in the region of the nervous genital apparatus makes it obvious that in neurasthenia we are not only dealing with a "neurosis" in the ordinary sense of the word but with a neuro-psychosis, and that the phenomena of functional change and disturbed innervation here observed have a more or less potent but invariable and permanent influence upon the psychical factors. While the activity of the cortical and "transcortical" (psycho-sensory) sexual centers from individual portions of the sexual apparatus is variously stimulated by centripetal paths—by the cerebrospinal conduction tracts which are thus brought into activity, and are indicated in the diagram by 5 and 7—upon the other hand, from these sensory and also psychomotor sexual centers associated with them (intercentral), influences of partly exciting, partly regulatory, and partly inhibitory nature are conducted through the centrifugal conduction tracts 8 and 6 to the peripheral, motor, vasomotor, and secretory centers of the genital apparatus. In referring to what has been previously stated in regard to this subject, I need only emphasize that, from the unanimity of combined psychical relations, this psychical deficit so constantly appears in the neurasthenic that, in the neurasthenic forms of impotence, we must often consider chiefly or almost exclusively "**impotence of a psychical (psycho-genetic) origin.**" In the main this is due to some inhibiting influence, neurasthenic obsessions and constrained conceptions concerning the

mechanical occurrence of erection and its associate processes, intraurethral discharge from the glands, the orgasm, and the ejaculation. Inasmuch as these inhibitive conceptions are usually the result of depression or the product of intense feelings of disgust engendered by former sexual acts or immediately following these, we have before us phenomena of psycho-motor inhibition due to excessive or anomalous psycho-sensory irritation; therefore, true symptoms of "irritable weakness."

These residual conceptions of inhibition, according to their mode of development and the severity and frequency of the preceding sensations of aversion, may be of general nature or may be aroused by special conditions in the individual case, or they may be only slight and transitory individual factors. Where the latter is conspicuously the case, we designate these as "relative" and "temporary" forms of impotence, which are nevertheless always important psychical (psychogenous) forms. By this we mean that impotence is always associated with certain preconceptions and exists under definite circumstances (for example, in natural sexual intercourse, not, however, in unnatural modes of satisfying lust), or of definite persons (for example, of the wife, not, however, for the mistress)—"*relative impotence*"; or it appears only periodically while at other times there is but slight or even unabated potentia coeundi—"*temporary impotence.*" In relative impotence the sensations of aversion and the inhibitions consequent upon these are not generally associated with all but, in the main, with definite forms of sexual intercourse, or to sexual intercourse with certain individuals who for some reason have become repulsive and antipathic; in "temporary impotence" the inhibitive influences which prevent the impulse are transitory and periodic, certainly are not always active to the same extent. Therefore "relative" like "temporary" impotence frequently corresponds to early stages of the affection, which gradually with a further increase of the inhibitive influence becomes absolute and permanent (total) psychical impôtence. This terminal stage is usually reached only after a long time, if at all, and this depends greatly upon the individual conditions, the temperament, the character and mode of life, and especially also upon the morale and duration of the treatment which has in the meantime been instituted.

Of course, *in the female* no condition arises which corresponds to male impotence. What is known in women as anaphrodisia, or frigidity, more closely resembles deficient sexual libido in the male; but it cannot be denied that *the absence or insufficient power of erection* of the clitoris may and quite frequently does exert an influence. This female organ is innervated from the vesico-vaginal plexus, the lowest portion of the inferior hypogastric plexus, as well as by the sympathetic, and in its development shows, quantitatively and qualitatively, i. e., in regard to its erogenous action, extreme variations. Naturally we learn very little in regard to this, for to the physician this area is in every sense a "*noli me tangere,*" and to the great majority of married men the physiology of the female organism is such a *terra incognita* that they neither acquire knowledge which can be utilized nor are they able to impart it. A certain amount of information on this point, as well as on other subjects relating to erotic functions, would in some cases do no harm and would probably prevent many a marital calamity.

(e) HYPOSPERMIA (OLIGOSPERMIA) AND ASPERMIA

Among the symptoms of sexual neurasthenia, particularly in advanced cases accompanied by impotence, diminished secretion of the sexual glands (*hypospermia, oligospermia*) should probably be included—but concerning this we know very little. The secretion of the male genital glands is influenced by sympathetic nerves which originate in the seminal and prostatic plexuses of the inferior hypogastric plexus. These plexuses, as well as the plexus deferentialis connected with them, furnish simultaneously the motor nerves, which, reflexly stimulated, bring about the discharge of the glandular secretion in the posterior portion of the urethra as the first stage of the process of ejaculation. It is not inconceivable that the stimulus originating from the periphery of the genital apparatus may change these motor and vasomotor processes of erection, as well as the secretory processes of the genital gland, in a definite or modifying, quantitative, or qualitative manner. So far as the male generative gland and the secretion stored in the seminal vesicles alone come into consideration, the amount and consistence, the turbidity and viscosity of the seminal fluid, the number of seminal corpuscles contained therein, the appearance and composition and the motility and vitality of the latter may present varying conditions. But we have no definite knowledge concerning these conditions in sexual neurasthenia, particularly if we leave out of consideration those cases which are due to gonorrhea or a gonorrheal local infection in the form of posterior urethritis, prostatitis, epididymitis, etc., and to the more or less marked changes in the formation and composition of the secretion which may appear in the clinical picture.

We are most likely dealing exclusively with such complications in the *alterations of form* and the changes in *vitality of the spermatozoids* which, under the designations respectively of "*asthenospermia*" (Fürbringer) and "*azoospermia*" indicate decided diminution and suspension of motility and loss of vitality of the spermatozoids, which conditions are recognized and dreaded, and play an important part in male sterility. It may be regarded as certain that profound azoospermia, in particular, and consequently absolute impotentia generandi, in itself is never the result of sexual neurasthenia; on the contrary, these cases always prove the existence of another condition, particularly a bilateral gonorrheal epididymitis which is readily recognized. On the other hand, the possibility of a decrease in number ("oligozoospermia"), a decrease in size and also in the motility of the spermatozoids, cannot be denied in isolated cases of severe sexual neurasthenia; corresponding cases may be met with frequently in which pathologic emissions have occurred for years, probably with the usual simultaneous weakening of the potentia coeundi; in fact, a more or less decided decrease of the potentia generandi may appear in these cases. But the difficulties and sources of error in accepting and utilizing findings of this kind—resulting partly from the absence or admixture of the prostatic secretion with the secretion of the seminal vesicles, and from the decrease or lack of acidity of the prostatic secretion, etc.—are so important as to warn us not to be too hasty in the individual case in forming conclusions, especially from uncertain generalizations of such individual findings.

The rare condition known as "*aspermia*," in which there is complete

arrest of the secretion from the sexual glands, is even less likely than azoospermia to be related to sexual neurasthenia. In the majority of cases designated as aspermia (or, more commonly, as "*aspermatism*") there is less of a suspension of glandular production than of excretion, and this is due to congenital or acquired mechanical excretory disturbances; for example, to strictures.

ETIOLOGY

It is difficult to obtain accurate statistics as to the relative and absolute frequency of sexual neurasthenia—as well as of neurasthenia in general. In the hospital and clinic, of course, the affection is not observed, and in polyclinics it is comparatively rare; nerve specialists, on the other hand, particularly in private practice, have frequent opportunities of noting the condition, the cases forming such a large proportion of their clientele that they are inclined to form exaggerated opinions concerning it. To give an approximate idea of its prevalence, I will state that among 3,414 private patients of whose cases I have made notes in the last four years 593—therefore, 17.4 per cent.—presented such symptoms that they may be designated as "*neurasthenics*" (excluding others which denoted complications, and also traumatic cases); among the 593 were 179—therefore, 30.1 per cent. of the neurasthenics, and 5.2 per cent. of the total 3,414 patients—presenting the undoubted type of "*sexual neurasthenia.*" Upon the average, therefore, we may assume that of every three neurasthenics one is almost always a sexual neurasthenic, which may appear somewhat too high a proportion; it is probably accounted for by the fact that sexual neurasthenics, from the nature of their affection, especially when there is decreased or total impotence (or when they believe this), are impelled to seek professional aid.

Sexual neurasthenia occurs chiefly in the male; well developed, typical, pathologic pictures are not observed in the *female*, or are so rarely seen that we might be inclined to combine the pathologic conception of sexual neurasthenia with other *a potiori* closely related symptomatic designations. Among the above 179 sexual neurasthenics there were but 6 women—only to 6 cases could I apply the designation sexual neurasthenia, and then only because of the symptom of pathologic pollutions (without other abnormal local finding) combined with decided and general neurasthenic symptoms. Two of these cases occurred in masturbators, in the anatomical sense both still "virgins," and of the psychologic type of the "demi-vierges"; the others were married women, two probably being masturbators, and two addicted to homosexual practices. Analogous cases are probably not rare, but only exceptionally come under the notice of the physician—these cases are perhaps more familiar to gynecologists than to nerve specialists. In regard to the influence of *age*, sexual neurasthenia is, of course, a disease of *puberty* or of *active sexual* life, therefore prone to appear in the three decades of life between 20 and 50; but neither the earlier nor later life, at least in the female, is entirely exempt. The "pre-juvenile form" of sexual neurasthenia has been partially described under symptomatology, also a "pre-senile" form; but, under sexual neurasthenia another must be considered which actually belongs to senility, and is characterized by decreasing power and growing antipathy to the ordinary methods of satisfaction, and results in an *increasing tendency to sexual per-*

versions, to flagellantism, to sadism, to fetichism and to symbolistic acts, and the like.

Among the "causes" or, more correctly, the *conditions for the development* of sexual neurasthenia, just as for neurasthenia in general, a peculiar neuro-psychic predisposition, a "*nervous constitutional weakness*," as we explicitly designated this in the introduction to this article, plays a paramount rôle. Nevertheless, not to exaggerate the importance of this constitutional factor, the original predisposition, we must not withhold the empiric statement that not a few persons become sexual neurasthenics who at the beginning showed no such predisposition, but in whom the development may be traced to purely local genital affections, to a "genital neurosis" which becomes widely distributed, or to a general neurasthenia. This is analogous to the conditions so frequently found in nervous diseases due to accidents, especially accidents due to occupation—in which at first a localized neurosis in connection with the accident ("traumatic local neurosis") develops and subsequently the slowly progressive symptom-complex of post-traumatic neurasthenia (or hysteria). In other cases neurasthenic symptoms referable to other regions, or to general neurasthenia, long precede, and only when genital local affections appear in consequence of succeeding *occasional* or *accidental* damage does the symptom-complex assume the typical form and color of "sexual neurasthenia."

Such an "occasional" or "accidental" damage, active in the development of the disease, is above all caused by *local diseases of the male genital apparatus* (especially from gonorrhea)—*and onanism.*

Among the so-called infectious "sexual diseases," in a restricted sense, the *chancroid* plays no part, and *syphilis* only a comparatively subordinate rôle, in the development of sexual neurasthenia; in many ways they may produce general neuroses in the form of neurasthenia and hypochondriasis (syphilophobia)—but never or only exceptionally with the local coloring which characterizes sexual neurasthenia. Here we do not so much consider "primary" local affections as the later and ultimate general consequences of syphilitic infection, syphilitic anemia and cachexia—to say nothing of nervous syphilis or of suspicious post-syphilitic ("metasyphilitic") diseases of the central nervous system which may still exist. The most weighty influence is, therefore, to be attributed to *gonorrhea*, the extraordinary nosogenetic importance of which was revealed to us by the pioneer discovery of the gonococcus by Neisser two decades ago, which gradually enabled us to comprehend fully some of the important findings in the pathology of these organs, and also in diseases of the nervous system, our study of which is as yet by no means concluded, being continuously complemented and broadened by fresh communications.[1] Here general neuroses (neurasthenia and hysteria), different forms of nervous local diseases, neuralgia, muscular atrophy, gonorrheal neuritis and myelitis, etc., are most important—in the latter conditions the toxic products ("toxins") which have developed in the course or remained after the cessation of chronic gonorrhea are decisive factors. The conditions which here interest us especially and which have been described,

[1] Compare my article, "Gonorrheal Nervous Diseases." *Deutsche med. Wochenschr.*, 1900, Nr. 43.

gonorrheal local affections of the posterior portions of the urethra, chronic and "latent" gonorrheal forms, neglected forms or those incorrectly treated, especially by too powerful or too protracted local treatment which has intensified the local condition, lead to the development of sexual, and subsequently to general, neurasthenia. Perhaps one-half of all the cases of "neurasthenic" impotence are of gonorrheal origin, and in these cases we will almost invariably find that there was a preceding and prolonged inflammatory condition of the posterior urethra, or even of the bladder or still higher portions of the urinary apparatus, which may still be present—in which condition we do not necessarily always find gonococci in the urine and in the urethral secretion, for gonococci encapsulated in the follicles of the urethra and prostate gland are impossible to demonstrate; besides the examination is often made much more difficult by secondary invasions and mixed infection (bacterium coli, etc.). This is not the place to discuss minutely this condition; in the individual case due stress must be placed upon the finding of gonorrheal local infection, of gonorrheal urethritis, balanoposthitis, cavernitis, epididymitis, prostatitis, spermatocystitis, etc., and upon the proof of prostatic urethral disease; the possible implication of the bladder, ureters, and pelvis of the kidney must also be seriously considered. An experienced specialist should be consulted, for a urethroscopic and cystoscopic examination for this purpose is often indispensable, and is simultaneously of therapeutic interest.

Local affections of the male urogenital organs, other than gonorrheal, are comparatively seldom the cause of sexual neurasthenia; but even these—phimosis, balanitis, varicocele, non-gonorrheal epididymitis, posterior urethritis, prostatitis—if there is no suspicion of infection, must always be considered of pathogenetic importance.

In all of these chronic, local affections it is not so much the nature and severity of the pathologic local changes as the intensity and long continuance of the irritation which acts upon the urogenital nerve terminations—for instance, in the "*area sensitiva*" of the prostatic portion of the urethra—and subsequently its general influence upon the nervous system; not rarely the psychical effect is of decisive importance. The latter becomes still more prominent in the second predisposing condition, *onanism*—which sometimes produces general and sexual neurasthenia not by itself alone, but by "ideal concurrence" with other deleterious agents, such as alcoholism, nicotinism, etc. It is erroneous to regard the influence of onanism as brought about, in the main, by consecutive local pathologic changes, by swelling and inflammation in the prostatic urethra, etc., or to regard such changes as specially significant. These opinions were formerly held, and are to some extent to-day, chiefly owing to the weight attached to the well-known views of Lallemand. In many cases the absence of findings in urethroscopic local investigations proved the fallacy of this opinion, which has been questioned for some time. On the other hand, in the immediate consequences we must differentiate sharply between those due to *masturbation* and to the *psychical nature of onanism.* In the former we seek evidence of the effect upon the sensitive nerves of the external genitalia, the glans, the prepuce, the skin of the penis, inevitable manual and mechanical irritation from friction or other acts of masturbation whereby erection, orgasm, and seminal discharge

are produced reflexly through the sympathetic ganglia, and if the act be frequently repeated, or, without this, from slight exhaustion in the affected tracts and centers, it may cause neurasthenic changes in these areas of the nervous apparatus which may finally become permanent. These irritations from the nerves of the external genitalia (see Impotence Diagram, under No. 1) are increased by irritation centripetally conveyed from the nervous apparatus of the urethra, also from the sympathetic centers, even when an orgasm has followed, and there is a consciousness of satisfaction, perceived as an increased sensation of lust, which may almost cause syncope. Hence the cortical, transcortical, and psychical centers may gradually be implicated. This occurs to the greatest extent in the cases in which from the onset there is uncontrolled, perverted action of the fancy, or onanism of the most exhaustive kind. Not rarely they are highly educated persons, people of intellect, who succumb to these refined but undoubtedly dangerous "psychical" forms of onanism. While the human idiot, like the ape—"*simia quam similis turpissima bestia nobis*"—in masturbation merely obeys a dull impulse without "thinking" of, or experiencing, anything more than momentary "animal" satisfaction, the normal human being, endowed with a higher intelligence and strong sensibilities, rich in ideas but weak in will power, may in a remote by-way lose himself and drift aimlessly into the dream-land of an erotic paradise, there to lose not only time and strength but the best of his personality and—only too frequently—lose it beyond redemption. If to this be added bitter self-reproaches, mortification and pain at the remembrance of his own conduct which, nevertheless, he feels he cannot alter, above all tormenting fears aroused by baneful literature which irresistibly points to bodily and mental invalidism, not only the sexual neurasthenic develops but, as we have seen in countless instances, at the same time the neurasthenic hypochondriac, the tabophobe, and the paralysophobe. This psychical onanism also directly impairs the health by bringing about "neurasthenic impotence"; the subject is always dissatisfied, constantly seeking new experiences, and in choosing and basing his conceptions upon objects independent of time and space he finds sensations such as actual life can never furnish, or only in the poorest and most artificial form. All lawful pleasures and enjoyments which these realities can offer, or which are readily furnished, he accepts with feelings of unspeakable loathing, esteeming them lightly or hating them. A constantly increasing repugnance to legitimate marital and normal sexual intercourse, in fact to any kind, and a hungry seeking after sensations and perversities which he has not yet enjoyed or pleasures which have not yet been his ensues. The *physical* practice of masturbation, no matter in what form, does not as a rule lead to neurasthenic impotence (on the contrary, its injurious effect in this as well as in other directions is still greatly exaggerated), but the inseparable *psychical* reaction, the influence upon the character and the temperament, the consequent relaxation and depression which may be increased to hypochondriasis; in other cases the *original psychical* coaction combined with the exaltation of the imagination laboring in a pathologic direction, the fulfilment of the conception of a dream world filled with erotic pictures and scenes as remote from those of ordinary life as Heaven is from Hell, and the sovereign preponderance of dream creations compared with the impossibilities of dust-born reality demonstrates this fully.

Without an intermediate stage of sexual neurasthenia, onanism in its various forms and methods of practice is naturally the origin or immediate cause of many sexual anomalies and perversions (for example, mutual masturbation in patients, as well as also in girls, may favor the development of homosexual perversions). The minute consideration of this subject is remote from the purpose of this article.

On the other hand, I may reiterate that onanism is in many cases not only the cause but in itself the symptom or sequel of sexual neurasthenia which has developed to an extreme degree.

The effects of onanism are not markedly different in the female. If comparatively few sexual neurasthenics develop among the many female masturbators, this is due in the first place to the nature and manner of masturbation in women, the physical and, generally, the psychical reaction also being decidedly less than in men (this cannot here be explained more minutely); also by the circumstance that other neuropathic and abnormal conditions designated by different names, as dyspareunia, vaginismus, sexual hysteria, nymphomania, female sadism, tribadism, etc., not infrequently are produced by this cause.

What is true of onanism is also true of *sexual excesses and aberrations* in general; they may be the causes, or the symptoms and sequels of sexual neurasthenia. Where they are active as etiologic factors there is, as a rule, already a more or less marked neuro-psychopathic basis; nevertheless I have known the typical symptoms of sexual neurasthenia to develop acutely in previously healthy men immediately after very exhausting orgies. A gradual development of sexual neurasthenic symptoms is much more frequently observed in such men than in those who for years practise the *congressus interruptus* with their wives or mistresses. The explanation is obvious; the inhibitive exercise of the will is probably early interrupted, the orgasm never occurring normally, and the damaging effect of this reaction is to be sought in the genital centers of innervation. Cases of this kind in which, by desire of the wife, the *congressus interruptus* is practised year in and year out are becoming more and more frequent among that class of people denominated as of "education and property." Other practices in the realm of this so anxiously desired "facultative sterility" are less deleterious to the male, although these also, for example, the employment of condoms and, to some extent, exclusive pessaries, prevent satisfaction or make it difficult. Certain forms of anomalous intercourse, namely, *fellation* and *irrumation,* are exciting and therefore exhausting. The latter is little practised in Germany, and hence less dangerous than fellation which may be practised immoderately by experts in the art of love, and conceals a "snake among the roses." All of these habits may sometimes produce merely impotence, at other times general neurasthenia with a sexual coloration, at other times pathologic fear or a suggestion of impotence is firmly rooted in the consciousness—a condition which must be distinguished from sexual neurasthenia in a restricted sense as "*sexual hypochondriasis,*" although the distinction in individual cases is often quite difficult.

Early acquired (or congenital?) homosexual tendencies and habits may only lead to sexual neurasthenia—but then very speedily—when such individuals in opposition to their natures, for conventional reasons, at the urging

of relatives, etc., consent to marriage—either thoughtlessly or to be obliging, with the hackneyed self-comfort that all will be right. But all is not right; on the contrary, everything is wrong; and then may follow bitter words of scorn such as the old cynic Martial [1] hurled at a candidate for marriage of homosexual tendencies who was dragged to the altar:

> "Heu quantos æstus, quantos patiere labores,
> Si fuerit cunnus res peregrina tibi."

In these cases extremely stubborn forms of psychogenous impotence are prone to develop and are exceedingly difficult to cure, also general psychical depression and neurasthenic hypochondriasis. Almost as bad are those cases of young men in whom an ultra *ascetic* or *too pietistic* education has suppressed or completely arrested every impulse belonging to sexual life, and who are then without the slightest instruction or experience launched carelessly into marriage. These cases in which there is a repugnance to the sexual act originating in exaggerated ethical and religious ideas, yet the performance of which in the nature of things is felt to be a duty, may by the combination of severest pangs of conscience and strongest copulative forces seem serio-comic to outsiders. But they may also have an unexpectedly tragic termination. I saw an instructive and lamentable case of this kind a few years ago in a count, aged 30, who, after being married for two years, committed suicide.

On the other hand it would be considered a fairy tale—a fairy tale gladly heard and repeated—to state that *sexual abstinence in itself* is capable of producing sexual neurasthenia; in such cases we can only consider the influence of sexual abstinence when there are other complicating factors, neuropsychopathic predisposition, faulty education and mode of life, a tendency to psychical onanism, etc., and when actual sexual life is refrained from or feared for other motives. In these exceptional cases the conditions may be similar to those previously mentioned. Finally, we can hardly assume that the *excessive use of alcohol and tobacco* is the sole cause of the development of sexual neurasthenia. Complicating factors must be added, although we cannot deny that these deleterious agents have a highly predisposing effect. In some cases of presumable impotence which I observed in men of middle age who drank little but were much addicted to cigarette smoking, it appeared on the whole to be due to a diminution of libido or even its complete disappearance rather than to impotentia coeundi in the usual sense.

PROPHYLAXIS

The prophylaxis of sexual neurasthenia of course is to a great extent the prevention of neurasthenia in general, but in part it belongs to the realm of *sexual hygiene*, a branch of public and individual well-being which, for many reasons, is still neglected and has not been generally recognized as important. Many prejudices confront us in this realm which necessitate delicate consideration and handling, and the free and unrestricted discussion of these conditions is almost prohibited in the public forum, and, in fact,

[1] Book XI, 78.

by all classes of people. Prudery, which is not really indigenous to the German blood, has been artificially inoculated to the detriment of naturalness of existence, especially in regard to the processes of sexual life, and the censorship of the police has been established also in the realms of literary and artistic productions, even in that of the public press itself; in journals reputed to be of interest to modest readers of both sexes this strict censorship presents insurmountable obstructions to any explanations or attempts at improvement in this direction. To mention one example, typical in its silliness, in an article of mine on "School Nervousness," published by one of the most prominent German belletristic journals, I found to my great astonishment that the word "onanism" had been stricken out. Therefore onanism is not to be mentioned to ears polite, and prostitution and sexual diseases still less so! Under such circumstances how can there be a discussion of sexual hygienic questions and problems? May we not soon perceive that we are practising a thoughtless "head-of-the-ostrich-in-the-sand" policy in thus screening the shame of the people which proliferates fearfully in secret rather than uncover it to the full, healing light of publicity? Perhaps the "*German Society for the Repression of Sexual Diseases*," just formed, may gradually bring about an improvement in this matter.

This is not the place in which to discuss the general corporeal and psychical tonic measures necessary to prevent neurasthenia, particularly in those congenitally predisposed and neurophathically tainted. The special prophylaxis of sexual neurasthenia necessitates, above all, *educational influences* so that, on the one hand, sexual excitement in the growing youth shall be controlled and kept within bounds, and, on the other hand, that he shall receive at the right time and in a proper way the instruction which the young need. As to the exact mode in which this knowledge shall be imparted, general rules cannot be formulated; this must be left to the tact of the parents or other members of the family, to the teacher, or to the professional adviser who should attempt in each individual case to acquire an influence over the youth in question, and thus gain control of his psychical life. The worst breeding places for onanism are schools, pensions, and similar institutions (military schools had formerly a particularly unenviable reputation in this respect, perhaps not without justice). What the school may do for the prevention of onanism, Hermann Cohn[1] has stated in a small book which at the same time reveals the disastrous frequency of school onanism. He demands, first of all, that the pupils be watched by the teacher during the hours of instruction as well as during the intermissions for the prevention of mutual onanism—a requirement the fulfilment of which may unquestionably be greatly facilitated by an improved arrangement of class-rooms, particularly by a more suitable position of the school desks, as well as by the prevention of the present over-crowding of most school-rooms. Cohn insists, too, that the teacher should warn the pupils of the injurious effects of auto-onanism as well as of mutual onanism, and that by means of lectures and pamphlets parents and guardians should have their attention called to the duty of teaching children the dangers of masturbation. As is obvious from

[1] Hermann Cohn, "Was kann die Schule gegen die Masturbation der Schulkinder thun?" Berlin, Richard Schoetz, 1894.

the last statement, the school should not be expected to do all; on the contrary, parents and family, to a much greater extent than has heretofore been the case, should remember their duties in this respect. Children who are inclined to onanism—the tendency often betrays itself very early, usually at the fifth or sixth year—must be carefully guarded both while awake and asleep, and, above all, exciting agents and evil companionship must be avoided; in boarding-schools, etc., common sleeping rooms should be strictly and most carefully watched. The most severe punishment should be inflicted on the one who starts mutual onanism; if necessary, expulsion from school. In auto-onanists—male and female—it should be carefully noted whether local irritation is present (among other factors in both sexes, oxyuris); this, however, is rarely a cause. A hygienic mode of life as to clothing, sleep and nutrition (the blandest diet, avoiding everything stimulating, especially alcohol); systematic exercise even to extreme fatigue most effectively combats the morbid desire for masturbation.

Another factor in prophylaxis consists in the prevention of *venereal diseases,* above all, in the prevention of *gonorrheal infection,* which we have learned to recognize as one of the most important and most widely distributed etiologic factors of sexual and subsequently general neurasthenia, especially of its most severe and most dangerous forms and sequels. What may be here accomplished is evidently not to be found alone in useful advice concerning personal protective measures (employment of the drop apparatus after Blokusewski and similar ones) and thereafter the earliest possible (*abortive*) treatment of acquired gonorrhea by antiseptic injections which are readily employed. Far more vital and important is the instruction, particularly of boys, as to the dangers of illegitimate sexual intercourse in general, above all of intercourse with prostitutes; this has, for example, been attempted by a society of physicians in St. Petersburg,[1] and also recently in an article from the pen of Prof. A. Herzen[2] addressed by German teachers to the students in the high schools. Much can still be done in this direction; the difficulties mentioned at the beginning of this chapter must be gradually overcome by gravity and patience. The belief still prevalent among the young as to the dangers of sexual abstinence must be combated by scientific reasoning. Alcohol, which stimulates sexually, particularly in the ridiculous forms in which it is used by students in their drinking bouts, must be strenuously tabooed. Whether an *early marriage* or marriage at all is advisable depends upon many and varied circumstances; very generally as it is done, I by no means advise marriage as a "prophylactic measure" for neurasthenically predisposed individuals. Experience proves that marriage does not protect from onanism, does not prevent either temporarily or permanently the overpowering and long-continued desire for self-satisfaction by onanism, particularly for "psychical onanism," because for the predisposed individual marital pleasures are uniform and soon exhausted, while the pleasures of

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[1] "Zur Frage der sexuellen Hygiene." Vorträge, gehalten in der Sitzung des deutschen ärztlichen Vereins am 3. Mai, 1899, von Dr. Ernst Hörschelmann, Ernst Blessig, Ernst Masing, *Petersburger med. Wochenschrift,* 1839–41, 1899.

[2] A. Herzen, "Wissenschaft und Sittlichkeit, ein Wort an die männliche Jugend." Vortrag, gehalten in Lausanne und Genf. Autorisirte Uebersetzung. Verlag des "Vereines Jugendschutz," Berlin C 2, 1901. (Vorwort von Adolf Harnack.)

imagination present alluring forms and unceasing variety; of course these individuals are not restrained either by Church or State from practising new refinements and seeking illegitimate intercourse. Moreover, the neuropathically predisposed person lacks to a great extent the psychical quality of adaptation, the faculty of resigning his own to another individuality, even the degree of forbearance and abnegation so necessary for a happy or even tolerable existence in the married state. Only after carefully considering the conditions upon both sides and the character of the parties should a neuropathically predisposed person be permitted to marry, but marriage should never be lauded and extolled as, *quand même,* an active preventive.

TREATMENT

Sexual neurasthenia admits of a uniform and specific treatment as little as it admits of a uniform prognosis; on the contrary, we must be content in the individual case with the apparent causes, with the relation to general neurasthenia, with the special symptomatic indications.

The treatment should be based upon the etiologic factors embraced in the preceding description, among the most important and most frequent of which are *local diseases of the genital apparatus (particularly gonorrhea in men), onanism,* the abuse of *coitus,* and *sexual perversions.*

In regard to the first factor, *existing local diseases,* we must beware of *over-estimating* the value of *local therapeutic remedies and agents* at the onset. The statement, usually cautiously made during the treatment, "cessante causa, cessat effectus," may here be generally regarded as even less applicable, since no immediate causal relation between the local genital affection and sexual (and general) neurasthenia need by any means exist. On the contrary, as we have seen, the local affection is often merely an "*accidental cause*" which, acting upon a predisposed nervous system, produces the neurasthenic disturbances, giving them special form and color, but does not actually produce them. The condition of irritable weakness in the nervous tracts and centers, nourished and maintained by local effect, may, therefore, after the local effects have disappeared or been removed, continue substantively and undiminished. These considerations should lead us to estimate with cool criticism the importance of local therapeutic treatment in sexual neurasthenia (also in the hysterical form); we should refrain from all superfluous or doubtful procedures, but proper local measures with a definite purpose should not be neglected or held in abeyance. It is sometimes very difficult to draw the line between "too much" and "too little," and in doubtful cases we should rather incline toward the side of the harmless "too little." If examination reveals the existence of gross structural changes in the male urogenital apparatus—especially chronic gonorrhea and residual changes in the urethra, bladder and prostate gland, which form by far the most frequent local affections leading to sexual neurasthenia, appropriate treatment is necessary. What can be done here—aside from the symptomatic treatment to be later described—belongs chiefly to the realm of the specialist, such as the treatment of gonorrheal and non-gonorrheal posterior urethritis, cystitis, prostatitis, urethral strictures, and hypertrophies of the prostate gland. In rarer cases diseases of the penis, phimoses, prepucial concretions,

balanitis and the like are the cause, and local measures (operation for phimosis, circumcision for a moderately long and hypertrophied prepuce, etc.) are indicated. The same views as to treatment are applicable in the sexual neurasthenia of the female, in so far as local genital diseases (caused by irritation of the vulva in masturbation, vaginal and cervical catarrhs, displacements, etc.) are, in the individual case, causes of disease or factors which increase irritation. On the whole, the greatest care in, sometimes even the avoidance or neglect of, active local treatment is advisable, since it is difficult to foresee to what extent this excessive irritability with its reflex and resultant psychical effects may be due to such treatment, or what undesirable changes in the general condition or the individual symptoms may be produced thereby. At all events it is wiser in cases of this kind to try to relieve the neurasthenia by general treatment, and to combat by palliatives the local effects which are not so urgent.

Instead of the preventive measures which have been described under prophylaxis, an attempt has been made to treat *onanism* partly by mechanical and partly by physical and suggestive means; both, as is well known, being productive of but slight results. Some of the innumerable, mechanical, protective appliances which have been so greatly lauded by their inventors may be regarded as useless toys, sometimes very uncomfortable, and only exceptionally beneficial or protective. The relief of local, irritative conditions by suitable measures, if necessary even by slight operations (circumcision, cauterization of the male urethra and of the vulva), is permanent only in isolated cases. "Suggestive therapy," here as well as in many other diseases, has not fully met our expectations; the results are usually slight or temporary, often wholly lacking; nevertheless in severe cases this method may at least be tried—also for the sexual anomalies and perversions which are rarely etiologic factors. In other cases, particularly in impressionable persons and those whose intellects are of high order, hypnotism is often unnecessary; here occasionally the counsel of the physician, the appeal to reason and conscience, the awakening of the dormant powers of the will, the stimulation of the moral energy, while at the same time allaying the fears as to the presumable consequences of onanism which are often painted as dark as possible and declared to be insurmountable, will slowly but surely produce the desired result.

The abuse of coitus, particularly the congressus interruptus which aims to prevent conception and of course necessitates its avoidance, perhaps the substitution of other anticonceptional processes which have lately been advocated in increasing numbers, and no single one of which actually prevents the condition, should be discountenanced.

For the *general treatment* of neurasthenia, fundamental laws cannot be established nor minute directions be given in this article. One point only must be emphasized. It is well known that in many neurasthenics who come under treatment we attach great importance to the benefits to be derived from prolonged rest in bed with simultaneous hypernutrition and the methodical stimulation of muscular activity by massage and electricity. In the majority of sexual neurasthenics this mode of treatment is rarely indicated; on the contrary, from the beginning active muscular exercise to the point of fatigue by means of suitable gymnastics or sport, or, in fact, any bodily or mental

exercise that they prefer, is more beneficial. Here we note simultaneously the salutary effect of psychical influence. It is necessary to remove the thoughts as far as possible from the sexual sphere, and concentrate them elsewhere; we must impress upon the minds of these patients, and steadily maintain, that man has higher and more important functions in life than those connected with the genital system; we must endeavor to awaken interest in other things, and to provide them with work adapted to their powers and inclinations. Change of residence and climate, prolonged journeys upon land and sea, the new impressions which they receive and the new train of thought which follows the changed mode of life often assist greatly. Under all circumstances the patient must be made to understand that he not only will recover, but also will most rapidly recover by a mode of life which conforms to all the requirements of rational hygiene, and which at the same time necessitates a strict and energetic self-denial—for example, in many cases sexual abstinence as well as the avoidance of smoking and drinking and other injurious habits of life. This psychical pedagogic influence, with the simultaneous strengthening of the body and mind, and the most careful avoidance of all threatening and baneful factors, is the only one which, in cases at all curable, is sure to yield satisfactory results, not merely transitory or apparent results—while in these cases local and symptomatic treatment is often merely an auxiliary measure, rarely a necessity, the exaggerated use of which prevents or warps the cure.

Nevertheless, some of the symptoms of sexual neurasthenia are in themselves of such great importance, and may produce such an intense reaction upon the entire psychical life of the patient that, in many cases, we cannot refrain from considering these and attempting to combat them with all the remedies at our command. These are certain, so-called, "*irritative symptoms,*" *frequent emissions, spermatorrhea* and *prostatorrhea, frequent erections* (*priapism*) on the one hand, and "*debilitative symptoms,*" the *neurasthenic forms of impotence,* on the other hand—although such a contrast as we have seen does not exist pathogenetically, and according to experience we are often in a position to treat simultaneously the symptoms of both forms; for example, frequent emissions and a decrease in sexual power, and our therapeutic indications must be upon this basis.

In the symptomatic treatment of these conditions—as well as in the general treatment of neurasthenia—*physical* curative measures, hydrotherapy, balneotherapy, electrotherapy, and kinesitherapy, and regulation of the diet and the mode of life are of paramount importance, while *drug* treatment, except for a few remedies, is less valuable.

The exceedingly numerous ways in which *hydrotherapy* may be utilized for thermic irritation also permit its varied employment in the conditions we are here discussing. Given a rational indication for hydrotherapeutic measures, we must proceed on the assumption that the fundamental activities are generally lessened by cold and increased by heat; this "law" does not, of course, embrace the very brief application of cold by which sensory as well as motor functions are frequently increased, and which acts favorably on exhaustion; on the other hand, moderately warm temperature has a quieting and anodyne effect. As a rule, considering these fundamental views, in cases of painful irritation, in dysesthesia and neuralgia, in dysuria and

strangury, in irritative secretory anomalies, or when emissions and priapism are frequent, we preferably employ warm water (35° to 32° C.) and rarely long continued applications of cold water; while, with symptoms of debility, especially in the neurasthenic forms of impotence, stimulating procedures such as alternate cold and hot applications are especially to be recommended. This indication is met by the very common use of sitz baths. When we are seeking to relieve pain and irritative phenomena, *protracted, moderately warm* sitz baths (once or twice daily) are most suitable, and may be continued from a quarter to one-half or even three-quarters of an hour; in very sensitive patients we gradually include irritation by heat. The same is also true of irrigations, of compresses, and of applications of dry heat. Where, on the contrary, we wish a stimulating effect, we may employ once a day either very quick cold or hot sitz baths, at a temperature of 40° C., from fifteen to thirty minutes. Flowing sitz baths, as well as sitz baths with an ascending douche and massage baths, may also be here considered. Two forms of local employment of hydrotherapy are by means of Winternitz's *psychrophore* and Chapman's *back coil* (as well as similar apparatus) which are much used. I employ the "psychrophore," as a rule, in the irritative conditions previously mentioned, beginning with a temperature of from 30° to 25° C., gradually reducing it to 20° C. or lower, and simultaneously decreasing the time of application from 15 to 10 or even 5 minutes; this application is generally made every other day; but with very sensitive persons only twice a week. In cases of impotence hot water, 40° to 35° C., is applied with the psychrophore for fifteen minutes. As Chapman's coils are kept on the body a longer time and gradually become warm, they also may be used in irritative conditions at a reduced temperature (20° to 15° C.). After from one-half to three-quarters of an hour the water must be changed. These coils may be used while the patient is walking about, but when he is in the recumbent posture the pliable *metal coils of aluminum*, which are manufactured by different firms as coolers for the back, for the abdomen, for the penis, and for the scrotum, are preferable. For long continued application of heat the well known thermophore compresses, as well as electrothermic compresses, etc., may be used.

Among the curative agents of *balneotherapy* in a restricted sense, the *acratotherms* of moderately warm temperature (Schlangenbad, Wildbad, etc.) as anodyne and quieting baths, and those which increase heat (Bormio, Gastein, Teplitz-Schönau, Ragaz-Pfäfers, Plombières, etc.) may also be employed as stimulating remedies in sexual weakness. In the latter case the ultimate effect may be decidedly increased by the altitude of the spa in question (Bormio, Gastein) as well as by combining the baths with mechanical irritation, douches or massage. *Sea baths, thermal salt baths containing carbonic acid* (Oeynhausen, Nauheim), and *iron baths containing carbonic acid* (Franzenbad, Marienbad, Driburg, Pyrmont, Cudowa, etc.) have the same effect. According to reliable observers an immediately stimulating sexual effect follows *carbonic acid baths and gas douches*, a fact which should receive greater consideration than has been accorded it by physicians or even, as a rule, by the management of spas (Franzenbad and Marienbad furnish excellent examples of facilities of this kind).

Electro-therapy in the usual forms (hydro-electric baths, general frank-

linization and arsonvalization, electro-magnetic therapy) may be resorted to for its anodyne effect on sensory conditions—as a local application, however, it should be employed with the greatest care and only by specialists. In this realm of disease the thoughtless, haphazard application of the galvanic or faradic current, often without reliable apparatus or with one which perhaps does not record the exact force of the current, by physicians and of course still more often by quacks, is an actual crime, and not only is electro-therapy thereby greatly discredited, but, what is worse, the patient is often seriously injured. For reasons previously stated a local treatment which does too much is, in the majority of cases such as we are considering, generally detrimental. I do not favor the frequent attempts at intraurethral as well as intrarectal galvanization and faradization. All of these processes—and also local massage which will soon be discussed—are well calculated to generate or to maintain a local functional irritation and, at all events, they direct the attention of the patient too much to the local affection, and prevent a soothing psychical influence or, at least, render it very difficult. From the standpoint which should be maintained in the neurasthenic forms of impotence, all local remedies, especially those which to the laity seem to be quieting, electric, local procedures, prove to be unpleasant and often painful. Naturally there are exceptional cases—individuals for whom a somewhat energetic local treatment is indispensable for its suggestive effect—but even in such cases I would urge the greatest care in the employment of intraurethral faradization and galvanization, and that *galvanization be limited to the percutaneous* (the cathode in the form of large pliable lead-plate electrodes placed upon the vertebral column, the anode as Hirschmann's scrotal electrode, or as a pliable plate of about 50 ccm., in the perineal region; where there are symptoms of irritation the stabile current, inclusion and exclusion; in debility these should be carefully and alternately introduced at the conclusion of a sitting which should last from five to seven minutes). Where there is a large static machine, the employment of spark currents (currents from the external cover of the Leyden bottle or condensor plates) with a large flat condensor electrode along the vertebral column and a foot plate, this may be used for several minutes daily. If occasionally for special reasons we desire to employ intraurethral electrization with the constant current the greatest care is necessary. The urethral electrode (the best is in the form of a Nélaton's bougie, well sterilized) is to be introduced within the prostatic urethra and, to lessen the local irritation, should always be combined with a positive pole while the cathode as a large, flexible, lead plate is placed upon the perineum, in the gluteal fold, or upon the sacrum. Inclusion and exclusion is with the rheostat, the current to be feeble (not more than 1 to 1.5 milliampères), producing merely a faint local sensation; the current is to be continued from one or two to four or five minutes, and the duration gradually increased; the application should not be oftener than two or three times a week. Faradization through the urethra and rectum—the latter for the production of reflex stimulation, analogous to the treatment for enuresis—is less difficult to carry out, but, as may be assumed, is always of questionable value, or certainly has but temporary effect.

Kinesitherapy (or, as some designate it, "mechano-therapy")—*gymnastic exercises and massage*—for general strengthening is exceedingly valuable in neurasthenics; its local employment, particularly massage, is subject to the

same criticism as electric local treatment. That not only the abdominal muscles but also the muscles of the floor of the pelvis—especially the levator ani—may be affected by systematic gymnastics is not a new principle although it has lately been again promulgated by Thure Brandt (for massage treatment in gynecology). Tapping the lumbar region, pressure over and shock to the hypogastric plexus and the pudendic plexus, are old and well tested Swedish movements—they were employed and taught in my father's Gymnastic Institute fifty years ago—although modern masseurs are continually rediscovering and claiming them as the progeny of their own fertile brains. Instead of manual resistance, gymnastics, apparatus gymnastics (Zander) have lately been employed but without benefit, and the manual practice of shock massage has been substituted for and often supplanted by stationary and portable apparatus, vibrators with hand or foot motor, electro-motor, or carbonic acid motor, etc.[1] O. Preiss, in Elgersburg, has introduced "*massage baths*"[2] in which the effect of baths and massage is combined; the bath itself gives the massage, the water being set in motion by a sufficiently strong power (pressure of at least three atmospheres) after the patient is so fastened by a suitable apparatus that the water may act upon a desired part of the body. Preiss states that with massage baths given on this principle he has seen favorable results in *sexual weakness with emissions.* During the bath the patient was in the recumbent posture; the lumbar portion of the spinal cord and the region of the perineum were treated while he stood in the tub; the temperature was either cool or lukewarm (18° to 24° C.). In youthful masturbators of both sexes these baths have proven of decided value.

In the treatment of emissions, spermatorrhea, and prostatorrhea, as well as in impotentia coeundi, Zabludowski has lately employed a system of massage of the genital region combined with massage of other parts of the body, therefore more general massage.[3] According to Zabludowski these manipulations affect the genital apparatus and its adnexa, as well as the spinal cord, by way of the reflexes and radiation, the skin of the body, the muscles, the blood and lymph vessels of the entire body, finally, by stimulating conceptions in the cerebral centers. A definite series of effects is produced; the patient is manipulated in the recumbent posture, the right and left lateral positions, and the knee-elbow position, the patient lying upon the belly, and, if necessary, resuming the recumbent posture. This massage, with one or two treatments daily, should be continued, upon the average, for six to eight weeks. In sexual neurasthenia I do not regard this method as perfectly harmless, and great care should be exercised in the choice of cases, since, as a rule, for reasons previously mentioned, its use in masturbators and youthful patients with emissions, in fact, where irritative symptoms are present, is not advisable. In some cases of neurasthenic impotence no benefit has followed its prolonged use, while marked improvement was promptly brought about by other remedies (for example, by injections of yohimbin).

In the *diet* of sexual neurasthenics, just as with neurasthenics in general,

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[1] Vergl. J. Zabludowski, "Technik der Massage." Leipzig, Georg Thieme, 1901.

[2] O. Preiss, "Massagebäde." *Zeitschrift für diätetische und physikalische Therapie,* 1900, 1901, Bd, Bd. IV, Heft 3.

[3] Zabludowski, "Zur Therapie der Impotentia virilis." *Zeitschrift für diätetische und physikalische Therapie,* 1899, Bd. III, Heft 4.

all alcoholic drinks must be absolutely prohibited and foods containing alkaloids (coffee, tea, cocoa), as well as tobacco, must be decidedly limited. In the majority of cases total abstinence from stimulants, alcoholic as well as non-alcoholic, cannot be enforced nor is it always desirable, because the beneficial stimulation of the central nervous system, particularly the heart and vasomotors, by its careful use, cannot be entirely dispensed with. Much is accomplished if we induce the patient to give up alcoholic drinks, to limit the use of tobacco and, instead of strong coffee, to drink weak tea, cocoa, or substitutes for coffee (malt coffee). Spices also must be limited; in young neurasthenics especially, as well as adolescents in general, spiced food frequently does harm and produces severe nervous disturbances during the period of the body's development. What is true of most vegetable spices is also true of the essential constituents of meat broth, the *extractive products of meat* (the so-called meat stock); therefore "strong bouillon," which is recommended over and over again as a presumably strengthening remedy, is for these cases decidedly objectionable. The same is also true of beef tea and the like prepared at home, for although they have a high nutritive value on account of dissolved proteids which they contain, they also have the same stimulating effect as ordinary meat broth. Since in nervous persons in general, the ingestion of *meat* is to be limited, and although strict vegetarianism is not to be advised, the albumin requirement of the organism should chiefly be satisfied by vegetables combined with animal products (milk, butter, cheese, eggs). For supper, at least, no meat should be taken under any circumstances; the amount of food eaten should be small, and the food should be easily digestible. It should be eaten early, and afterward little or no fluid should be drunk (least of all beer, but preferably small quantities of carbonated table water, or grape and other fruit juices free from alcohol).

It may appear to be a contradiction of the foregoing when I say that, from most remote times, some foods, especially delicacies and spices, have been lauded as presumable *aphrodisiacs*, the belief being that they increased sexual desire. This was believed—partly because of the odor which characterized them—of animal and vegetable foods such as game, fish, caviar, oysters, truffles, celery, asparagus, chestnuts and the like, and still more so of strong spices (pepper, cloves, nutmeg, vanilla, etc.) and the more powerful alcoholic liquors. But the presumable aphrodisiac effect of these substances is just as chimerical as that of most aphrodisiac drugs; and, if they have any effect, it consists in stimulation and congestion, chiefly of the urogenital organs, and is therefore not harmless.

In patients with emissions, the "hygiene of sleep" must be carefully observed. In addition to the restriction of solid and fluid food in the evening, we must endeavor to bring about free and regular bowel evacuations before going to bed; habit has a great influence, but this must be assisted by suitable laxatives taken during the day. Patients must not go to bed earlier than two and a half to three hours after supper, they should amuse themselves by play or light reading, not too exciting, or by a short walk; the bed should be hard and cool, the night clothing and bed-covering light; therefore, no feather beds nor heavy blankets, and no large, soft, and heating pillows. Lying on the back during sleep should be avoided; the legs should be propped high; if emissions have frequently occurred in the early morning hours the

patient should rise at proper times during the night (either by auto-suggestion or the aid of an alarm clock) to relieve the bladder.

In *drug treatment* we consider the so-called anaphrodisiacs, i. e., those remedies which diminish sexual desire, also those which increase it, the aphrodisiacs, although the benefit to be derived from either is very doubtful. The anaphrodisiacs are especially recommended to combat the tendency to masturbation as well as to correct the frequent pollutions and erections, priapism, etc. The *preparations* of *bromin* and camphor are usually employed for this purpose; and *monobromate of camphor,* which is a combination of or substitute for camphor, is often used internally, 0.1–0.5 in capsule or pill at evening or several times during the day. Exceptionally sedatives and tonics (chloral hydrate and digitalis) are useful. Lupulin (glandulæ lupuli), which contains an ethereal oil and the bitter product of hops, probably has no anaphrodisiac action, certainly it is very unstable and uncertain (dose 0.3–0.6 in powder or pill).

Among the *aphrodisiacs, cantharides* or Spanish flies (lytta vesicatoria) is first mentioned, the action depending upon the cantharidin (up to .5 per cent.) which they contain; but, on account of the extremely severe and, in many cases, dangerous irritation of the urinary passages which they cause, preparations of cantharides are rarely employed by physicians for this purpose; they are much used, however, in various love potions, bonbons, and pastilles (Diavolini, pastilles galantes) which are supposed to have an erotic effect, and which are still employed in Southern countries as they were in the time of Casanova and de Sade. Various kinds of beetle act similarly to cantharides, especially the melœ varieties, among which the *May worm* (melœ majalis) probably has this action because of the amount of cantharides it contains. The various resins and balsams, myrrh, galbanum, balsam of Peru, vanilla and crocus are illusions as far as their aphrodisiac effects are concerned, as is also probably true of phosphorus, including the nerve tonic, of the glycerin phosphoric acid preparation (lecithin) recently so much praised, and of *arsenic.*

The effect of strychnin and of drugs containing strychnin is better, but they are undoubtedly dangerous, and must be given in comparatively small doses. Lately we have found a substitute in an alkaloid of the same group, and it appears to be the only effective drug aphrodisiac; namely, *yohimbin* (Spiegel). This is prepared from the rind of the West African yohimbin —also from the similar but weaker yohimbenin—and was isolated by Spiegel in the form of white crystalline needles; the alkaloid injected experimentally in rabbits and mice, and especially in dogs, produces enlargement and hyperemia of the sexual organs (the testicles and epididymis) with very marked erections. Given in larger doses it produces general spasms, and death from respiratory paralysis. For therapeutic purposes yohimbin tablets (each containing 0.005) are given internally two to four times daily, or a solution is prepared by dissolving the drug in 100 parts of distilled boiling water (10 drops of this solution correspond to one tablet; therefore 1.0 to 2.0 in solution should be given in the course of a day).

I have used these yohimbin preparations in numerous cases of neurasthenic impotence in the last two years, and in general am well satisfied with the results. The principal effect is that they cause erection, generally after

using the remedy for one or several days, and thus make coitus again possible after it had been impossible for years; there are no secondary symptoms. In the first case in which I used it, a man about fifty years of age, owing to the absence of erections, had been unable for *twelve years* to practise coitus, but he reported in a few days that erections had reappeared and he was now able to perform this function. We cannot always count upon such brilliant results. In severe cases in which the remedy has failed to have any effect after long internal use, I have lately resorted to *subcutaneous injections*, and have, at least in cases of neurasthenic impotence, seen much benefit; for this purpose I use a *two per cent.* solution. The solution must be kept in a dark bottle as it is slowly decomposed by light and changed to a yellow color. A drop of chloroform assists in preserving the fluid. It is best to order only small quantities at a time, sufficient for from ten to twenty injections. The prescription is about as follows:

℞ Yohimbini Spiegel 0.2
solve in
Aq. dest. sterilisat 10.0
D. ad vitr. fusc.
M. D. S.: 2% yohimbin solution for subcutaneous injection (dose 0.5–1.0).

I begin with an injection of one-half the contents of a one gram syringe (= 0.01 yohimbin); this solution is injected upon the inner surface of the thigh, and if no effect is produced or if it is insufficient I increase the dose to 1.0; I have never had occasion to go beyond this. At first the injections are given daily, but after they have taken effect only every second or third day or even at longer intervals, and after about twenty injections have been given the drug should be discontinued for a considerable time. I have never seen any disagreeable secondary effects; in isolated cases (3, so far) a temporary chilliness and outbreak of sweat with weakness appeared after the first injection, but not subsequently. The effect, which is to stimulate erection, slowly appears; as a rule, in the morning after an injection on the preceding afternoon or evening.

Although yohimbin can by no means be regarded as a *specific for impotence* I believe that, at least in the treatment of neurasthenic impotence, it renders all other drug treatment superfluous. This is true not only of the previously mentioned "aphrodisiacs" but also of the various forms of *organotherapy*, of treatment with Brown-Séquard's *testicular fluid* ("séquardine," liqueur testiculaire) which is usually diluted with an equal quantity of sterilized water, 2.0–6.0 and subcutaneously injected, also of its substitutes for internal administration, the "tabloids" of *didymin* (one or two three times daily) and similar preparations. The *sperminum Poehl*, which also belongs to this category, is an organic product of metabolism, undoubtedly valuable, which may be employed in neurasthenia as a general tonic, but is not a specific for impotence. It is generally used subcutaneously (a one or two per cent. solution kept in a sterilized bottle), but is also administered internally (as "essentia spermini," a 4 per cent. solution, from 25 to 30 drops to be given in warm, alkaline, mineral water).

In conclusion we must refer to attempts which have been made to cause filling of the corpora cavernosa mechanically by the local use of instruments on the glans penis, thereby causing an erection and orgasm. The number of "Venus-rings" employed for this purpose, also instruments bearing similar names which are often advertised in the newspapers as harmless (sometimes as "rubber articles"), is quite large. Physicians will not advise the use of any such instruments.

If the preceding description seems incomplete, if some points have not been discussed, it must be remembered that we are not dealing with the treatment of impotence as such, but only as one of the symptoms of sexual neurasthenia, and that here and there we have digressed somewhat beyond this limit. In conclusion I must reiterate: All local therapeutic and symptomatic endeavors, important as they may appear to be in the individual case, must be subordinated to a psychical, pedagogic influence—or effect this as auxiliaries—the cure depending chiefly upon a *quieting effect, upon the strengthening of the will power, upon diverting the mind from the sexual sphere, upon stimulating the will to combat individual tendencies, and upon the stimulation of the mental and bodily energies by compensating exercise and activity.* Only when this treatment is strictly adhered to will it be possible, with all our professional knowledge and experience, for us to bring about actual and *permanent cures,* not *apparent successes* such as are achieved by professional and non-professional charlatans, wonder-workers, quacks, hypnotists, magnetizers and Christian Scientists.

EPILEPSY

By W. VORKASTNER, Berlin

HISTORY

Epilepsy, or better so as not to prejudice the case, the epileptic attack was known to the ancient world; it has always been the subject of professional interest and constantly stimulated medical investigation to a high degree. The epileptic attack produces one of the most striking clinical pictures. This impression is reflected in a characteristic manner by the phantastic relations with which the underlying condition producing the epileptic attack has been surrounded. The best known designation perhaps is *morbus sacer.* This probably originated from the unsophisticated observation of the frequent delirium with ecstatic or religious coloring or even from the fact that persons of high mental development and of great prominence quite often are subjects of epilepsy. Celebrated historical epileptics are mentioned in all text-books and it is unnecessary to refer to them here. The belief in similar mysterious relations is betrayed by designations such as *morbus dæmonicus,* or the term which indicates a connection with the constellations, *morbus astralis* or *morbus lunaticus.*

At the present time we have emancipated ourselves from views which referred the origin of epilepsy to the ominous play of supernatural powers. Nevertheless, and in spite of eager investigation for centuries, the final and intimate nature of this remarkable disease has not been completely unveiled. As we shall see, later on, it has been possible experimentally to produce this or that isolated component of the epileptic attack in animals; it is true the microscope has revealed minute changes in the epileptic brain; more than once we have thought to have neared the goal, but prosaic criticism pointed out that a phase of the attack was not the attack itself and that the microscopic alterations were probably not of a primary, etiologic nature, but secondary and a consequence of the prolonged series of attacks. Thus we were finally forced to accept the humbled position, of bestowing upon the great X of the epileptic question a name, and, for this purpose, the term *epileptic change* was coined. That this is nothing more than a paraphrase and a tacit admission of our ignorance need hardly be emphasized. Therefore anyone who attempts, at the present time, to give a description of epilepsy stands by no means upon the firm foundation of positive facts. This existing uncertainty, however, goes still further; it includes the limitation and extent of the conception of epilepsy, and this may in itself be designated as exceedingly indefinite.

Féré holds that epilepsy is only a symptom-complex, that the realm of

so-called *genuine* or *primary epilepsy* will become appreciably more restricted the further we advance in our investigations. This prevalent symptomatic conception is associated with an extremely wide limit of the conception of epilepsy, for, Féré includes quite a number of pathologic conditions which, according to our present views, have nothing in common with epilepsy, such as angina pectoris, Ménière's disease, gastric vertigo, infantile convulsions, and above all Jacksonian epilepsy. Binswanger denies this predominant symptomatic character which would degrade epilepsy to a mere collective term. On the contrary, he adheres to the opinion that epilepsy from its clinical symptoms as well as from its course is a well characterized chronic disease of the nervous system. We shall follow this latter view in our description, for to gain a general survey this is necessary, and besides it is the opinion of the greatest majority of authors and probably deserves the greatest consideration. By this it is not intended, however, that we entirely reject the views expressed by Féré.

A special description is probably required by the adjective *genuine*, by which the great majority of epileptic attacks are characterized, in contrast to the so-called *symptomatic epilepsy*. These 2 terms have been applied in a different sense and may, therefore, readily give rise to errors and misunderstandings. In using these terms the anatomical and etiologic condition has not always been kept in mind but only the clinical. Originally the designation genuine epilepsy was intended to characterize the affection as a functional neurosis, as a neurosis without an anatomical lesion, without a known, definite cause. Symptomatic epilepsy, i. e., epileptic or epileptiform attacks, in which gross anatomical lesions were found which might be regarded as the direct causes of the disease, was contrasted to this. Among the cases that are due to a focal disease of the brain, such as encephalitis, some are characterized clinically and symptomatologically by the fact that during the attack, at least in the beginning of the malady, there are marked admixtures of partial (Jacksonian) epilepsy. In some of the other cases the character of the spasmodic attack and the course throughout correspond to epilepsy but there is no anatomical lesion. Therefore if we consider genuine epilepsy from a purely clinical standpoint, we may say that, in such cases, upon the foundation of a focal disease, genuine epilepsy has developed; perhaps to some a paradoxical manner of expression. The term symptomatic epilepsy has, now and then, been applied to convulsions; these may deserve the term symptomatic, but not that of epilepsy, according to the previous definition (uremic convulsions, convulsions from cerebral pressure, etc.).

GENERAL SYMPTOMS

Following these introductory remarks we shall first describe the symptoms of the individual attack and its varieties, then we shall enter somewhat more minutely into the genesis of the spasm and its accompanying conditions, as well as upon the nature of the hypothetical epileptic change, and, finally, the course of the malady which is characterized by the attacks of spasm, the etiologic factors, the differential diagnosis, and the treatment. (The mental disturbances have been discussed in the Volume treating of Disease of the Mind.)

In many cases the epileptic attack is preceded by certain premonitory symptoms; these are partly of a subjective, partly of an objective, nature. We must further distinguish: prodromes, which may precede the attack for a longer or shorter time—hours or days; then Binswanger's "remote prodromes" and symptoms which immediately precede the attack, while the patient is still fully conscious, these signalizing the approach of an attack. They have been designated "*auræ,*" the term given them by Galen. Some authors confound these two varieties and include the "remote prodrome" with the aura. Clinically, it is true, there is identity between many of the prodromes and the symptoms of aura. We sometimes have the impression that among the prodromes there is, to a certain extent, a protracted or an anticipated aura.

The "remote prodromes" relate principally to the psychic sphere. The underlying feature in the epileptic change in temperament, the abnormal irritability, is often very distinct for some time before the appearance of the attack. The patient resembles an explosive mixture; a spark, an insignificant cause, is sufficient to provoke a severe outbreak of rage; this may be accompanied with an attempt at violence upon those about the patient. Occasionally there is more of mental depression; a heavy load appears to rest upon the patient, a gloomy apprehension of coming evil, of something productive of harm; he is uneasy, restless, and—like an animal suffering from hydrophobia in the *stadium melancholicum*—cringes before his fellows. The description "*of being charged*" has been used, something like the oppressive sultriness of the atmosphere before a storm; in fact the attack in such cases acts like a lysis, like a clearing thunder storm; the patient in contrast to other patients is relieved, as if a heavy load had been taken from him. Sometimes a cheerful, exhilarated condition has been noted. In a few instances a conspicuous increase in the sexual desire has been reported; this was associated with erection and emission; it may appear also without preceding libido as a prodrome and is then included among the somatic symptoms. The *somatic prodromes* consist of a number of complaints which might be readily designated as neurasthenic: lassitude, headache, paresthesia, hypersensitiveness to light and sound, restless sleep interrupted by dreams. One of my patients always noted the approach of an attack by dreaming the night previously of corpses and coffins. Binswanger calls attention to the similarity of some of these prodromes with the symptoms of the stage of incubation in the acute febrile diseases. If we add to these the occurrence of heaviness in the limbs, nausea, vomiting, rushes of heat to the head, and chilliness, the resemblance is even greater. Often there are digestive disturbances (diarrhea). Féré refers the continuous yawning which is sometimes noted in the prodromal stage to the insufficient respiration which was determined by him prior to an epileptic attack. Singultus and sneezing have also been noted. *Vasomotor* prodromes, such as erythema, urticaria, circumscribed swelling of the skin, cyanosis of the hands, as well as transitory cutaneous and mucous membrane symptoms, have been observed. (Case of Féré: a patient had a tickling sensation in the uvula followed by a guttural cough, 24 hours afterward an epileptic attack.) Particularly in childhood short circumscribed or isolated muscular contractions have been seen to precede the attack for some time (Charcot, Ballet).

The **aura** is very much more common than the remote prodromes. Formerly it was regarded as an integral constituent of every attack, but recently the regularity and frequency of its occurrence has been much questioned. Lasègue has been bold enough to state that: "no individual who has an aura is epileptic." In how far this is justified our later considerations will determine. It is a matter of fact that a large number of persons who present clinically the picture of genuine epilepsy have an aura. Delasiauve was the first to classify the various kinds of auræ; he mentions the *sensory*, those of the *special senses*, the motor, and the intellectual auræ; a classification which has maintained itself, at least in principle, up to the present time.

The patient during the aura, as already mentioned, is in possession of his mental faculties which the aura reaches and there, by a process of memory, produces the conception of a coming attack. This conception is often accompanied by intense fear, which—although not often—gives rise to a motor reaction, in the form of an attempt to run away. This forward running associated with fear is psychologic and should not be confounded with the *aura cursativa* which will be mentioned later on. Other patients behave in a different manner, and, with the signal of the aura, they may prepare for the attack by assuming a suitable position. The circumstance is worthy of mention that some patients, even after the aura has appeared, with all of their power attempt to maintain consciousness and thus abort the attack by an act of the will (Oppenheim). An attack need not necessarily follow the aura; corresponding to the condition of consciousness the memory of the aura is well retained by the epileptic; only in cases of retrograde amnesia is it absent.

In proceeding historically in the description of the individual forms of aura we must begin with the *sensory* or *sensitive* aura; for the remarkable feeling of being blown upon, which some epileptics have at the beginning of the attack, has given the entire group the name aura (Galen). More common than this form of sensory aura is paresthesia, or even pain, sometimes of a general nature, often localized. "A peculiar sensation rises from the feet, when it reaches the head, consciousness is lost." Local paresthesia often begins in the distal parts of an extremity advancing proximally. Often in an extremity there is a sensation of numbness which constitutes the aura. The mucous membranes may be the seat of local paresthesia; a patient of Féré suffered from burning in the urethra. It is of importance to state that a *true globus* may be present as an epileptic aura. The very interesting kinesthetic aura must also be included among the sensory ones: the patient has a sensation as if there were a change in position in one of his extremities. Vestibular hallucinations (a feeling of swaying) have also been reported.

An aura of the *special senses* is more common than the sensory sort. Every special sense may be the seat of an aura, even that of smell and taste; those of sight and hearing are, however, most common. There is sudden darkness, the patient loses sight, or he sees sparks, lights and lightning, a red sea of flame. Red plays a great rôle in epileptic hallucinations, as well as in the hallucinations of sight, which are occasionally very complicated. The figures usually have something red about them, a red cap, a red shawl, or the like. I noted a peculiar visual illusion in a patient: his aura consisted in noting that the faces of those about him suddenly became pale and

corpse-like; soon afterward he lost consciousness. The "increase and decrease in size of objects" as an aura, *sensu strictiori*, belongs to motor auræ, as this is evidently due to a change in tonus of the muscles of accommodation. Oppenheim states that this condition is often associated with vertigo. In regard to hearing, the elementary hallucinations consist of groaning, whistling, rushing sounds, which form the aura. The attack is not infrequently ushered in by tinnitus aurium, or a sudden sensation of deafness, in one or both ears. Hearing melodies or akoasmia is rarer. One of my patients after a preliminary attack of tinnitus had a sensation "like the sound of well known voices heard in a dream," but without being able to understand words or appreciate them.

A *motor aura* is much rarer. This consists of isolated clonic contractions or of a tonic spasm of an extremity. This resemblance to Jackson's cortical epilepsy may give rise to errors in diagnosis; we shall return to this in the discussion of the differential diagnosis. Symptoms of absence of function in the form of a paresis or paralysis of an extremity have been reported (Nothnagel). The motor auræ in the form of coördinate voluntary movements (wiping the nose, etc.) or of locomotor movements are very interesting. The *aura cursativa* belongs to this category, consisting in propulsion, with the retention of consciousness and without a psychologic motive. Further among the motor auræ are: singultus, respiratory spasm (inspiratory spasm of Seeligmüller), yawning, coughing, sneezing, and deglutition spasms; the 3 last named often associated with a corresponding sensory aura (tickling in the throat). The occurrence of aphasic speech disturbances must also be mentioned.

Delasiauve's *intellectual aura* had better be called a *psychic aura* for, not only intellectual but mental disturbances appear as auræ. The latter appear as sudden sensations of fear or as an outbreak of anger; more rarely as sensations of happiness or liveliness. The first are shown by absent-mindedness, by sudden inability to follow the train of thought, or the sense of what has been said, or as a constrained conception. A lightning-like review of a large number of memory pictures from the patient's life; or a so-called memory identification counterfeit (a feeling as if the patient had already encountered the same situation) is noted.

The *vasomotor auræ* consist of cardiac palpitation, congestion, outbreaks of sweat, numbness, or cyanosis of the fingers and toes, erythema of the skin. It must be remembered that a number of these vasomotor phenomena are secondary, due to fear of a coming attack. This is especially the case if, as is not uncommon, these vasomotor symptoms are associated with some other form of aura. In fact it may be stated at once that combinations of various kinds of auræ occur. The *visceral auræ* may be divided into the sensory, and into that form which passes to the special senses. The visceral sensory auræ consist of nausea, vomiting, a sensation rising from the stomach; the motor visceral aura, of rumbling in the intestines, acts of vomiting, etc.; strangury and tenesmus may be a visceral sensory aura as well as the consequence of motor phenomena.

The Attack.—The *first phase of the actual attack* is the general tonic spasm. If the patient is standing he falls to the floor unconscious; often while falling there is a piercing cry. Whether the fall is the consequence of

the loss of consciousness or independent of it cannot be answered; but we shall recur to this point later on. The fall is abrupt and occurs with great force so that serious injuries may result. The patient often falls upon his face, striking the forehead. Epileptics may often be recognized at the first glance by a deep cicatrix upon the forehead. But it is not rare for the patient to fall backward. The proximity of dangerous things, such as a heated stove, becomes disastrous to him. Especially if there be no aura severe injuries may result.

Simultaneously with or immediately after the fall the *tonic spasm* begins. It is to be regarded as a rule that it involves both sides of the body in a uniform manner. The head is either somewhat retracted or the so-called "conjugate deviation" of the head and eyes appears (Binswanger does not mention this among the symptoms of the typical attack). Sometimes the head is inclined to one side and rotated slightly upward; if there is at the same time conjugate deviation of the eyes, postures result that are very difficult to define. In other cases the eyes are rigid, or rotated upward, the eyelids either wide open or spasmodically closed, the jaws tightly locked. If the reaction of the pupils to light is tested, it is most often found that it has disappeared; the corneal reflexes are also absent; the tendon reflexes cannot be tested on account of the general tonic tension (see below); the trunk either shows opisthotonos or emprosthotonos, rarely is there curvature to one side. The muscles of the neck stand out tensely and even those of the abdomen show tonic spasm; respiration is arrested. The extremities are either in a position of flexion or extension and are rigid; an attempt to alter the position of these spasmodic, tense members meets with the greatest resistance. Often at the onset of the attack, simultaneously with the lateral rotation of the head and eyes, we note the legs, which are separated, and the arms which are abducted and raised, assume positions of flexion. The fingers are mostly flexed; enclosing the thumb in the hollow of the hand has been regarded as an important criterium of the epileptic attack since ancient times. The face, which was at first ashy pale, gradually takes on a dark blue, cyanotic appearance. This tonic stage lasts but a few seconds. While the body is still tense (muscles of the trunk) the second, the *clonic stage* begins: in the large joints of the extremities there are brief spasmodic flexion and extension movements, the trunk also shows marked clonic contractions, the eyes move to and fro, the muscles of the face are in constant motion, the jaws are brought together exposing the tongue, which is also in clonic spasm, to severe injuries; bloody foam appears at the mouth, the patient gives expression to peculiar gurgling sounds, urine and feces are frequently voided involuntarily, provided this has not already taken place during the tonic stage. An emission of semen is rarer; vomiting is extremely infrequent. Gradually these clonic convulsions lessen in severity, there are longer intervals between them, which may sometimes be occupied by isolated tonic spasms. Finally they cease and there is a feeble tremor of the entire body.

In addition to the convulsions just described there may be other movements in this stage which have a more coördinate character (kicking, etc.). In fact the convulsions of this stage are not uniform but are a *mixtum compositum* and can only be called clonic *cum grano salis*. If there has been conjugate deviation in this stage there is commonly the "fatigue deviation"

of the head and eyes to the opposite side. A deep sigh is often the termination of the attack in typical cases.

Binswanger divides these clonic conditions into:

(1) Locomotor movements (hitting, throwing, pushing and stepping motions);

(2) Jerk-like contractions of the head, trunk, and extremities (especially of the proximal portions of the latter);

(3) Tremor;

(4) Isolated clonic convulsions of individual muscle groups, especially of the distal areas.

Féré calls attention to the fact that the convulsions of the clonic stage frequently show a preference for one side of the body. The duration of this stage is from 1–5 minutes.

In connection with the attack there is usually a deep sleep which lasts several hours. It is much less common for the patient to regain consciousness at once and to resume his work without noting any of the effects of the attack. The patient often has himself awakened from this post-paroxysmal sleep, but this is not advisable, as he is then tired, worn-out, "dizzy in the head," irritable, and out of humor. After being awakened in this manner it may happen that he is in a condition of "dream-like confusion" which corresponds to the post-epileptic dazed condition of epileptic mental disturbance. These post-paroxysmal phenomena are suggestive of restless sleep and apparently associated with dreams; the patient throws himself about, occasionally opens the eyes, mutters a few incoherent words, or performs some complicated movement. The more quiet and deeper the sleep the less difficulty does the patient experience after a spontaneous awakening. In rare cases the post-epileptic sleep is uncommonly profound, so that the patient does not react to any stimulus, the condition corresponding to coma. I recently saw a case in which there was persistent coma lasting 24 hours after an attack; the attending physician having made a diagnosis of meningitis. In these paroxysmal sleep and comatose conditions, it must not be forgotten that Babinski's toe reflex is commonly present upon both sides (as is the usual condition immediately after an epileptic attack); this is quite an important diagnostic factor. Oppenheim's leg phenomenon has been frequently demonstrated. Fürnrohr has demonstrated the presence of the deglutition reflex.

After the Attack.—This leads us to the objective and subjective symptoms which remain after the epileptic attack. Féré has devoted special attention to these and by exact and persistent researches has enriched our knowledge in this realm of motor and sensory phenomena. Many patients suffer for hours and days. These symptoms relate to general discomfort, lassitude, debility, and difficulty in applying their thoughts. A slowing of associated activities may be recognized by determining the reaction time. There are often digestive disturbances (diarrhea). The temperature after the attack rises a fraction of a degree as may be seen from accurate temperature investigations. A great change in metabolism is indicated by the loss in weight which is frequently observed. Often there is albumin, more rarely sugar, in the first urine that is voided; polyuria is infrequent. Mendel and others were the first to call attention to an increased excretion of phosphoric acid. It was

determined, by Lépine and Jacquin, that this increase concerns principally the earthy phosphates and not the alkaline ones, and Féré was led to the belief that the excretion of the total phosphates was not only due to an increase of metabolism, but also to a special nervous process. The entire question as to the phosphates, however, is a disputed point. The increased toxicity of the urine, discovered by Voisin, must be mentioned. In the blood after the attack Hénocque found a decrease in the reducing property of the hemoglobin; decreased amount of hemoglobin with hyperglobulia; Féré found an instability of the corpuscles (after being removed from the circulation they soon took on a spherical shape). Donné reported an increase of leukocytes; Hayem hematoblasts. We shall return, later on, to these investigations in metabolism, as they are the foundation of a special etiologic theory. Féré proved experimentally that the physiologic action of pilocarpin upon the secretion of sweat, after an attack, was decidedly decreased and slower than during the apparently healthy interval. On the other hand salivation has been frequently reported after an attack. Edema and erythema has occasionally been seen. In the cerebrospinal fluid, removed by lumbar puncture, I have been able to demonstrate a slight lymphocytosis after an attack. If this observation proves to be correct upon subsequent investigation, the inference will be obvious that we are concerned with a secondary condition due to a hyperemic process of the meninges. Donat found cholin in the cerebrospinal fluid.

Symptoms referable to the motor functions are regularly found in the form of muscular debility, but this is never so great that it appears clinically as paralysis; it shows itself as a decrease of power. This may be easily and distinctly demonstrated if, as was done by Féré, dynamometric curves of the patient are taken during the period free from attacks. A slight paresis of the ocular muscles is shown by slight nystagmus or nystagmiform movements. The tendon reflexes, after an attack, are decreased, as a rule, after severe ones they are absent, so that in the degree of the decrease there is some measure regarding the severity of the last attack. Féré calls attention to the increase of the idiomuscular irritability.

Severe general muscular debility is noted when quite a series of attacks follow one another in rapid succession. Then the clinical impression of paralysis is present. The patients, especially children, cannot stand, the movements of the arms are uncertain and ataxic. If, by way of a test, the child is stood upon its feet, it collapses; the head falls forward or to the side. Speech is severely impaired, it becomes lalling or indistinct. This last named condition coincides with the fact that even after a single attack sometimes slight speech disturbances are noted. Another sign noted by Féré, with the glossodynamometer invented by him, is that the tongue is always involved to an extraordinary degree in this post-paroxysmal weakness. Babinski's toe-reflex can usually be demonstrated. Thus a picture results which closely resembles infantile pseudobulbar paralysis. Only spasms are not present; on the contrary there is rather hypotonia; the tendon reflexes are not increased. The paretic symptoms disappear when the attacks cease; on the other hand the condition may become stationary, provided the attack continue in undiminished frequency. In a case that I saw recently there was a peculiarly distributed choreiform athetoid unrest over the entire body. Op-

penheim reports the case of an epileptic, aged 11, in whom there was high-grade incoördination of all movements and dysarthria following a period of cumulated attacks.

We cannot as yet discuss the question whether localized motor symptoms of paralysis may transitorily or continuously follow an attack—provided there are many. The same is true of transitory conditions of motor aphasia. Binswanger regards the existence of post-paroxysmal circumscribed symptoms of absence of function as a proof of the existence of a focus and hence an indication for operation. In favor of the fact that we are concerned with symptomatic epilepsy, in cases of a non-traumatic genesis in which there are localized post-paroxysmal paralyses, is this, that in such cases the attack differs from the genuine epileptic one (the attack beginning in one member, with distribution according to the Jacksonian type). The transitory post-paroxysmal monoplegias and hemiplegias then would indicate the clinical manifestations of a latent etiologic factor. Naturally only the most minute microscopic examination, in such cases, would reveal to us whether or not a focal disease was present. On the other hand it must be remembered that not infrequently atypical attacks of genuine epilepsy present distinct features of partial (Jacksonian) epilepsy (Binswanger). There are even cases which most closely resemble a cortical disease and Jacksonian epilepsy, in which no anatomical lesion has been found and these have in part (whether correctly or not) been included with genuine epilepsy (Leo Müller's *idiopathic hemiepilepsy*, pseudo-Jacksonian epilepsy).

Oppenheim assumes that the more developed the post-paroxysmal paralyses and the longer they continue, the more probable is the existence of symptomatic epilepsy. Binswanger reports cases in which there were originally no paretic symptoms pointing to the presence of a localized disease of the brain, the attacks throughout having the character of true epilepsy, which were, however, succeeded by more or less extensive transitory pareses, until finally well marked symptoms of paralysis dominate the inter-paroxysmal clinical picture. He believes that these are transitional cases between functional and organic epilepsy in that there are minute pathologic foci forming the starting point of the affection which cannot be recognized upon a macroscopic examination of the brain. These cases are, perhaps, identical with Voisin's *epilepsia spasmodico-paralytica.*

A case that I saw recently illustrates the difficulties of the question under discussion.

A boy, aged 13, presented nothing in his history of note, except that he was asphyxiated at birth and had a mild attack of measles at 4 years of age. When 7, peculiar attacks first appeared: the patient became pale, and with a rigid facial expression there was a semi-rotation about the longitudinal axis of his body. (The parents did not know to which side the rotation occurred.) The attacks then ceased up to his tenth year; upon their recurrence there was, in addition to the rotation of the body, a fall with loss of consciousness and involuntary evacuation of urine. The attacks then changed their entire character, for convulsions appeared which, according to the report of the parents, began in the left half of the face, principally implicating the left half of the body and these were succeeded by transitory weakness of the left half of the body. In these attacks there was involuntary passage of urine. The attacks that we observed corresponded to those of ordinary epilepsy, only the tonic tension of the left half of the body was distinctly stronger, there was conjugate deviation of the eyes to the right, consciousness was lost at once at the onset

of the attack, when there was at the same time involuntary passage of urine; neither did the clonic stage present peculiarities, at most a slight predominance of the convulsions upon the left side of the face; but the cessation of the attack was quite peculiar, corresponding entirely to the Jacksonian type. The convulsions first ceased upon the right side, then in the left leg, and, finally, there was only convulsion in the muscles supplied by the left facial nerve. After the attack there was transitory paresis of the left half of the body, with an increase of the reflexes, and also Babinski's sign. After an accumulation of attacks the paresis became continuous. After these attacks were arrested by the administration of chloral, there was a distinct tendency to improvement of the paresis.

Any of the organs of special sense, just as they may be the seat of an aura, may present residual symptoms of absence of function. A limitation of the field of vision was noted by Féré in all patients in whom an examination had been made during the first 30 minutes after the attack. Thomsen states that he has noted these symptoms of absence of function only after the psychic equivalent of the epileptic attack. Bilateral or unilateral deafness, decrease of the acuity of hearing, have been reported as post-paroxysmal phenomena. A very interesting observation has been recorded several times (Charpentier), namely, simultaneously, anesthesia of the pinna and of the mastoid process upon the affected side. In post-paroxysmal amblyopia, there is said to be a similar condition in the region of the eye. The most common disturbance in sensation is a diminution of the pain sense which continues for a long time. By some authors this is associated with the slowing of the associated activities which exists after the attack. In its most severe grades (*epileptic stupor*) it is particularly well developed. Localized disturbances of tactile sensation are often found in combination with local motor paralyses. Finally, post-paroxysmal ageusia and anosmia must be mentioned.

Varieties of Attack.—The epileptic attack does not always present a typical character. There are deviations and VARIETIES which are just as necessary for the physician to understand as the appreciation of the attacks that run a typical course. These *atypical* attacks appear either between the typical ones or exist alone forming the exclusive clinical picture. Binswanger differentiates the following varieties: 1. The *fully developed atypical attack,* 2. the *rudimentary attack,* 3. *abortive attacks.* The plainest case is when all phases of the typical attack appear and only present an irregularity in their course. Thus it may occur that the clonic stage precedes the tonic, or that clonic and tonic spasms alternate, or are present in different members at the same time, further that the tonic spasm appears before consciousness has been lost so that the patient notes the "stiffness" of his limbs. Binswanger points out that in these latter cases the tonic convulsion does not set in as a generalized spasm but that it is frequently unilateral, or in the form of paraspasms. These fully developed atypical cases are contrasted with the rudimentary attacks. Here, besides the loss of consciousness, there is only a single component of the convulsion, clonic or tonic spasm, or they are substituted by coördinate or locomotor movements, in part or entirely. Loss of consciousness and partial tonic spasm is quite common in the *apoplectiform variety* of epilepsy; this is often confounded with conditions of syncope. In these cases the tonic spasm, if present, affects the muscles of respiration, so that breathing is arrested and cyanosis appears. The patient is entirely immobile as if in the deepest syncope, but even in the absence of tonic spasm

of the muscles of respiration, there is often involuntary evacuation of urine. The pupillary light reflex is absent. Loss of consciousness and clonic convulsions are also noted in other cases that closely simulate syncope, and in which the flaccid and motionless patient presents momentary spasms. Further, those cases which resemble the infantile *tic de salaam* or *hysterical shaking spasm* belong to the category of Binswanger. There must be included here also those cases in which there is tonic spasm, in the members of one lateral half of the body, perhaps in an arm, while the other side of the body shows coördinate movements—most frequently there are brushing and grasping motions. I recently saw a case in a boy, in whom, besides the loss of consciousness, there was tonic spasm only. During the time of the generalized tonic tension of the body rhythmic masticatory motions were noted. This observation leads us to the discussion of cases in which the tonic or clonic spasm is replaced by coördinate or locomotor movements. Among these is the so-called "*manège epilepsy*" and *epilepsia rotatoria*, in which there are circular movements upon the axis of the body performed while the patient is unconscious, and *epilepsia procursiva*, in which there is unconscious propulsion and then the patient falls or in which these movements cease upon regaining consciousness. *Epilepsie rétropulsive* has been described, and *epilepsie marmottante* must be mentioned, in which there is the repetition of words without sense, the patient being unconscious.

The final group of atypical cases of epilepsy includes those in which either the motor spasm or unconsciousness is lacking or in which one of these components is merely indicated. Here we must mention first: the lightning-like, transitory, abortive attacks or **petit mal**, so-called since antiquity. During these attacks the patient suddenly with visible pallor (or blushing) ceases his activity, permits an object to fall out of his hand, or suddenly stops in the street while walking, looks rigidly into space, immediately resuming his walk or going on with his previous occupation. The knowledge of these forms of the attack, although quite ancient, has unfortunately not yet become the common property of physicians, so that many cases of this kind remain unrecognized. I cannot refrain, at this point, from paying a tribute to the excellent faculty of observation of the Berlin school teachers, who send many patients of this kind to the Clinic, often with a complete diagnosis. These patients frequently do not know of their attacks, provided there is no aura; not rarely the loss of consciousness is not so profound, the memory of the attack, therefore, being retained, it is regarded by the patient as an attack of vertigo or syncope (*vertigo epileptica*). Often there are isolated motor symptoms mixed with these abortive attacks; slight contractions in an extremity or still more frequently coördinated movements (snuffling, smacking the lips). Involuntary evacuation of urine is not rare. I have at present under observation a child, who during an attack of petit mal while eating does not cease chewing, becomes pale, and gazes fixedly into space, not responding to questions.[1] In other cases of petit mal the motor symptoms, in proportion to the loss of consciousness, are more prominent. These may consist in isolated clonic contractions or in a brief tonic spasm of an ex-

[1] It must be remembered that in some attacks only inhibitive processes affect the cortex, while the subcortical centers that bring about the activity of the principal movements (perhaps also mastication) continue with undisturbed function.

tremity. If there be no loss of consciousness these cases are very difficult to differentiate from motor auræ, especially if no attack follows. Vasomotor symptoms (pallor) are present as a rule. Some few, isolated, muscular twitchings may appear inter-paroxysmally and we must not fail to include Unverricht's cases in which there is a *combination of myoclonia and epilepsy.* Then there are also cases in which the patient, without loss of consciousness, suddenly collapses in the knees or falls and immediately rises; attacks which Binswanger characterizes as isolated discharges of inhibition in the motor regions, corresponding to his genetic theory, which will be discussed later on.

Another variety of the attack must be described in which vasomotor symptoms are very prominent, these cases having been given the name *vasomotor epilepsy.* These attacks run their course with profuse outbreaks of perspiration and a rise in temperature; the amount of urine is greatly decreased.

When we refer to the *epileptic equivalent* we mean the psychical paroxysms of epilepsy. The term equivalent is also employed for a number of neuropathological phenomena. The so-called *epileptic vertigo,* in the majority of cases, is not true vertigo, but an attack of petit mal, with but slight disturbance in consciousness. But true attacks of vertigo may also occur as epileptic equivalents (Féré). There must also be mentioned outbreaks of perspiration and attacks of tremor (Féré), as well as attacks of *narcolepsy,* from which the patient cannot be roused. Oppenheim mentions, as an equivalent of this kind, a comatose condition lasting several days.

INDIVIDUAL SYMPTOMS

In proceeding to discuss the individual symptoms of the epileptic attack, quite a number require but a brief consideration, for they are only the secondary results of some of the important manifestations of the attack. For a considerable number an explanation, or more correctly an attempt at an explanation, will be found, when we have learned to understand the mechanism of the attack. The initial cry belongs to the first group and is due to the air being forced through the spasmodically narrowed glottis, the muscles of the abdomen and of respiration being in tonic spasm. The old view of Marshall Hall must be mentioned, in which the tonic spasm of the muscles of the larynx was regarded as the primary condition and all of the succeeding conditions as the result of a carbonic acid saturation of the blood (*trachelismus*), a view which was very easily disproved for, after producing a fistula in the trachea, the attacks did not cease. Spasm of the muscles of respiration and the dependent dyspnea certainly cause the increasing cyanosis, which succeeds the initial pallor. Small cutaneous hemorrhages (or hemorrhages from the mucous membrane: conjunctiva), are probably due to venous stasis; these are often seen as the residues of the attack. Binswanger points out that the region about the mastoid process is the area of predilection of these hemorrhages. Others refer these small bleedings to the greatly increased blood pressure which is said to be present at the beginning of the attack; this has, however, not been definitely proven. Other investigators maintain, on the contrary, a fall in blood pressure at the onset of the paroxysm. Thus. it was noted that a bleeding wound of the forehead ceased to ooze blood at

the beginning of the attack; Binswanger, however, points out that this may have been due to a tonic contraction of the muscles, thus causing the hemorrhage to cease. Investigations of the blood pressure by the sphygmometer during the attack encounter great technical difficulties so that a definite conclusion cannot be obtained. The reports in regard to the vessels of the eyeground, from which it was expected that an idea might be obtained as to the condition of those of the body and especially of the cerebral arteries, differ to such an extent that they are valueless. Sometimes dilatation is found, other observers report contraction of the retinal arteries. Bechterew, from investigations in dogs and cats, arrived at the conclusion that during the entire course of the attack there was raised arterial and venous pressure. Examinations of the cerebrospinal fluid by the same author render it likely that there is an increased arterial flow to the brain. My finding of a postparoxysmal lymphocytosis is in favor of the same view.

Among the sequels of the clonic stage there must be mentioned the injuries to the tongue; this organ is tossed to and fro between the jaws during the clonic convulsion. These to and fro movements of the tongue probably also produce an increase in the salivary secretion, and, partly from the effects of the spasm of the muscles of mastication the saliva becomes foamy and intimately admixed with the blood which originates from the bitten tongue; then it appears as bloody foam at the lips. The clonic contractions of the diaphragm explain the peculiar sounds which the patient emits. Evacuations of urine, feces, and semen are most likely due to an involvement of the smooth muscles in the convulsion during the tonic and clonic stages, or they are the consequence of the effect of spasm of the transverse striped muscles of the abdomen, anus, perineum and sphincter of the bladder. The same considerations may be invoked to explain the vomiting, in which probably also an accumulation of swallowed air, due to spasm of respiration and deglutition, plays a rôle.

THEORIES OF EPILEPSY

Passing to the origin of the tonic and clonic spasms, as well as the other symptoms of the attack, we must first investigate the *theories of the causation of the epileptic insult and the seat of the epileptic lesions* and in this connection inform ourselves in regard to the results of animal experiments. The historical development of these theories gives us at the same time an interesting picture of the evolution of our views in regard to the functional activity of individual portions of the brain. So long as it was maintained that the cortex of the cerebrum was not concerned with the motor functions, these being associated with the mid-brain, the "medulla theory" of epilepsy was in bloom. When Fritsch and Hitzig published their celebrated discovery in 1871 upon the electric excitability of the cerebral cortex and thereby deprived the old Flourens theory, which stood for the functional equality of all portions of the brain, of all foundation, it was obvious to assume that all the symptoms of the epileptic attack were due exclusively to processes that affected the cortex of the cerebrum. We will see that here also we have taken too much for granted and that the true solution is probably between these views.

The medulla theory was favored by Kussmaul and Tenner. It was based upon the observation that if the arterial supply in animals was cut off (hemorrhage, bilateral ligature of the carotids) this was capable of producing epileptiform convulsions. These convulsive movements, consisting in striking, pushing and treading, also took place when the cortex of the brain and the basal ganglia were removed. In the human epileptic attack this cutting off of the arterial blood supply was supposed to result from a primary stimulation of the vasomotor centers in the medulla oblongata. The resulting anemia of the cerebrum gave rise to loss of consciousness; that of the mid-brain causing convulsions.

Binswanger raises the following objections to this view:

(1) Kussmaul and Tenner did not succeed in demonstrating that the medulla was actually the region causing the convulsion, for later experiments (Frensberg, Luchsinger, Schroff) proved that the spinal cord in itself, after being separated from the medulla, was capable of producing similar spasms;

(2) It could not be proven that an irritation of the vasomotor center in the medulla oblongata gave rise to an anemia of the cerebral cortex. On the contrary, the investigations of Riegel and Jolly demonstrated that neither section of the cervical sympathetic nor irritation of the central stump had any influence upon the filling of the vessels of the pia.

The medulla theory appeared to gain some confirmation in the pathological findings of Schröder van der Kolk's, who found small vascular dilatations in the fourth ventricle, and in the experiments of Nothnagel, who succeeded in producing spasms in animals by mechanical irritation of some points in the medulla oblongata. Nothnagel modified the medulla theory to the extent that simultaneous and coördinate with the irritation of the vasomotor center which produces anemia of the cerebral cortex, there is an irritation of the "hypothetical spasm center" in the pons. That this irritation of the "spasm center" in animal experiment may be merely reflex is emphasized by Nothnagel as well as by later investigators. The transmission was supposed to occur by way of the sensory trigeminus root. Nothnagel's experiments on rabbits were later repeated, extended, and supplemented by Binswanger and Ziehen. If we study the results of these experiments, which refer in part to the region of the pons (Binswanger), in part to the corpora quadrigemina and the optic thalamus (Ziehen), 3 series of associated phenomena appear: 1, tonic or tetanic tension of all of the muscles of the body; 2, associated movements, such as kicking, stepping, running and tramping; 3, locomotor movements. As these symptoms appear in the picture of spasm even after the influence of the cerebral cortex is cut off, the assumption is justified that in the cerebral areas under discussion there are special centers for these previously mentioned movements. Pathologic irritation causes them to become spasm centers; their physiologic significance being similar, particularly the last 2 named varieties, in which we have but an abnormal increase of the physiologic form; occasionally this may be quite different and is true especially of the tonic spasm. Binswanger was unable to produce associated movements from these areas of the brain in dogs nor has it been possible to produce them from higher portions of the canine brain. Binswanger deduces, from this fact discovered by him, that even in the dog, the view of a pure medulla origin of the epileptic seizure is not justified.

The medulla theory received a severe blow by the Fritsch-Hitzig discoveries and above all by the further fact reported by Hitzig, that in the dog, after extirpation of the motor cortical centers, there may be spontaneous recurring attacks which very closely resemble the epileptic seizure in man.

This at once altered the entire position of affairs and the cortex of the cerebrum was looked upon as the point of origin of the epileptic attack and very soon this region was regarded as the only area from which the symptoms of the seizure could be produced. Unconsciousness, as a clinical symptom, was readily understood from this new conception. The clonic contractions originating from irritation of the cortex, which first appeared isolated in circumscribed muscle areas then being distributed in conformity with the anatomic law of position in relation of the cortical centers, did not resemble the epileptic seizure in the human being very closely; nevertheless, there could be but little objection to the above mentioned view, for, not only could the clonic component of the epileptic attack be produced by cortical stimulation but also the second component, the tonic convulsion, was brought about by the application of stronger currents.

Even to-day numerous exponents of the pure cortical theory, as described above, regard the tonic spasm as the effect of an extraordinarily strong summation of irritation; a single one being capable of producing a clonic contraction. Gowers is of the opinion that it is the venous stasis which causes the tonic convulsion in the second stage of the epileptic seizure to dissolve itself into clonic convulsions. But we have already seen that tonic spasms and other symptoms (motions of running and walking) which very closely resemble some of the phenomena that were described in the symptomatology of the epileptic attack, may be produced in animal experiment by irritation of infracortical regions.

The experiments of Ziehen have rendered it very likely that even the contractions obtained upon cortical stimulation may be subdivided into cortical and infracortical components. Ziehen extirpated a certain cortical center and stimulated the neighboring area by faradism. It was noted that with medium strong currents the extremity corresponding to the extirpated center revealed tonus, while clonic cortical spasms were predominant elsewhere. Upon stimulation of the exposed medullary layer only tonic convulsions appeared in the extremity. These researches gave birth to another view than the one mentioned above, which considered, not only the cortex of the cerebrum but also the infracortical centers, as responsible for the epileptic seizure. The intimate functional relationship of the different parts of the brain to some extent justifies this view. The irritation first affects the cortex —this is indicated primarily by unconsciousness. It is most probable that at the same time an irritation of the infracortical centers also takes place.

But all of these animal experiments have not produced an epileptic attack which in all points resembles the seizure in man, and, therefore, what we endeavored and hoped to attain (namely, an explanation of the seat and the lesion of epilepsy) and especially an insight into the nature of epilepsy has as yet eluded us. The entire subject remains entirely hypothetical. For a long time the comparison of a "discharge" or, something more concrete, the comparison with the process in a Leyden jar (Schröder van der Kolk) was employed. The nerve cells, it was believed, in consequence of their dis-

turbance in chemism have lost the faculty to eliminate and free energy in a normal manner. This leads to an accumulation of energy which is then explosively discharged—in the form of an epileptic attack. Hughlings Jackson referred to the "discharging lesion"; he believed, that in consequence of an arterial occlusion, particularly in a certain group of cortical cells, especially at the periphery of the region which was deprived of function by this arterial occlusion, the faculty mentioned above was lost. This group of cells was regarded by him, to a certain extent, as a "hyperphysiologic parasite," the epileptic being the host, and when these cells became active they not only functioned abnormally themselves but also compelled the other areas of the brain to assume the same extravagant activity. Binswanger accepts this picture of a discharge. According to this author such a discharge may become operative in 2 different directions, firstly in the sense of a stimulation and secondly in that of inhibition. In every nerve cell besides stimulating there are also inhibitive processes. The reaction of a cell to irritation does not depend upon the force of the stimulation and upon the degree of the irritability of the cell (this also has been invoked to explain the epileptic change), but upon other stimuli which reach the cell, i. e., in other words, whether the cell is in condition of rest or activity. Binswanger regards the nature of the epileptic alteration as a disproportion between stimulating and inhibiting processes, in an unequal relation between capacity for action and resistance to action in the cortical cells. If the process of irritation is increased in the active cell, there is, finally, a discharge of stimulation; in the quiescent cell the process of inhibition is increased to a discharge of inhibition. Here it is necessary to discuss whether the expression "discharge of inhibition" is happily chosen. It can only be understood if we regard the irritative and inhibitive processes in the cell as physiologico-chemically equal functions. In general an absence or inhibition of function rather raises the thought of a minus energy becoming liberated than of a discharge. Therefore, in explanation of the occurrence of the inhibitive symptoms of the epileptic seizure, other possibilities come into question. It is possible that the irritative discharge, in certain cell groups, may have an inhibitive action in other regions of the brain, this might explain the coincidence of the tonic spasm and the loss of consciousness (Gowers). Or in the districts of inhibition there may have previously been a latent stimulation so that there may be exhaustion and not inhibition. This latter view is favored by Féré. Binswanger admits that it is very difficult to discriminate, clinically, between inhibition and exhaustion but he holds that Féré confounds these two series of symptoms.

Binswanger's theory unquestionably has great didactic value, for it refers the complicated symptoms of the seizure to the simple formula of a discharge of stimulation and inhibition. If we regard the various symptoms of the attack in the light of this theory, the various auræ appear as—frequently circumscribed—discharges of stimulation and inhibition of the cortex. On account of their localization it was thought that they should be regarded as focal symptoms which indicate the region of the original irritation, and cases of epilepsy especially after focal diseases of the brain were pointed out in which the aura actually corresponded to the functional nature of the cortex at the lesion. Hence the doubt expressed by Lasègue as to the value of the

aura as a genuine epileptic symptom. Authors even went so far as to refer all cases of epilepsy to a latent focal disease, so that the conception of genuine epilepsy would be entirely repudiated.' These conclusions have not, however, up to this time been confirmed by pathologico-anatomical findings, so that a justifiable doubt of their correctness is certainly reasonable. It is true we do not know the causes for the limitations of many auræ and, as I must add, of many paroxysmal and post-paroxysmal symptoms. Binswanger in addition to his view, cited above, calls attention to the fact that the degree of activity of the individual cortical area may be decisive for the effect of an irritation. Perhaps individual conditions (varying degrees of stimulation and exhaustion of different cortical areas) play a rôle. The attack begins with a diffuse discharge of inhibition in the cortex, which is shown clinically as loss of consciousness and a sudden fall (inhibition discharge in the motor region). Both symptoms, according to Binswanger, are not dependent upon each other, but are coördinate, for on the one hand, there are epileptic conditions in which, in spite of profound unconsciousness, the patients do not fall, and on the other hand, there are instances in which the discharge of inhibition is limited to the motor region, discharges of inhibition in a restricted sense. During this period of the seizure the discharge of stimulation is supposed to occur only in the infracortical areas.' The expression of this infracortical stimulation discharge is the tonic convulsion. Only after inhibition has ceased in the cortex do stimulative discharges appear in the form of clonic contractions. The infracortical component (striking and stepping motions) often even now may preponderate. Other authors maintain that the fall is the result of loss of consciousness (Oppenheim). This view is logical if the point of origin of the tonic spasm is referred to the cortex.[1]

In regard to the other classical symptoms of the epileptic insult (salivation, circulatory and respiratory disturbances), according to Binswanger, there is no objection to the opinion that they originate from stimulation of cortical and infracortical centers. Clinically this is favored by the frequent, isolated appearance of these symptoms in abortive attacks. The initial pallor deserves special mention for this was regarded formerly as the clinical expression and proof of a primary alteration of the vasomotor center in the medulla oblongata. Binswanger refers it to an infracortical discharge of stimulation equal to the cortical discharge of inhibition; Jackson and Gowers believe that it is secondary. Jackson thought that it was directly dependent upon cortical discharge (stimulative discharge), for, drawing his conclusions from peripheral vascular spasm, he assumed an arterial spasm in the affected cortical region. Gowers considers the arterial spasm as reflex, the consequence of cortical discharge.

INTERVALS BETWEEN ATTACKS

Up to this time we have devoted our attention exclusively to the individual epileptic attack. A seizure, no matter how much it may correspond to the

[1] That the original point of attack of the irritation which gives rise to the spasms is only the motor region, is disputed by Unverricht, who upon the basis of some animal experiments (which are, however, disputed) also regards the posterior cortical areas (occipital lobes) as epileptogenous.

epileptic type, is not yet epilepsy. The characteristic of the affection known as epilepsy consists in the repetition of attacks from time to time, either spontaneously or as a reaction to some deleterious cause.

The INTERVALS between the various attacks vary in different epileptics according to the severity of the malady. There are epileptics who have an attack only every year or every month, others in whom there are but weeks or days between the seizures. Sometimes there is a variation in the frequency of the attack, in the sense that there are periods of infrequent seizures which alternate with those in which the attacks are numerous. Especially the rudimentary and abortive attacks (Binswanger) are the ones which show a cumulative effect. Seizures with long intervening periods mostly correspond to the fully developed type. Under the last named circumstances it may happen occasionally that a fully developed attack is replaced by a number of abortive or rudimentary ones; being to a certain extent divided or broken up (Binswanger). As a matter of fact the greatest variation in the course is possible and this may be still further modified by the intervention of psychical epileptic equivalents. Frequently only slight vertigo, during long intervening periods, indicate the continuance of the malady. If an accumulation of seizures takes place we speak of a *series of attacks.* These series may present a mixture of various types and may be followed by severe conditions of bodily and psychical exhaustion (*epileptic stupor*). If, during a prolonged series of attacks, there is no return of consciousness in the interval between the convulsions, we speak of the *status epilepticus.* These conditions, endangering life, are characterized by the addition of some other symptoms, above all by a decided rise in temperature. Often there is an early and serious paresis of the muscles of deglutition. Binswanger differentiates between a convulsive and a comatose stage of the *status epilepticus.* The latter either corresponds to profound coma or to a condition of unconsciousness with low muttering delirium. Death is often due to pulmonary edema or acute cardiac insufficiency.

The time of day at which the attacks appear varies greatly. Féré noted a preference for the early morning hours. There are, however, cases in which the attacks occur particularly or exclusively at night (*nocturnal epilepsy*). These attacks are especially important because they often escape recognition. The cases of *enuresis nocturna,* arising in late childhood, are particularly suspicious in this respect. In these instances the enuresis may be a symptom of the attack. But true *enuresis nocturna* may introduce epilepsy; this is also without question in the case of *pavor nocturnus* and of somnambulistic conditions, which, however, belong to the realm of epileptic mental alteration. In these instances we refer to *larval epilepsy.* If, as is not rare, there are at first only nocturnal attacks, and then late in the course of the malady these appear during the day, we may easily be misled as to the period of the onset of the seizures. In regard to the affection in general it may be stated that it appears particularly during youth, during the first 2 decades. In childhood there is very often a connection with acute infectious diseases, a subject which we shall investigate more thoroughly in the description of the etiology.

The views are far asunder in regard to infantile convulsion being of epileptic origin. These infantile convulsions (*eclampsia*) differ from reflex

epilepsy in that the convulsions are due to a transitory peripheral irritation, disappearing after this has ceased; in reflex epilepsy there is a permanent peripheral irritation of which, finally, the convulsions may be independent, so that after removal of the irritation they do not cease. Binswanger, in contrast to Féré, separates eclampsia from epilepsy. These convulsions, which result from the most varied kinds of irritation, especially those resulting from the intestinal tract, are due (Binswanger) either to an increased tendency to convulsions, which exists at this period of life, or they they are due to a hereditary neuropathic predisposition—and this he regards as an etiologic point of contact with epilepsy. He differentiates from eclampsia, the convulsions of the first days of life, regarding them only as a "signal of incapacity to live." That persons who have suffered from infantile convulsions may develop epilepsy as well as other neuroses, such as hysteria, later in life is in favor of Binswanger's view of the neuropathic genesis of eclampsia; but the fact of the occurrence of hysteroepilepsy cannot be used in favor of a distinctive position of eclampsia. This question is therefore still *sub judice.*

Puberty shows a special preference for the development of epilepsy, especially in the female. In this decade, as may be gathered from statistics, there is a preponderance of feminine epileptics which is decreased later on. With the third decade there is a decrease in the frequency of the malady, but cases have been reported which originated in the fourth decade and even later; however, these cases arising late in life occupy a special position (*epilepsia tarda*) in regard to etiology in so far as they are often due to cerebral arteriosclerosis and hence usually must be included with symptomatic epilepsy.

PROGNOSIS

The *prognosis of epilepsy* is always serious, and *quoad restitutionem* at all events dubious. There are some cases in which apparently a cure takes place, but this number is small, although formerly it was probably underestimated (about 5%). Various dangers threaten the epileptic, above all, progressive mental deterioration and dementia. This is directly proportional to the frequency of the attacks, so that the prognosis is the more serious the greater the number of attacks. Cumulative attacks of *petit mal* are quite justly feared, especially as they are very apt to be exceedingly resistant to bromin treatment. But direct danger to life also threatens the epileptic in the form of severe injuries during the attack (I recently saw a fracture at the base of the skull the consequence of an attack), severe post-paroxysmal conditions of exhaustion with dyspeptic symptoms, serious psychoses with marked motor irritation, and above all the *status epilepticus.* Fatal cases during the attack have also been reported, the cause in such cases being embolism, softening, hemorrhage, rupture of the heart, asphyxia. Epileptics are said to have a shorter period of life than normal persons. Among intercurrent diseases that cause death, tuberculosis is particularly mentioned. A variety that is especially unfavorable, rapidly leading to dementia and death, has already been mentioned, Voisin's *epilepsia spasmodico-paralytica.* I should like to call attention to a fact, at this point, in regard to prognosis, namely, that in hereditarily predisposed and psychically degenerated indi-

viduals (*dégenerés supérieurs*, hereditary, psychopathic constitution) epileptic or epileptiform attacks occur occasionally as a reaction to some deleterious factor. In these cases the prognosis is usually unfavorable although only one attack may appear. But psychical degeneration, just as in the acute degenerative psychoses, sometimes has rather a favorable prognosis. Westphal's cases probably belong to this category, in which an isolated epileptic seizure introduced a very complicated psychical pathologic condition.

ETIOLOGY

In turning to the etiology we may refer to much that has already been stated, for it was necessary to consider many of these points in the preceding subject matter. The immediate causes of the epileptic change are shrouded in deepest mystery, provided we do not consider as such the anatomical brain lesions which are sometimes present. Among the mediate causes we must differentiate between the predisposing and the exciting ones. Binswanger requires a further division, into preparatory and productive causes of the epileptic alteration, and of the individual attack. In practice this division cannot be carried out in the majority of cases; predisposing and exciting causes frequently appear to be the same, the exciting cause of the epileptic change is often the exciting cause of the first attack; the predisposing causes of the first attack, as a rule, escape our knowledge. On the other hand, we often find the exciting cause of the individual attacks in the existing epilepsy to be the accidental cause. We have learned already that epilepsy may be due to focal disease of the brain.[1] This corresponds to Hitzig's observation, that, after extirpation of motor cortical centers in animals, epileptiform convulsions develop. It also explains the not infrequent combination of epilepsy and infantile cerebral convulsions. This genuine epilepsy, developing upon the floor of a focal disease, must not be identified, at least not clinically, with Jacksonian epilepsy, which is the expression of a circumscribed cortical irritation. If the focus has an infracortical location, partial tonic convulsions (or tremor) may occur, according to Binswanger; these are neither genuine epilepsy nor Jacksonian epilepsy. There are cases of focal disease in which there are, at first, convulsions of the Jacksonian type, these being replaced later by true epileptic seizures. This may give rise to an intermediate stage of convulsions which may present features of Jacksonian epilepsy as well as of the ordinary attack (hence the proper doubt of the primary nature of all epilepsy in which the attack reveals a decided admixture of incomplete convulsions). It may then be said that the irritation, which was at first local, has distributed itself gradually and in a diffuse manner over the entire cortex of the brain; it is, therefore, usually independent of the focus which gave rise to the original circumscribed irritation, for it continues to exist after the (operative) removal of the focus.

We have already referred to the enormous influence of hereditary factors. At the present time this is hardly questioned except by Delasiauve who does not recognize it as of importance. In the overwhelming majority of cases it is a hereditary, constitutional, neuropathic predisposition upon the founda-

[1] Cysticercus epilepsy belongs to this category.

tion of which epilepsy develops. Any damage in the individual's life is sufficient to set the stone rolling, to produce the epileptic alteration and simultaneously the first epileptic attack. This heredity depends either upon the fact that the germ plasma of one of the two progenitors was deficient from the start or, that during life it suffered a damage of some kind which deprived it of its complete function. In the former case we note traces of the neuropathic predisposition also in the ascendants. Epilepsy is very rarely transmitted from the parents to the child; the figures, however, increase to a decided extent if we include in the statistics children of epileptics perishing from infantile convulsions. Usually there is not a direct, similar heredity, but a direct or indirect dissimilar heredity, i. e., in the direct ancestors or in a collateral line of the ascendants there are other affections, which also develop upon the foundation of a neuropsychopathic predisposition, namely, neuroses and psychoses of the most varied kind. An especially intimate relation has recently been recognized between migraine and epilepsy, not only because migraine is often found in the ascendants of epileptics, but because migraine and epilepsy are often associated in the same individual. In the second instance it is due to the action of chronic intoxications and chronic diseases of the germ plasma. Here I must first mention, and particularly emphasize, chronic alcoholism; in the direct ascendants of epileptics it is found with extraordinary frequency. Formerly to acute alcoholic intoxication was ascribed a deleterious influence upon the germ plasma and etiologic importance was attached to procreation under the influence of liquor. Recently emphasis is laid upon the point that neurotic and psychopathic individuals readily tend toward alcoholic excesses, also that they are often intolerant of alcohol and in the condition of alcoholic excess show a special tendency to sexual activity. Among other chronic intoxications there are to be mentioned: chronic lead poisoning and morphinism; among chronic diseases, syphilis, tuberculosis, arthritis deformans, malaria, diseases of the blood, gout, and diabetes. Here it must be remembered that the two last named affections are also regarded by some authors as the expression of a neuropathic predisposition. Among the cases of acquired inferiority of the germ plasma those may be included in which the only etiologic factor of a neuropathic predisposition consists in the advanced age of the parents, especially of the mother. A convergent predisposition, i. e., the coincidence of a congenital and acquired deficiency of the germinal products of both parents is particularly serious to the offspring. Féré sees great danger in the tendency of neurotics and psychopathics of all kinds entering into wedlock. In so far as epilepsy is concerned, in these and other cases of grave degenerative heredity, there is not infrequently a quickening of epilepsy with a congenital inhibition of development, forms of feeble mindedness up to complete idiocy, or with the varied neuroses or psychopathic conditions. In milder cases there are frequently only a number of somatic signs of degeneration (asymmetry of the skull, anomalies of the ear, and of the teeth, etc.) in addition to those of a psychical sort. Not only is there a hereditary predisposition, but there is also an acquired one, developing early in the life of the individual, intrauterine or extra-uterine, which may furnish the predisposition for the development of epilepsy. It is probable that harmful agencies which act upon the fetus involve the organism, the more seriously the earlier they act (Bins-

wanger); then either severe anatomical disturbances in development appear, or, in milder cases, a general constitutional weakness of the organism. The later this effect occurs, after the individual organs have begun to develop, the more the individual organ appears to be affected and especially the nervous system; in milder cases only in the sense of a constitutional inferiority. In regard to injuries of the fetus, bodily and psychical trauma (fright) in the mother are especially important. The latter probably acts as a circulatory disturbance. But trauma at birth (forceps, narrow pelvis, asphyxia, fall of the child) and also that immediately following birth is not without importance in the etiology, apart from the fact that by causing an injury to the brain, it prepares the ground for the development of epilepsy. In regard to the factors which are active in later life there are especially two points to consider: first, we will find among them a number which we have already recognized as damaging the germinal plasma, and second, a number of these agents may produce the epileptic change or they may play the rôle of the causative factor.

That physiologic processes may assume this part has already been stated; the best examples of this kind are the physiologic processes at puberty. Quite frequently the first menstruation is the cause of an outbreak of epilepsy; if the later menstrual periods are the invariable, accidental causes for the appearance of the attack, periodicity appears which gives rise to the *menstrual* or the *pre-menstrual type of epilepsy.* Here pregnancy and lactation (epilepsy of pregnancy and lactation) must be mentioned. During the climacterium sometimes an old quiescent epilepsy reappears, just as other neuroses show an exacerbation at this period. Coitus is said to be an exciting, etiologic factor; it is positive that this is much more frequently the accidental cause of an attack when the affection already exists.

The acute infectious diseases are of great etiologic importance, especially in childhood. It is a well known fact that epilepsy may develop after measles, scarlatina, pertussis, diphtheria, enteric fever. The effect of the infection may vary; either it is the exciting factor, provided the predisposition is already present, or, in consequence of toxic influences or otherwise as by an accompanying encephalitis (which may be without symptoms), it gives rise to the epileptic alteration. The conditions are still more complicated in the case of the chronic infectious diseases, especially syphilis, and here hereditary as well as acquired lues must be taken into account. We have already learned to recognize syphilis in the parents as a factor capable of damaging the germinal plasma. Here syphilis is not transmitted; only the neuropathic predisposition which may give rise to epilepsy is produced. Should the virus be transmitted we must again differentiate two conditions: first, epilepsy may develop from specific meningitic or circumscribed gummatous processes (symptomatic epilepsy), or it appears as a pure dynamic disturbance probably due to the action of toxins. The same differentiation is true of cases of epilepsy after acquired syphilis. "Functional" syphilitic epilepsy, according to Fournier, is said to appear especially during the secondary stage and to disappear with this stage (Fournier's *parasyphilitic epilepsy*). This disappearance is denied by other authors. Occasionally syphilis may act as the exciting factor, provided the predisposition is present. It must always be remembered that epileptiform convulsions may for a long

time be the isolated prodromes of a metasyphilitic disease (dementia paralytica). Féré mentions the frequency of the simultaneous occurrence of tabes with epilepsy; this is probably a combination of tabes with arteriosclerotic epilepsy (analogous to the combination of tabes with cerebral thrombosis, etc.) or even with syphilitic epilepsy. Among the other chronic infectious diseases malaria must be mentioned. Other chronic affections that have been associated with epilepsy are: rickets, scrophulosis, anemia, chlorosis, leukemia, gout, arthritis deformans, and diabetes. In regard to rachitis and scrophulosis, these diseases, as emphasized by Binswanger, are mostly the signs of a general constitutional debility. As this is frequently associated with a neuropathic predisposition, in so far as the latter is merely a partial condition of the former, epilepsy and the previously mentioned affections should not be placed in etiologic dependence, but are rather to be regarded as conditions parallel to one another. Nevertheless, the severe nutritive disturbances in rickets and scrophulosis may be concerned to a considerable extent in the development of epilepsy. The etiologic association of epilepsy with gout and diabetes insisted upon by French authors is rejected by Binswanger. That these two affections may be regarded as stigmata of a neuropathic predisposition has already been stated. When they occur early and are associated with epilepsy we must at the same time consider a parallelism in the symptoms. As a rule, however, the convulsions appearing at the onset of an attack of gout or diabetes must be included with the group of toxic convulsions and these, according to Binswanger, have nothing in common with true epilepsy.

The theory of the etiologic connection of epilepsy with profound disorders of metabolism and the phenomena of auto-intoxication compels me to incorporate a discussion of this genetic association which attempts to refer epilepsy in general to processes of auto-intoxication. Zeni is the latest author to draw conclusions of this kind from his theory of the auto-cytotoxins and anti-auto-cytotoxins. The foundation of all of these views is the previously mentioned investigations of metabolism. In regard to the toxicity of the urine, Féré and Voisin reached contradictory results, one finding a hypertoxicity and the other a hypotoxicity of the urine after the attack. I should like to point out that the toxicity of a urine, according to the latest researches of Schumacher, depends largely upon its concentration, and its specific gravity. Oliviero and Voisin isolated from the urine of an epileptic an exceedingly toxic product having an odor of ammonia and nutmeg, which, when injected into the circulation of animals, produced violent convulsions. Krainski, to whom we owe comprehensive studies of metabolism in epileptics, found that defibrinated blood, taken during a seizure, when injected into animals produced convulsions, but that taken during the period free from attacks did not have this result. The same conditions were found by Cololian. Krainski demonstrated a constant relation between the excretion of uric acid and the epileptic seizure, in the sense that a change in the excretion of uric acid precedes the attacks; this is the more marked the more severe the succeeding attack. Haig considered the retained uric acid as the toxic agent which produced the convulsion. Krainski holds ammonium carbamate, which originates from uric acid, responsible; this substance being normally used for the synthetic formation of uric acid. As a matter of fact, according

to the researches of Krainski, disproportionate quantities of carbamic acid, as well as of ammonia, were found in the blood of epileptics, the quantities being the greater the severer the attacks. On the other hand ammonium carbamate when injected into animals produced serious nervous symptoms (somnolence, epilepsy, tetanus). It may be stated, in regard to these investigations of metabolism, that the proof has not yet been furnished that they actually provoke the epileptic seizure and that we are not concerned merely with the resulting phenomena of the epileptic charge and discharge. Binswanger believes that this is due directly to a disturbed chemism of the nerve cells, but the effect of the motor discharge must also be considered. But even in the first instance, that altered metabolism is the direct "*agent provocateur*" of the epileptic discharge, the dependence upon periodic nervous disturbance (change in the activity of secretion of the kidneys) must be considered. But even this would not relegate the conception of the epileptic alteration out of existence; besides these disturbances of metabolism are present only in some epileptics (Hebold-Bratz, Binswanger). Binswanger holds that it is not impossible to separate this group pathogenetically as toxemic epilepsy from the other varieties. In this view he approaches the opinion of Féré to which in the main he is seriously opposed, namely, that what we regard to-day clinically as genuine epilepsy is still further divisible etiologically.

In addition to the chronic diseases mentioned above a number of special affections of the organs of the body must be included which are in etiologic connection with epilepsy. Among these are: the lungs, the heart, the liver, the pleura, the ear, the genitalia, the stomach and the intestines. Here *reflex epilepsy* (which will be considered later on) comes into play. Provided there is not an accidental coincidence of these maladies with epilepsy they are probably only the exciting factors, the predisposition being already present. In patients with cardiac disease, apart from reflex causes, the effect of circulatory disturbances must be considered. Whether all epileptiform conditions (Stokes-Adams disease) are to be included with epilepsy appears very doubtful. In gastro-intestinal diseases toxic influences are present. Early and prolonged masturbation is probably only a predisposing cause and besides must be looked upon as a neuropathic stigma. Among the chronic intoxications there are to be mentioned: alcoholism and chronic lead poisoning. Both may be considered not only indirect but direct factors giving rise to epilepsy by damaging the germinal plasma. *Alcoholic epilepsy* and *lead epilepsy* are usually considered special forms. Alcohol may occasionally act as a predisposing cause. Excessive use of tobacco is sometimes the cause of an attack, but it has been stated that chronic nicotin poisoning may directly institute a seizure. Among other poisons there are to be mentioned: carbonic oxid, chloroform, ether, cocain, antipyrin, camphor. Their etiologic relation to epilepsy is, however, very doubtful.

Trauma plays a much more important rôle in the etiology, especially that form which affects the head; but other varieties than those associated with severe bodily shock may result in epilepsy. When trauma to the head produces grave anatomical lesions, such as hemorrhage, meningeal hemorrhage, depression of bone, the picture of ordinary epilepsy may be clinically present. Under these circumstances it occasionally happens that a constant localized

aura or a post-paroxysmal sign of absence of function indicates the lesion and its seat. As a rule there are uniform, partial convulsions. In other cases of traumatic epilepsy there is no sign, at least macroscopically, of an anatomical lesion (whether or not there are minute miliary hemorrhages in the cortical substance cannot be answered). Again the fact must be stated that trauma may only be the predisposing cause. Among the cases due to trauma of the head the unique case of Breitung's must be included: epilepsy developing after the application of douches to the head. Psychical trauma must be included with bodily trauma; fright, and mental overexertion, as trauma with protracted action. These are probably not exciting but predisposing causes. That with bodily trauma, psychical trauma (fright), which accompanies it, may also be in etiologic relation, must be considered.

Reflex epilepsy belongs to the category of traumatic epilepsy. In regard to this variety we must mention the animal experiments by which epileptic convulsions were produced by various procedures acting upon the peripheral nervous system. These experiments are intimately connected with the name of Brown-Séquard who produced epileptic convulsions in guinea pigs after dividing the sciatic nerve; similar convulsions were produced by injuries to the spinal cord and later Westphal succeeded in causing them by blows upon the skull. These convulsions have certain characteristics, firstly, their appearance is associated with the irritation of definite zones, the so-called *epileptogenous zones*, which develop in the animal that is operated upon within the first few days and which may be observed externally (falling out of the hair, anesthesia). If a zone of this kind is irritated there arise, during the first period, reflex movements in the animal in the form of scratching and resistance in the limbs of the same side. Later these reflex actions are increased to general convulsions, of which it is questionable, whether they are associated with loss of consciousness. The convulsions in man, which are regarded as reflex epilepsy in the restricted sense, present certain analogies to those produced in the animal. A lesion of a peripheral nerve trunk is common to both (after injuries to, and diseases of, the spinal cord in man convulsions have been noted which may be included under the conception of reflex epilepsy). Often in man there is the formation of traumatic neuromata, embedding of nerve trunks in cicatricial tissue, etc. (perhaps also cicatrices in the skull). An epileptogenous zone is also present in these cases, for pressure upon the cicatrix often produces an attack. These attacks are still further characterized by the fact that after a sensory aura, which involves the injured part, an isolated tonic tension appears during which consciousness remains normal; later this tonic contraction also involves the other extremity upon the same side and finally the opposite extremities; loss of consciousness and the other criteria of the genuine epileptic attack are then added. Binswanger does not discuss the question whether or not in these convulsions there is a step-like progression from the level of the centers in the spinal cord to the cumulate centers in the medulla and from these to the cortex of the brain, or whether the old medulla theory is to be invoked to explain this group of epileptic attacks, according to which loss of consciousness is a consequence of irritation of the vasomotor center in the medulla oblongata and that the cortical anemia consequent upon this occurs simultaneously with the stimulation of the motor medullary centers. The

conception of reflex epilepsy has been extended to a series of cases which probably have in common only the existence of a peripheral irritation. This irritation, in the cases in question, probably has but a predisposing influence. In addition to the affections enumerated above I must add: dental caries, anomalies of refraction (?), polypi of the ear and larynx, adenoid vegetations, phimosis, intestinal parasites. The last named are said to give rise to epilepsy by the production of a toxin. The formation of spontaneous epileptogenous zones in man is exceedingly doubtful.

There are many accidental causes for the appearance of an attack when the affection exists. In great part they have been already mentioned; bodily as well as psychical overexertion is an important factor. In cases of nocturnal epilepsy physiologic sleep is regarded as an accidental cause.

PATHOLOGY

I may be very brief in regard to the *pathological anatomy.* All lesions in focal diseases or severe cortical changes with simultaneous idiocy naturally need not be considered. Sclerosis of the hippocampus major, for a time, was considered of great importance; it was shown, however, that this sclerosis is not present constantly in the brain of epileptics and, again, that it is present in persons who have never suffered from epilepsy. Oppenheim is of the opinion that it is an unimportant stigma of degeneration. Recently attention has been directed to minute lesion in the cortex of the brain of epileptics (destruction of nerve cells, proliferation of glia fibers in the cerebral cortex, Chaslin, Alzheimer, and others). It is very doubtful whether these histologic changes can be regarded as the cause of the epileptic attack and that they are, therefore, merely sequels of the existence of the affection; just as this is self evident of many pathologic lesions found in the brain of persons perishing during the attack or in the *status epilepticus.* The lesion discovered by Schröder van der Kolk also belongs to this category. Binswanger regards it as a step backward in pathophysiologic knowledge if all disturbances of nerve function, even the exceedingly minute expression of activity of the central nervous system, is constantly and exclusively referred to gross lesions of the nervous substance. He also maintains that at the present time a causal pathological lesion has not been demonstrated. Liepmann is of the opinion that there is no pathologico-anatomical lesion in epilepsy, a view that every unprejudiced investigator must accept, in spite of the many lesions that have been reported in the brains of epileptics.

DIAGNOSIS

The diagnosis, in the greatest majority of cases, is very easy, provided we adhere to the characteristics of the attack described previously and consider that epilepsy is a chronic disease.

According to Binswanger, infantile convulsions may be differentiated from epilepsy by a preliminary stage of motor unrest or of somnolence associated at first with isolated contractions in the muscles of the face, the clonic stage, a tonic stage appearing later. In addition eclamptic convulsions show a tendency to accumulative appearance. Ziehen regards the preponderance

of the tonic spasm as characteristic. There is usually the demonstration of some etiologic factor (dentition, gastro-intestinal affection, fever) which serves as a differential criterium.

When there are convulsions resembling epilepsy, especially if they occur in later life, the possibility of another underlying affection must be considered. Oppenheim calls attention to cases in which a brain tumor revealed its presence by the symptoms of epilepsy which were present for years. In such instances the brain tumor (like any other focal disease) became the exciting cause of the epileptic attacks. In other instances it is the intracranial pressure accompanying the brain tumor which is the cause of the epileptic attacks. Here the epileptiform attacks accompanying cerebral syphilis must be included. The toxemic convulsions in gout and diabetes must be remembered. Dementia paralytica not rarely runs its course with epileptiform spasms; usually these are only incomplete or of the Jacksonian type; but we have already mentioned some cases in which the attacks closely resembled genuine epilepsy. These may precede the appearance of the paralysis for years. Epileptiform attacks also occur in multiple sclerosis. These references are sufficient to indicate the importance of making a careful examination of every patient with epileptic attacks. It may always be regarded an incomplete examination if an ophthalmoscopic investigation of these patients is neglected.

Jacksonian Epilepsy.—Nor is the recognition of the JACKSONIAN FORM difficult. Here the convulsions, clonic contractions, occur in a circumscribed muscular region and are gradually distributed, in analogy with the anatomical situation of the motor cortical centers. There is either entire absence of loss of consciousness or this occurs only after the convulsions have attacked the other half of the body. Difficulties can only arise if the convulsions are unilateral or if a motor aura precedes them; and even here the early loss of consciousness will indicate the correct diagnosis. There are intermediate forms which correspond neither to the epileptic attack nor to the Jacksonian variety but present a complicated picture. These cases are always suspicious of an organic foundation and here we find the post-paroxysmal circumscribed symptoms of absence of function which were detailed in the description of the symtomatology.

The *apoplectiform attacks* of epileptics may cause confusion with *syncopal conditions*. Here we must accurately note the isolated spasm of the respiratory muscles, the cyanosis which results and which is so common in this condition. The person in syncope is pale, there is pronounced cardiac weakness, the pulse is feeble and rapid; in the epileptic attack the pulse is full and tense. We must never omit an inquiry as to the prodromes of the loss of consciousness. In epilepsy there is often the characteristic aura; in syncope there is a sensation of weakness and nausea. Quite characteristic and of some value in differential diagnosis is the common appearance of an outbreak of cold sweat prior to an attack of syncope. The sweating at the beginning of an epileptic attack is usually associated with intense heat. In doubtful cases the involuntary evacuation of urine favors epilepsy. Nevertheless, Oppenheim hesitated in a case in making a diagnosis of epilepsy: A woman with mild vasomotor disturbance suffered from attacks of unconsciousness prior to her menses, accompanied with involuntary evacuation of

urine and feces. He believed cerebral anemia, due to vasomotor disturbance, to be the cause and this opinion seemed to be correct for no other attacks took place. At this point we must mention the so-called "laughter-stroke" of Oppenheim (*Lachschlag*); a condition in which there is a brief loss of consciousness after excessive laughter. We must bear in mind that excessive laughing may occasionally be a predisposing cause of an epileptic attack. *Ménière's disease,* included by Féré with epilepsy, may readily be differentiated from an epileptic attack on account of its very characteristic symptom-complex, even when associated with unconsciousness. There is often difficulty in the differential diagnosis between *arteriosclerotic epilepsy* and of the *vasomotor climacteric neurosis*; such difficulties arise when arteriosclerotic epilepsy occurs as an attack of vertigo combined with slight mental dullness and introduced by sensations of ascending flashes of heat. The vasomotor attacks of climacteric neurosis are not associated with loss of consciousness; an examination of the mental condition is very important in these cases for in climacteric neuroses intelligence is normal, while in arteriosclerotic epilepsy there are commonly the signs of beginning arteriosclerotic mental feebleness. Binswanger states that in the clinical picture of neurasthenia there are psychical attacks of inhibition with symptoms of motor irritation and those of absence of function; these are liable to cause confusion with *abortive epileptic seizures.* In one case there were vertigo, difficulty in speech, spasm of the jaws, dimness of the field of vision, pallor of the face, but without loss of consciousness. The original diagnosis of epilepsy proved to be incorrect.

The differentiation of epileptic and hysterical attacks is of the greatest importance; this, in very many instances, is by no means easy. Even the prodromes (increased irritability) and the aura of the hysterical attack (*globus*) occasionally present great similarity to epilepsy. Féré believes the diagnosis of hysteria to be assured when the globus rises from the ovarian region; the fall, in the hysterical attack, is more gradual so that severe injuries do not result, so that the presence of an injury in a doubtful case is in favor of epilepsy. Many hysterical attacks present a so-called epileptoid phase, a phase of tonic tension of all of the muscles of the entire body, in which the respiratory muscles may be implicated and in consequence cyanosis may result. If this epileptoid phase is succeeded by *clownism, arc de cercle, attitudes passionelles, dream-like delirium,* or if any of these phenomena appear alone, the character of the attack becomes clear. Even if the convulsions of this clonic stage are not excessive they nevertheless frequently bear the character of voluntary movements. Rhythmically these patients protrude the tongue, throw the head backward, ball the hand into a fist, throw their arms about, etc. The physician must be cautious not to regard the automatic coördinated movements which sometimes appear in atypical or rudimentary epileptic attacks as hysterical. These clonic convulsions, in hysteria, are often associated with crying, scolding, etc. A rhythmical protrusion of the abdomen (coitus movement) is very characteristic. According to Féré this may occur during the interval of a series of epileptic attacks. The greatest difficulty arises in those cases in which the epileptoid phase presents all the characters of the hysterical attack; all the symptoms which have been described as appearing in the epileptic seizure may also be found

in hysteria: complete loss of consciousness, rigidity of the pupils, biting the tongue, involuntary evacuation of urine. On the other hand pupillary rigidity may be absent in epilepsy. It may, however, be stated in general that pupillary rigidity, biting the tongue and involuntary evacuation of urine are exceedingly rare in hysteria and in a doubtful case are in favor of epilepsy. On the other hand it is not rare for hysterical patients to bite and gnaw their lips during an attack. Even when the tongue is bitten, it may be noted that this is due less to a decided bite upon the margin of the tongue than to surface injury from rubbing and friction of the tongue against the teeth. In doubtful cases an attempt should be made to produce an attack by pressure upon a hysterogenic zone or to abort a paroxysm by this means. Among the post-paroxysmal signs small hemorrhages behind the ear and in the conjunctiva are in favor of epilepsy. The condition of the phosphates in the urine, described previously, may be utilized in the differential diagnosis. Attacks of vertigo with momentary loss of consciousness, which resemble epileptic *petit mal*, appear also in hysteria, according to Féré. There are also attacks which present distinct features of the epileptic as well as of the hysterical convulsion; these are known as *hysteroepilepsy*. Féré points out that commonly one of the components, either the epileptic or the hysteric, is prominent in the attack. Those cases are also designated as hysteroepilepsy in which, in addition to undoubted epileptic seizures, conclusive hysterical paroxysms appear; in regard to the first mentioned category as well as to these cases it may be taken as a rule that they are subjects with the signs of severe neuropathic predisposition. The occurrence of hysteroepileptic attacks in the first mentioned sense, is, however, still a disputed question.

Malingering.—Finally the occurrence of *simulation* must be considered. Of the most recent instances of this kind I must mention the case reported by Leubuscher to the Society for Neurology and Psychiatry. The epileptic seizures were simulated by this individual with great accuracy even to biting the tongue. Of especial note in this case was the duration of the malingering. In a suspicious case pallor of the face, cyanosis and the reaction of the pupils must be observed. Voisin lays special stress upon a study of the pulse curve; Féré upon the pneumographic curve.

If we are in a position to observe an attack the diagnosis is much easier. But if it is necessary to content ourselves with the description of an eye witness all the details should be insisted upon and the intelligence of the witness must be taken into consideration. We should search for the remains of an attack: cicatrices upon the tongue, other injuries, the small hemorrhages appearing after an attack and all the other phenomena of a nocturnal attack.

TREATMENT

The therapy of epilepsy must follow causal factors. The treatment of a coexisting affection that is in etiologic relation to epilepsy is self evident. Such maladies were enumerated in the description of the etiology; I shall only mention diseases of the genitalia and of the ear, carious teeth, adenoid vegetations, phimosis. Unfortunately we cannot always succeed in relieving the epileptic alteration by the removal of these causes; what we can avert are the exciting factors. A permanent cure is, therefore, not to be expected.

The conditions are more favorable in reflex epilepsy due to peripheral cicatrices. Here operative removal of the source of irritation, excision of the cicatrix, provided it is done early enough before an ineradicable diffuse condition of irritation appears in the cortex of the brain, cause the attacks to disappear completely. It is, therefore, advisable, in these instances, not to lose valuable time with useless internal treatment.

In regard to traumatic epilepsy after injuries to the skull (in so far as this is not included within the meaning of reflex epilepsy) there is only an indication for operation when there are undoubted partial convulsions of the Jacksonian type which point to a cortical lesion, and in those cases showing the picture of ordinary epilepsy, when there is depression of bone or the previously mentioned symptoms (characteristic aura, post-paroxysmal signs of absence of function) which render the presence of an anatomical lesion likely. The chances are the more unfavorable the more remote the trauma and in consequence the more the epileptic change has had an opportunity of imbedding itself. Surgery has shown but very few enticing results in genuine epilepsy, no matter whether the operation consisted in tying the carotid artery, resection of the sympathetic, trephining or ventricular drainage. These cases are, therefore, to be regarded as a surgical *noli me tangere.* Here must also be included symptomatic epilepsy after diseases with cortical foci, when the pure picture of Jacksonian epilepsy is not present.

The therapy of (genuine) epilepsy consists in a careful regulation of diet and of the entire manner of living. Alcohol is a dangerous poison for every epileptic. Coffee, tea, bouillon (on account of the large amount of creatinin), strong spices and tobacco are prohibited; the food should consist largely of vegetables and much milk (in the country a milk diet may be employed). With nocturnal attacks, light food is to be given at night. Recently complete withdrawal of table salt, following the advice of Richet and Toulouse, has been tried; these authors pointed out that with the withdrawal of table salt much smaller doses of the bromids are sufficient. With this treatment it is necessary to see that there is no salt in the bread; if there is great repugnance on the part of the patient the food may be salted with small quantities of sodium bromid. The patient must be protected from all corporeal and psychical injuries. For this purpose it is necessary that the patient have a suitable occupation which neither causes physical (bodily exertion, heavy work) nor mental (great responsibilities) overexertion. Masturbation and sexual excesses are to be prohibited. In mild cases, in which there are long intervals between the attacks, an attempt may be made to treat the patient by diet and the aid of general hygienic means, perhaps also by cautious hydrotherapy. As a rule, however, in order to obtain good results it is necessary to combine the dietetic treatment with the administration of the bromids. This is still the sovereign remedy in the treatment of epilepsy. Potassium bromid, sodium bromid or ammonium bromid, or a combination of the three salts may be administered in doses of from 4–8, exceptionally from 10–12 grams daily. In children and in young persons the dose of bromid must be calculated according to the weight of the patient (0.1 gram of potassium bromid to the kilogram). Erlenmeyer's bromid water and Sandow's effervescent bromid salt enjoy a great reputation. More recent substitutes are bromalin (in doses of 1–2 grams several times daily in powder) and 10%

bromipin (a tablespoonful contains 2 grams of potassium bromid). The latter is a solution of the bromid salt in sesame oil and has at the same time a nutrient value, besides *bromid acne* is said to be less likely to arise. For the latter condition there may be employed prophylactically, warm baths, soaping the entire body with soft soap, regulation of the bowels, etc. A mild grade of bromid acne does not indicate the immediate withdrawal of the bromid; an attempt may be made to combat the condition by the administration of small doses of Fowler's solution. It is sometimes necessary to continue the bromid treatment for years and all that is required is to guard against the appearance of severe symptoms of intoxication (sleepiness, cardiac disturbance, disappearance of the corneal reflex). According to Flechsig the good effect of the bromid is increased if a six weeks' treatment with opium precedes the administration of the bromid (extract of opium gradually increased until 0.9 are taken daily). The opium-bromid treatment may be combined with that suggested by Voisin (cool baths; beginning with medium temperatures and while shortening the duration of the bath decreasing the temperature of the water about 1½° F. daily). In *senile* (arteriosclerotic) *epilepsy* it is well to combine the bromid with a cardiac tonic (digitalis, strophantus, adonis), for example in the form of Bechterew's prescription (inf. adonid. venal. 2.0:180.0, codeini phosphor. 0.1, natr. bromid 10.0. M. D. S.: tablespoonful 3 times daily). Among the substitutes for the bromids there may be mentioned: belladonna (extr. belladonnæ, fol. belladonnæ pulv. āā 1.0, succi liquor. q. s., ut fiant pilul. No. 100. 2–6 pills daily), atropin (0.0005 3–5 times daily in pill) and borax which has been recently recommended (3–5 grams of natr. biborac. in solution). Radix artemisiæ and zinc oxid are obsolete remedies. During the attack we may limit ourselves to the protection of the patient so that he does not injure himself; it is advisable to place a cork between the teeth, if possible, to prevent the tongue from being bitten. To abort the attack the following means have been tried: constriction of the member from which the aura arises, administration of a tablespoonful of common salt or the inhalation of a few drops of amyl nitrite. In the *status epilepticus* chloral hydrate (2 grams), or amyl hydrate (7 grams), by the rectum are advisable. For example 2 grams of chloral hydrate are given by the rectum and after each succeeding attack 1 gram.

In the preceding description I have given a presentation found in the majority of text-books: that genuine epilepsy is a well founded uniform disease with definite clinical symptoms to be strictly separated from other symptomatic convulsions (uremia, convulsions due to cerebral pressure). From the standpoint of clinical description such a conception certainly has decided advantages. I cannot close my discussion without again referring to the uncertainty still prevalent as to whether what has been mentioned is actually in accordance with the facts and must, therefore, again call attention to Féré's opinion. Féré states the following:[1] "A separation of epileptoid affections from the great trunk of the neuroses always makes itself felt when a new circumstance is recognized which is capable of producing the symptom-picture of epilepsy. The realm of so-called idiopathic epilepsy is restricted

[1] Ebers's translation.

from day to day while that of symptomatic epilepsy is constantly being enlarged. At the present time we can no longer employ the designation idiopathic epilepsy as synonymous with epilepsy without cause, but only for epilepsy with an unknown cause. No uniform, definite, definable pathologic change can be stated as a cause for epilepsy, but, on the contrary, we must regard the view as well founded that the most variable manifestations of epilepsy may be referred to very different functional damages or alterations. Therefore epilepsy can no longer be considered a uniform pathologic picture but only as a group of symptoms."

If we may apply the valuable animal experiments of Nothnagel, of Hitzig, of Binswanger, of Ziehen, and others, to the conditions in man—and this we may certainly do—the material for the mysterious mechanism of the epileptic attack is present in every brain, and also in the healthy brain and need not be produced by a morbid process. How does it happen that in one person fortunately this material is not utilized and in another this baneful force is set into action? There are only two possibilities: either irritation of an unusual kind or of unusual strength must act upon the brain, or the wave of irritation for the occurrence of the mechanism must be situated particularly low down. Both possibilities appear to be present in the pathology of epilepsy (I am now employing this expression in its wide sense for all epileptiform attacks). On the one hand we note the appearance of epileptic attacks, if the view is justified, when an abnormally powerful stimulation irritates the brain. I need only refer to the appearance of such attacks in the different acute intoxications. Perhaps toxemic epilepsy belongs to this category, although Binswanger with his conservatism declines to regard it as genuine epilepsy. In this condition the explanation why toxic products are formed in the organism still remains unanswered provided we do not care to assume that the normal organism occasionally develops toxic products and, as I must add, eliminates them without further damage and symptoms. It would follow that to this group uremic attacks also belong in which epileptiform convulsions appear, which in no wise can be differentiated clinically from genuine epilepsy. Uremic convulsions would then be nothing else than acute epilepsy, in Féré's sense. In regard to the second possibility we note the presence of epileptic attacks in cases in which irritations are present which would not affect the normal organism. I need only refer to the processes of puberty, the action of fright, and the like. Here a deep position of the wave of irritation must be thought of, and an increased faculty for convulsions, a *spasmophilia,* which is perhaps nothing else than the stigma of a congenitally deficient nervous system or one that has become so during the life of the individual. The eclamptic convulsions of infancy would belong to this group, in which two factors appear to play a rôle, first the increased tendency to convulsions in early life, therefore, a physiologic deep position of the wave of irritation, and secondly the neuropathic predisposition, therefore, a pathologic deficiency of the wave value. Whether the epileptic attacks recur, whether they show a cumulative effect or whether they do not present themselves again depends upon the greater or lesser facility with which this irritation becomes liberated in the central nervous system. An increase of this faculty will cause many attacks and even the most insignificant stimulus, as it were, a microscopic irritation will bring

about a seizure. This is probably a similar condition to that which arises in the patient with a psychopathic predisposition, his ideas constantly evoking constrained conceptions. Whether for this group of "*spasmophiles*" it is necessary to assume a special epileptic change is questionable. Both possibilities would naturally be in correlation to each other in the manner that the deeper the wave of irritation is situated the slighter the stimulation necessary to produce the epileptic attack.

All diseases of the brain might show their effects in two different directions, first in that they give rise to abnormal irritation, and this is especially true of focal affections, and second that they involve the entire nervous system in the sense of decreasing its power and in this manner bring about the increased tendency to convulsions; focal diseases perhaps especially in relation to their surrounding areas. In favor of the latter possibility, in regard to focal diseases, is the clinical fact that often in old cases of encephalitis Jacksonian seizures appear under the influence of a peripheral irritation and after removal of the source of irritation these attacks disappear. Jacksonian epilepsy differs from general epilepsy only by the fact that the irritation is local or by the decrease of resistance being localized. The intimate connection of both forms of epilepsy is favored by the close association of their clinical phenomena. But also the epileptiform attacks in the most varied diseases of the brain, in dementia paralytica, in multiple sclerosis, regarded from the same point of view, would have to be included with epilepsy. There would be no difference between genuine and symptomatic epilepsy, *sensu strictiori* there would be no such disease as epilepsy but only an epileptic attack.

HYSTERIA

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AMONG the *organic* diseases of the nervous system a precise differentiation of the individual forms gives rise to the greatest difficulties. These are further increased as we approach the *functional* neuroses, for here pathological anatomy does not aid us. Therefore it is not surprising that for many functional neuroses there is as yet no accurate limitation and definition. This is particularly true of the special functional neurosis, **hysteria**, that we are about to consider. As pathological anatomy does not aid us and, as we shall soon see, no uniform and exclusive cause for hysteria can be demonstrated, we are compelled to search for a comprehensive *pathophysiologic* stand-point under which all, or at most the majority, of functional disturbances can be grouped so as not to limit ourselves to a simple detailed account of its main symptoms and their relations. Such pathophysiologic definitions, in functional nervous diseases, must replace the pathologico-anatomical foundation. In fact such a uniform pathophysiologic stand-point may be recognized in the case of hysteria. We may say: Hysteria is characterized by an abnormal accentuation of emotional activity. These accentuated emotional conceptions are, in themselves, primarily normal; but while in the healthy individual such increased emotional conceptions are restricted to a relatively limited field in consciousness, sensation, thought and action, in hysteria these bounds are exceeded: an accentuated conception of fear of paralysis of the arm actually produces the paralysis, the accentuated emotional impression severs the play of motive and is discharged in impulsive actions or in spasmodically exaggerated expressions of movements. Favored by this increased activity it is transformed into hallucinations and illusions or it perverts the normal recollection into illusions of memory; for the time being it dominates the disposition and the emotions to an extent such as never exists in the normal person. Nor is the reverse lacking: non-accentuated emotional conceptions, in hysteria, are of abnormally feeble effect; thus the absentmindedness and many other symptoms of hysteria, which we shall encounter later on, may be explained. An attempt has often been made to characterize hysteria by the "*psychogenous*" nature of its symptoms. And it may be readily seen why this was done. The corporeal symptoms of hysteria are in fact psychogenous, but this designation is not sufficiently restricted; the bodily symptoms of melancholia are also psychogenous. We must state specifically: the corporeal symptoms of hysteria depend upon the abnormal increase of emotional activity. But even in another respect the definition of hysteria as a disease consisting of psychogenous symptoms is inadequate: it is too par-

tial, favoring the bodily symptoms while ignoring the psychic ones. Besides the psychogenous character of the symptoms, the increased power of *suggestion* has been pointed out as a main factor in hysteria. If we understand by this increased power of suggestion not only a heightened susceptibility for foreign suggestions but above all an increased effectiveness of autosuggestion this view approximates very closely that of our own. It is simply less accurate in so far as, firstly it extends the conception of suggestion unduly, and secondly it fails to emphasize the emotional excess of the abnormally active impressions. The increased power of suggestion is merely one of the most important auxiliary phenomena of the abnormal increase of activity in the emotional impressions according to which, in our opinion, the clinical picture of hysteria is dominated.

However, our own view requires a very important amplification. The exaggerated emotional impressions, of which increased activity is in general characteristic of hysteria, need not by any means be *actual* ones, also the so-called *latent* memory pictures, or, on the contrary, particularly the latent memory pictures, in so far as they are exaggerated emotional ones, present a pathologic increased activity. To make this important fact clear I shall quote the observation of a psychologist. He meets a gentleman every morning with whom he is unacquainted, and to whom he never bows. One morning he meets him again and bows to him. He is astonished on account of his unmotived greeting. The cause is soon found: he dreamt, during the preceding night, that he had had a prolonged friendly conversation with this gentleman. This memory apparently had a lingering effect. At the moment it did not *actually* come into consciousness, only the *latent* memory picture was active. There are numerous examples which show us the activity of such latent memory pictures in actual psychical life. Our entire process of thought and our complete emotional life depend upon the actions of such latent impressions. But I expressly chose the example just related because it may serve as the prototype for the pathologically increased activity of latent memory pictures in hysteria. It shows us the conspicuous effect of a single latent emotional impression quite isolated in the midst of normal life. In hysteria analogous actions accumulate to a pathological degree and extend their sphere of action very much further. Again, while in normal life an occasional prominent lingering effect—as in our example—is corrected at once, in hysteria this correction does not take place on account of the much greater activity of such emotional conceptions.

I recently had an opportunity of observing a very characteristic example of the importance of these *unconscious* emotional impressions. A girl, aged 18, with a hereditary predisposition and presenting all of the typical symptoms of the hysteric, psychopathic constitution, while studying art (which she enjoyed very much), showed a special preference for the "creation of Adam" as depicted by Michael Angelo in the Sistine Chapel. One day she noted that she assumed a constrained position. Only subsequently did it become clear to her that this position accurately corresponded to the very characteristic one which Adam occupies in this picture.

It may perhaps be held that in the view just developed hysteria actually becomes a psychosis. This is, in fact, correct and we shall soon convince ourselves that the *origin* of all or nearly all symptoms of hysteria are psy-

chical, and that, therefore, the psychic condition in hysteria is to be primarily considered. On the other hand hysteria differs from the psychoses in the ordinary sense to a considerable extent; the psychogenous symptoms, therefore, the expressions of the abnormal activity of emotional impressions, are much more diffused in the corporeal region even than is the case in the true psychoses. In addition to this the symptoms of hysteria may be especially referred to *latent* memory pictures while in the psychoses *actual* memory pictures have a pathologic effect. This will become clear at once if we contrast hysterical paralysis with the hypochondriac fear of paralysis in the many psychoses. The pathologic effect of the *impression* of paralysis is unmistakable in both instances but at the same time the difference becomes obvious: in hysteria there is no knowledge of such an impression of paralysis, there is a *latent* conception which develops an abnormal activity; the condition is reversed in those hypochondriacs in whom the actual impression of paralysis is the decisive element; the patient concerns himself day and night with this thought and even speaks of his fears of paralysis. At the same time we may convince ourselves of the great difference in the *sphere of action* in both cases; the latent conception of paralysis in the hysteric produces severe *corporeal* symptoms of paralysis; *it completely excludes the region of the paralyzed part from the range of actual associations while the psychical life remains relatively but little influenced*; on the other hand in hypochondriac conditions the actual impression of paralysis, in the various psychoses (and also in hypochondriac neurasthenia), influences the *psychical* processes, the mood, etc., to an intense degree; the *corporeal* symptoms, however, to such an insignificant extent that actual paralyses and similar conditions are rarely noted.

Finally, the uniform pathophysiologic concept which we have obtained of hysteria prepares us for still another important, principal fact. Although hysteria is characterized, in general, by the morbid, exaggerated activity of emotional impressions this does not exclude the fact that symptomatically, to a certain extent as an addition, a similar pathologically increased activity of emotional conceptions may not occasionally appear in other diseases of the nervous system. This is confirmed by experience. We find in the various functional and especially in the organic affections of the nervous system, for example, in brain tumor, in dementia paralytica, etc., occasionally *"superimposed" hysterical symptoms.* Sometimes these are so marked that we must speak of *"superimposed hysteria."* This observation, therefore, coincides with the principle which we have developed.

With this we have gained a general view-point and at the same time a consistent start. Naturally our subsequent description must show that this stand-point is correct and proper. At all events I may state at once that it is serviceable if we approach the great variety of hysterical symptoms with a guiding thought.

ETIOLOGY

Unquestionably the most important causal factor of hysteria is the *hereditary predisposition.* I have found this in over one half of all the cases; slight predisposing factors in the ascendant not being included. Very often there is a *marked* hereditary predisposition. Here hysteria is, to a certain

extent, in contrast to neurasthenia, for in the common forms severe hereditary predisposition plays no important part. The fact is particularly noteworthy that the predisposition in hysteria is conspicuously often inherited from both parents: father and mother of the patient having suffered from hysteria. Briquet in the following sentences has expressed himself very explicitly: "Individuals who are descended from hysterical parents show a twelve times greater predisposition to hysteria than others," and, "for the daughter of a hysterical mother being attacked by hysteria the probabilities are more than one quarter." But here it must be remembered that besides the direct hereditary influence imitation of the pathologic copy and an improper education are also active. This inheritance from both parents is by no means unusual. We recognize quite a number of analogies in which a psychopathic form of reaction is inherited from both parents: thus the tendency to pathologic depression, melancholia, is exceedingly apt to be inherited; we may, therefore, readily comprehend that the peculiar psychic mode of reaction in hysteria, the abnormally increased activity of emotional impressions, may also be inherited from both parents.

In the description of the course of hysteria we shall see that the pronounced symptoms of hysteria may develop in earliest childhood, or that a "psychopathic constitution," in the sense of hysteria, may precede the outbreak of symptoms for years, in fact may be present from earliest childhood. The frequent combination of hysteria with a hereditary psychopathic constitution, with or without debility, becomes clear in this manner.

Age also has a predisposing influence. We have just seen that in hereditary cases the first symptoms may appear early in life, even from the third year. The tendency increases as puberty is reached. I, as well as others, have treated numerous school-children with well developed hysteria. The maximum of morbidity occurs during the period of puberty and the following decades. After the 30th year there is a rapid decrease in the number of cases, which is only concealed in the male by the common occurrence of the *traumatic variety* of hysteria. Exceptionally the affection is also noted in the presenile and senile periods. For the most part a careful examination of these presumable forms of presenile and senile cases of hysteria reveals that the condition is either a relapse or an exacerbation of a former attack of hysteria, or that the hysterical symptoms are only superimposed, upon *dementia arteriosclerotica* or senile dementia. I must advise extreme caution in making a diagnosis of hysteria that is not superimposed if hysterical symptoms appear for the first time in the presenile or senile period.

The predisposing influence of *sex*, especially of the feminine, was formerly often exaggerated; nevertheless it cannot be doubted. According to my experience, excluding the cases concerned with suits for damages (the traumatic) the hysteric morbidity in the female is about 5 times as great as that of the male. In my opinion this predisposition may be explained from the fact that the characteristic manner of psychical reaction of hysteria is decidedly closer to the normal psychical type of woman. It cannot be explained from an unequal distribution of acquired etiologic factors in the two sexes; for, I have convinced myself that in the hereditary infantile cases and those appearing at puberty, in which an acquired etiologic factor as yet plays no rôle, that the female is much more commonly attacked.

In place of hereditary predisposition other deleterious factors may appear at any age and in either sex. Among these there must first be mentioned *prolonged psychical influences* which aid the development of an abnormal mode of mental reaction. Faulty education is one of the most important of these, for it neglects the duty of teaching the child to control its emotions and to keep its imagination in check. Association with hysterical persons may act in the same manner. The danger of attending *spiritualistic séances*, by which the expectation is actually pitched for the experience of abnormal emotional conceptions, is equally dangerous. It is obvious that all of these deleterious psychical factors are much more harmful in predisposed individuals, but I know of quite a number of cases in which these prolonged injurious psychical factors have produced typical hysteria where there was no demonstrable predisposition.

Intense emotional expectation, probably in addition to fright, is the essential factor in *traumatic hysteria*. From our knowledge of traumatic neuroses we know that on account of suits for damages the patient observes his own condition with exceeding care, particularly in regard to sequels of the accident; in addition to this there are also intense emotional impressions in regard to the financial consequences of the accident. It is, therefore, readily conceivable that trauma gives rise to pathologic conditions by means of these prolonged psychical influences which are either hysteria itself or are very closely allied. All of these facts become even of greater importance when we compare how slight the effect of other prolonged psychic influences is, such as sorrow, anger, and the emotional experiences of the constant struggle for existence, as well as intellectual overexertion.

The importance of *acute psychical influences*, especially of *fright*, must be estimated quite differently. Fright rarely causes hysteria, but it awakens a "slumbering" hysteria, either in that hysterical symptoms were absent previously (which I regard as exceedingly rare), or that the previously existing hysterical symptoms were relatively slight. With Charcot we may regard fright as an "*agent provocateur*" of hysteria. More frequently fright only causes the appearance of some new symptom, as a hysterical disturbance of speech, or a hysterical paralysis (for example in the arm, the patient having fallen upon this member); or even a modification of some of the old symptoms. In regard to *trauma*, in so far as it is associated with fright, a new etiologic relationship to hysteria is shown. In this connection it is immaterial whether the trauma consisted of a severe head injury, or a box upon the ear, an earthquake, or a lightning stroke. Psychical trauma is the decisive factor. It may even be that in such cases bodily trauma is entirely lacking.

Those cases are also to be referred to psychical trauma in which the outbreak of hysteria arises from *imitation*, from the observation of a spasmodic attack, or of chorea, etc., in a fellow pupil, an acquaintance or the like. The symptoms, then, often resemble the spasmodic convulsions. This "*imitation*" *hysteria* appears most frequently in children; occasionally in this manner *small epidemics* arise.

The etiologic relations of hysteria to definite, *material*, that is, *corporeal injury* is not so clear. And among these trauma would also come into question in so far as it is associated with shock. The question as to whether

there is pure, traumatic *shock-hysteria*—independent of the chronic psychical factors mentioned above with which trauma is commonly combined, and independent of traumatic fright—has not yet been decided. According to different observations, in which neither sorrow nor fright were combined with trauma and severe shock, I must answer this question of the rare occurrence of true shock-hysteria in the affirmative.

The importance of *chronic intoxication*, especially by alcohol and lead. is also a disputed question. Cases occasionally do occur, in which, without a demonstrable predisposition or cause, there develops, upon the basis of alcohol, lead, or morphin, a typical hysterical symptom-complex in addition to the symptoms characteristic of these poisons. In part these cases are to be included with superimposed hysteria. The same is true of *diseases of metabolism* and of the *chronic infections*, such as malaria. Both factors play only a very subordinate part in the etiology of hysteria.

Nor can an important part be assigned to *bodily exhaustion*. As important as is the latter condition in the development of neurasthenia and especially of many neuroses, so rarely is it involved to any considerable extent in the development of hysteria.

Great significance was formerly attached to the *female genitalia*. Even to-day the view is very prevalent that displacements of the uterus, chronic parametritis and endometritis as well as chronic oöphoritis are capable of producing *reflex hysteria*. Probably in all of the instances, in which there is actually a connection, the psychical influences caused by the genital affection are the active etiologic factors. The same is most probably true of the influence of castration and the climacterium. In both of these conditions we do not note the symptoms of hysteria, but pathologic pictures, which must be included partly with the vasomotor neuroses, partly with the vasomotor form of neurasthenia.

The other "*reflex*"-*hysterias* hardly deserve the name. We can only say that some few hysteric symptoms occasionally appear from peripheral irritation in a reflex manner. Thus in rare cases a superficial or deep cicatrix remaining from a trauma in an individual who has hysteric manifestations may be associated with clonic spasm in the neighboring muscles, which upon superficial observation appears to be reflex.

Much more protean is the

SYMPTOMATOLOGY

of hysteria which we shall now consider.[1] It is best to subdivide the symptoms of hysteria according to their occurrence in definite attacks or as to whether they are more or less permanent. The former are called paroxysmal symptoms (occurring in the attack), the latter symptoms of the interval or permanent symptoms. The prototype of the first is the ordinary hysterical spasmodic paroxysm. The permanent symptoms may be subdivided into psychical and somatic. The symptoms of the attack and especially of the hysterical convulsion are so intimately associated, consisting of psychical and somatic ones, that an analogous subdivision is not advisable.

[1] In women the examination should always be undertaken in the presence of a relative, a nurse, or of another physician.

I shall begin with the permanent, somatic symptoms, for these are calculated to show us the mechanism of the hysterical symptoms in the plainest manner.

Among the

PERMANENT SOMATIC SYMPTOMS

the signs of degeneration are least interesting. In correspondence with the frequency of a marked hereditary predisposition they are observed much more commonly in the hysteric than in the normal person; but they are not characteristic, for they are also noted in other neuroses and psychoses in which a marked hereditary disposition plays a rôle.

On the other hand we are in the midst of the characteristic hysterical symptoms as soon as we pass to the hysterical

DISORDERS OF MOTILITY

and among these we must primarily consider hysterical

Paralyses.

We have already informed ourselves in regard to their general pathophysiologic origin, from accentuated emotional impressions, and, as a rule, from latent emotional impressions. Often the origin is quite clear, as if after a fall upon the left shoulder there is paralysis of the left arm or after a fatiguing march there is paralysis of both legs. The momentary loss of speech or of power in the legs, which sometimes accompanies the effect of fright in the normal person, produces a permanent difficulty in speech or a permanent paraplegia, etc., in the hysteric. In other cases it is impossible to determine the latent conception by which the paralysis has been brought about.

The *distribution* of the paralysis is usually very characteristic. It appears in connection with the popular impression of the divisions of the human body and is independent of the anatomical grouping of the motor tracts and centers. The right, or the left arm, the right, or the left hand, the right, or the left leg, the right, or the left foot correspond to this unsophisticated conception of division. If two extremities are paralyzed these are commonly the arm and leg of the same side or both legs. The combination of both extremities of one half of the body coincides with the naïve conception of symmetry and is associated with the popular idea of an attack of apoplexy. The inclusion of both legs is apparently grounded upon their common activity in walking and standing. Thus we are able to understand that paralyses limited to a portion of the extremity as well as *monoplegias, hemiplegias,* and *paraplegias* appear in hysteria.

However, these forms of paralysis, in which, in the course of a special region, all conscious movements are arrested, present only *one* type of hysterical paralysis. A second type merely reveals the cessation of a special conscious *movement or of a group of these,* for example, of speech, walking, or standing, while the muscles, which do not function during this act, are normal under other conditions. Correspondingly we refer to hysterical *aphasia,* hysterical *abasia* and *astasia.* Nor do these paralyses correspond with any

degree of accuracy to the functional grouping of the centers in the central nervous system. In this second type the naïve ideas of the divisions of our body are not decisive for the paralysis, but merely the naïve performances of our important bodily movements. The paralyses of the first type may be designated as *psychogenous regional paralyses*, those of the second type as *psychogenous functional paralyses* or *paralyses of coördination.*

In regard to the two types the following points must be noted:

The *paralyses of the first type*, therefore, the *regional paralyses*, as a rule, do not involve the muscles supplied by the facial nerve, nor those of the tongue and eyes. The movements of the head and trunk are mostly normal. The limitation of the paralysis is generally dependent upon the previously mentioned naïve ideas. Thus, it occurs that in a regional paralysis of the hand the loss of power is not infrequently limited directly to the movements of the hand, while motion in the neighboring joints is quite intact. In the same manner we may explain the non-involvement of the leg upon the same side in a hysterical monoplegia of the arm; an occurrence which is rarely met with in an organic monoplegia.

Hysterical hemiplegias, paraplegias, and monoplegias, betray their psychogenous origin in still other directions, particularly in their instability. In contrast to the organic form these paralyses may improve suddenly under special circumstances, for example, under excitement a slight movement of gesticulation may appear in the paralyzed extremity, or being in danger of falling the patient puts out the hand for protection. Oppenheim has noted that by careful manipulation of the hysterical paralysis, now and then a slight contraction may be produced; that a slight condition of innervation may be noted under these circumstances I am in a position to confirm. Finally it is very significant that if the patient is under the influence of alcohol or of an anesthetic (both at the beginning of narcosis as during the early period of awakening) the paralyzed extremity may be normal in action. It is obvious that in all of these cases the pathologic action of the latent emotional conceptions is either compensated for or inhibited for the time being.

In many cases the paralysis is not absolute; on the contrary, there is a more or less marked *paresis.* In these instances it is particularly noticeable to what extent the intensity of the paralysis is under the influence of psychical conditions. The merest suggestion, such as directing attention to the paretic extremity, is sufficient to increase the paresis. If the condition concerns a hysterical hemiparesis it will be noted that *the gait* does not correspond to that of organic hemiplegia: the tip of the foot is almost never dragged and even less frequently thrown out into a convex arc, but is dragged in an almost straight direction upon the entire sole, or upon the lateral borders of the foot.

The *muscles of the tongue and those supplied by the facial nerve* are rarely involved in the paralysis, especially in the hemiplegia. Such a condition is sometimes simulated from the fact that the hemiplegia is occasionally accompanied with a crossed contracture of the muscles of the tongue and those innervated by the facial nerve. Only in the rarest instances is true paresis present. Naturally care must be exercised so as not to confound slight congenital asymmetry of the facial innervation with paresis. A case

of true, distinct paresis of the tongue, which I observed recently, revealed marked deviation upon protrusion, but it was quite characteristic that the paresis, therefore, the deviation, became uncertain when the patient observed the condition in a looking-glass. I was convinced that the conception of right and left became confused by the image in the glass, the paralysis thereby losing its definite character.

In hysterical paralysis the *muscles of the eye* are rarely involved and an isolated paralysis of hysteric origin is exceedingly infrequent. Only very exceptionally have I noted a paralysis of associated lateral movements of the eye to one side, which was due to a masked suggestion in the course of the examination, the paresis being characterized by the factors previously mentioned. For example the patient does not follow a light held before him but with an unexpected noise there is a lateral movement of the eye. A hysterical paralysis upward and outward occurs in quite an analogous manner. In rare cases there is a hysterical paralysis of all movements of the eye so that *ophthalmoplegia externa* is simulated. A true paralysis of convergence is somewhat more frequent which is also characterized by its inconstant nature, for example, its absence upon automatic fixation. I have never noted paralysis of accommodation without paralysis of convergence in hysteria. In isolated cases there is unilateral or bilateral ptosis. As a rule this condition is simulated by a spasm of the orbicularis oculi; it is then a case of hysterical *pseudoptosis*. True unilateral ptosis is exceedingly rare. It has been stated that in hysterical paralysis there is not so much actual paralysis as simple relaxation of the levator palpebræ. This view is undoubtedly correct, but it is equally true of all other forms of hysterical paralysis. At the same time it explains in an apt manner the limitation of hysterical paralyses: hysteric paralyses only occur in such muscle groups as are innervated voluntarily and which may, therefore, be voluntarily relaxed. The external rectus, which we cannot innervate isolatedly, we also cannot relax isolatedly and, therefore, we never observe hysterical paralysis of this muscle. The few cases of isolated hysterical paralysis of the external, or internal rectus, that have been reported in literature, will not stand the light of scientific criticism. Regional hysterical paralyses are nothing else than functional paralyses; they differ only from functional paralyses, in the restricted sense, that in well developed cases they involve *all* of the motor functions of a portion of the body.

The hysterical paralyses of the ocular muscles reveal this transition in a very demonstrative manner: hysterical paralysis of all bulbar muscles is a regional paralysis, which corresponds to the popular conception of the relation of the eyes and is absolutely analogous to hysterical paraplegia, while a unilateral hysterical glance-paralysis is a paralysis of function, which may be compared, for example, to hysterical abasia.

Quite an analogous condition is met with in the course of innervation of the palate, pharynx, and vocal cords. The innervation of the palate or pharynx may be arrested or decreased by hysteric paralysis, but here also in the inconsequent manner and form which is indicated by its psychogenous origin. Hysterical paralyses of the vocal cords are even more frequent. It is necessary to be cautious so as not to regard every hysterical loss of voice (aphonia) as paralysis; very often this aphonia is dependent upon peculiar

symptoms of spasm or of incoördination which will be described more fully later on. However, even after leaving these cases out of consideration there are enough instances of hysterical paralyses of the vocal cords in which the genuine psychogenous origin is obvious. The patients declare that it is impossible for them to speak aloud, and in fact upon laryngoscopic examination it is noted that upon attempts at phonation the normal movement of the vocal cords is lacking. In my experience this "paralysis" is always bilateral and symmetric. The tension of the vocal cords is normal and only in very rare, exceptional cases is there relaxation so that in inspiration and expiration they appear to fluctuate with the current of air. In contrast to organic paralyses it is very significant that only the constrictors and not the dilators of the glottis are involved.

A general factor of all hysterical regional paralyses is the *non-involvement of the reflexes.* The tendon and periosteal reflexes in the paralyzed member are not greater than in the homologous non-paralyzed one. But care must be exercised that we do not mistake a subsequent non-reflex increase as an exaggerated reflex. Occasionally even foot clonus may be simulated in the paralyzed extremity in this manner. The same is true of the periosteal reflexes. Only after a hysterical paralysis has persisted for many months may there be an actual change in the tendon and periosteal reflexes, a condition that I have positively observed. The behavior of the cutaneous and mucous membrane reflexes is somewhat more complicated. It is of clinical interest to differentiate between true reflex and *resistance reflex.* Plantar flexion of the toes upon stroking the sole of the foot is a true reflex; dorsal flexion of the foot, flexion of the thigh, etc., upon stroking the sole is a resistance reflex. In the hysterically paralyzed portions of the body the true reflexes are normal, not differing from those of the homologous non-paralyzed extremity, while the resistance reflexes are either decreased or abolished. This is also the reason why the Babinski reflex is never noted in hysteria. In testing the reflexes of the skin and the mucous membranes many errors are liable to occur. Thus exceptionally in hysterical cases the plantar flexion of the toes of the normal sole reflex is suppressed by an antagonistic contraction of the extensors of the toes; in others, exceptionally, there is dorsal flexion of the toes as a resistance reflex leading to simulation of the Babinski reflex.

Quite as characteristic is the behavior of the *electric contractility* of the hysterically paralyzed muscles; there is no change from the normal. Only in rare cases, after the persistence for years of a hysterical paralysis, is there a very slight quantitative decrease to direct and indirect galvanic and faradic contractility. Nor does mechanical stimulation of the muscles reveal a characteristic change.

In general there is no atrophy of the paralyzed muscles. Inactivity atrophy develops much later than in organic pyramidal tract paralysis. Only in very exceptional cases is marked atrophy noted in hysterical paralysis: this presents no severe and especially no qualitative alterations of electric contractility. The *isolated functional paralyses* of hysteria are of great importance; their definition has already been given. As a rule the most common functions are involved, viz., walking, standing, sitting, speech. Accordingly we refer to hysterical abasia, hysterical astasia, hysterical akathisia,

and hysterical motor aphasia, that is, anarthria. The latter is often incorrectly designated as *hysterical mutism*; it is better to reserve this term for those psychical, conscious processes upon which the silence depends. Hysterical abasia is commonly but not invariably associated with hysterical astasia. In typical cases this hysterical abasia-astasia is very characteristic: in the recumbent posture and in sitting these patients are able to execute all movements of their legs with ease and with normal power; but so soon as they are placed upon their feet "the knees bend as if made of cotton." If the condition is one of isolated abasia, standing is normal but the patient is not able to move a step; the soles of his feet appear to cling to the floor. Usually abasia is a permanent symptom, i. e., an attempt to walk is impossible as soon as the act is undertaken. I am familiar with some cases in which a hysterical patient, in the midst of normal walking, was taken with an attack of abasia and sometimes also with astasia. These are hysterical attacks of abasia and astasia. Naturally it is of the greatest interest, in these cases, to determine the accompanying psychical condition. In my experience *conscious* fear—for example of falling—rarely plays a rôle, in other words, actual hypochondriacal conceptions are rarely involved. But the conscious impression of not being able to stand and not being able to walk is not present as a rule; the patients are often surprised themselves by these conditions. If the patient is questioned as to whether she still knows how to stand and walk, the answer is sometimes that she does not; more frequently she states that she still knows how, but that the limbs refuse to act. This is in entire accord with our theoretic considerations at the beginning of this article. The circumstances are analogous with hysterical mutism: rarely do the patients maintain—in the sense of hysterical "aphasia"—that they no longer know the word (word in the motor sense); much oftener they state—in the sense of a hysterical "anarthria"—that the organs of speech refuse function.

We may still further specialize these functional hysterical paralyses. Thus I have seen hysterical patients who were incapable of enunciating some special letter of the alphabet, such as *r*, while all the other letters were articulated in a normal manner.

It is obvious that in these cases of isolated hysterical functional paralysis, strictly speaking, we are not concerned with a psychogenous paralysis, but with the psychogenous absence of function of some definite coördination.

Intimately associated with hysterical paralyses are the hysterical

Contractures.

As is the case with paralysis, hysterical contracture often arises in association with trauma. In some instances severe emotional shock is the accidental cause. Sometimes contracture arises immediately in connection with this, occasionally there is a longer or shorter period of incubation. Often the contracture is the result of a hysterical convulsive attack. Less frequently, as is also the case with hysterical paralysis, is it associated with the hysterical somnambulistic condition. Contracture is often accompanied with pain, or sensations, which, on their part, represent another hysterical symptom or some complicating peripheral affection (caries of a tooth, an

unguis incarnatus, etc.). I shall refer to apparent reflex contractures later on.

Hysterical contractures of the extremities like paralysis of the extremities may be monoplegic, hemiplegic or paraplegic. The distribution to the individual muscles and the position which may result is subject to great variation. Sometimes an organic contracture may be simulated in the most astonishing manner. In other cases the bizarre form of the contracture denotes its hysterical nature. Occasionally these contractures give an impression of coördination; there is an appearance as if the extremity had become rigid in the midst of a coördinated movement (Gorgon contracture).

Often the contracture is limited to a single portion of an extremity; isolated contractures of a hand or foot are not rare. In such instances the regional character, i. e., the limitation based upon the naïve conceptions of the divisions of the body, is easily recognized. Specialization in some instances may even go further than this; thus I know of cases in which only the thumb or the second to fourth fingers were involved by the contraction. It is unnecessary to describe the individual contractures; the variation is so great that a brief description is impossible, although it is true that some postures are more common than others (making a fist, position of writing, crutch posture of the leg, etc.) or that some are especially conspicuous (hyperflexion of the wrist, hyperextension of the ankle, etc.). Hysterical contracture may simulate tetany; as in these cases Chvostek's and Trousseau's signs may be temporarily present, the danger of an error in diagnosis is very great. Curschmann quite properly emphasizes that the increase of electric contractility of the motor nerves is the only sign which decides in favor of genuine tetany.

The intensity of these hysterical contractures upon superficial examination is hardly less than that of the organic form; on the contrary they appear to be more marked and almost extreme in comparison. Accurate investigation, however, shows that when the attention is diverted the contraction sometimes becomes decidedly less marked, while inversely if attention is aroused, for example, upon an attempt at passive motion, the condition is aggravated. In contrast to an organic contraction—in so far as special suggestions are not active—it does not lessen in a warm bath. During sleep the severest hysterical contraction may disappear completely, while marked organic contractures are less obvious but by no means abolished. It has also been correctly observed that hysterical contraction does not lessen if the point of origin and the insertion of the contracted muscle are approximated, while an organic contracture, under these circumstances, shows a certain degree of relaxation; in my experience there are many exceptions to this rule. Under the influence of an anesthetic hysterical contracture disappears completely, and even after the patient awakens from the narcosis the contracture reappears relatively late. Strong cutaneous irritation often increases the contraction.

Very interesting results are obtained by the examination of the degree of voluntary movement in the extremity attacked by the contracture. It has previously been stated that a hysterical contracture is often associated with hysterical paralysis in the same extremity. But this is by no means invariable; occasionally it is conspicuous—again in contrast to organic contracture

—that the contracted muscles present no evidence of paresis, as is easily shown by an attempt to overcome the contracture by passive motion. In the numerous instances in which contracture is actually accompanied with complete paralysis, the relation, in regard to time, of the contracture and the paralysis is noteworthy. While in organic paralysis early contracture is exceptional it may be looked upon as a rule that hysterical contracture and hysterical paralysis appear simultaneously or almost at the same time.

Just as there are *latent* organic contractures analogous hysterical conditions also occur. It is noted in hysteria that while the patient, in general, presents no contractures any severe cutaneous irritation is capable of producing one. This so-called *diathèse de contracture* is sometimes limited to one extremity, occasionally involves all extremities, and even, as I may state in advance, affects the trunk and the head. In place of cutaneous irritation, pressure upon the nerve trunks or upon the muscle brings about a similar result. The tendon reflexes are as little influenced by hysterical contracture as by the paralysis, apart from the purely mechanical decrease in the excursus of the reflex movement dependent upon the contracture.

Next in interest to contractures of the extremity are those involving *the muscles supplied by the facial nerve.* In well marked cases this appears in association with crossed hemiplegia or hemiparesis. Sometimes the condition is present alone. The branches of the nerve supplying the cheek are commonly most severely affected. As a rule the orbicularis oris is not involved nor the orbicularis oculi; this is also the case with the frontalis. A very characteristic facial expression appears. In one of my patients the somnambulistic states were always accompanied with a right-sided hemiparesis and a left-sided incomplete contraction of the muscles supplied by the facial nerve. At first glance it might be supposed that the condition was one of right-sided facial paralysis, but as soon as the patient was required actively to innervate the facial nerve distribution it was noted that the right side was entirely intact. Actual hysterical facial paralysis is rare (for example, in addition to crossed facial contracture). Another circumstance indicates to some extent that the condition is one of left-sided spasm. Upon careful observation we may observe clonic contraction at irregular intervals, sometimes also fibrillary tremor in the left-sided facial muscles. Not always, but quite frequently, this facial contracture is associated with a contracture of the muscles of the tongue upon the same side. This is noted from the fact that upon protrusion of the organ it deviates to one side, in left-sided contracture to the right, upon right-sided contracture to the left. Therefore a right-sided deviation of the tongue indicates a contracture of the muscles of the tongue upon the left side. That there is a contracture and not a right-sided paralysis of the tongue may be noted from the attempt to place the tongue in a median position when great resistance is encountered. Sometimes the deviation of the tongue may be noted while the organ is at rest upon the floor of the mouth; simultaneously the contracted half of the tongue appears smaller and more arched. Rarely in addition to unilateral facial contracture and that of the tongue there is a unilateral contracture of the muscles of mastication which may even be more prominent; under these circumstances when the mouth is opened the lower jaw may deviate (with right-sided contracture to the left, and *vice versâ*). More often there is

a symmetric contracture of the muscles of the jaw, with hysterical trismus, as an isolated condition.

In addition to the contractures that have been described there are also isolated contractures in the region of the eyes and forehead that are supplied by the facial nerve. Isolated contracture of the orbicularis oculi which the inexperienced physician regards as ptosis is, as a rule, dependent upon hyperalgesia of the optic region or to hyperesthesia. This will be described later. A unilateral contracture of the frontalis, which I have noted to be sometimes transitory, at other times more permanent, is independent of sensory disturbances; it may be associated with a crossed contracture of the orbicularis oculi and then produces a very peculiar contortion of the face.

Contractures also occur in the ocular muscles. According to my experience they are always associated ones, for isolated muscles which we cannot innervate singly never show hysterical contracture. Convergence contracture is most frequent, therefore, a contracture of both recti interni; or an abduction contracture, therefore, a contracture of both recti externi. In the latter case the axes of both eyes are parallel and immobile. In the former case a paralysis of the abducens, in the latter an oculomotor paralysis, may be simulated; that is the picture of a complete bilateral *ophthalmoplegia externa*. The difficulties of diagnosis may even be increased by the addition of true hysterical paralyses of the ocular muscles, such as were previously described. True diplopia and paralysis of the external eye muscles are rare in these hysterical contractures, on the contrary hysterical diplopia is, as a rule, monocular. Only exceptionally does there appear to be slight asymmetry, or on the contrary a congenital asymmetry of innervation in hysterical contracture and paralysis of the ocular muscles; then true diplopia may be present.

Contracture of the levator palpebræ is much less frequent. Isolated spasm of the ciliary muscle—with or without megalopsia—is sometimes observed; occasionally it is associated with convergence contracture. In my experience contracture of the sphincter iridis and of the dilator pupillæ are very rare; there is absolutely no reason to regard the very common mydriasis, which we find in hysteria as well as in other neuroses, as a contracture.

Contracture of the muscles which rotate the head, a tonic hysterical *torticollis*, is not rare. The associated nature of the contracture may here also be readily determined. Many cases which are now described in literature as "*mental torticollis*" belong to this category.

Contractures in the trunk appear especially in the extensor muscles. For the most part these are unilateral, or at least one side is especially involved, and this condition, therefore, produces deviation of the trunk which may simulate *scoliosis*. In addition to the muscles of the back, the glutæi may be involved in this contracture. Hysterical *pseudoscoliosis* is distinguished from genuine scoliosis by palpation, which, in the case of the latter, reveals a deformity and change in position of the vertebræ and ribs, and in the former only a spasmodic contraction of the muscles of the back.

In addition to contractures we also see other motor irritative phenomena, particularly *clonic spasms*. In so far as these are limited to the hysterical attack they will be described later on. Here we are concerned only with those that appear in addition to the hysterical attack, being more or less continuous.

Clonic Muscular Spasms

Hysterical myoclonus cases may be divided in a similar manner to hysterical paralyses, according to whether they involve the muscles of a special region (in the sense of the naïve conception of the divisions of the human body) or according to the muscles which act conjointly in a definite, isolated function or coördinated movement; but here also there is no sharp line of demarcation.

Regional clonic spasms in one or more extremities is very rare. But I have seen such cases, in which, for example, an arm or a hand revealed continuous clonic spasms for weeks without there being a possibility of recognizing a definite coördination of the spasmodic movements. Like all clonic muscular spasms of hysteria these also show a tendency to paroxysmal increase; sometimes there is actual intermission in the attacks. As the clonic spasm sometimes changes into the tonic form, just as inversely clonic contractions appear in an extremity which is the seat of hysterical contracture, the most varied and mixed pictures of contracture and myoclonus are presented.

Regional muscular spasms are much more common in the *area supplied by the facial nerve.* If, as is still very commonly done, all clonic facial spasms are designated as *tic convulsif,* this regional muscular facial spasm as it appears in hysteria may be designated hysterical tic convulsif. While true tic convulsif always depends upon an abnormal irritative condition within the lower reflex arc (trigeminal-facial, optic-facial), due to organic or reflex causes, the hysterical tic is due to psychogenous motor stimulation, in which perhaps peripheral irritation may be active, but which at all events does not arise simply in the course of the lower reflex arc. This is at once seen during the examination, for all organic symptoms are absent in the distribution of the facial nerve, while in the case of true tic convulsif they are common (disturbance of electric contractility, abnormal associated movements, such as distortion of the angle of the mouth upon forcibly closing the eyes, etc.), further from the fact that the relation which true tic shows to blinking is absent or nearly so, and, finally, that under psychical influences, emotion, under observation, suggestion, etc., it varies to a much greater extent than does the genuine tic. It is much more closely allied to tic impulsif of the facial region, such as we observe as an accompanying symptom of the *maladie des tics.* Besides the latter is often merely a symptom-complex belonging to hysteria. Sometimes the clonus attacks principally the orbicularis oculi, sometimes the muscles of the cheek; the muscles of the chin and of the forehead are less often affected and the orbicularis oris with exceeding rarity. Combination with tonic, facial spasm, facial contracture, occurs quite often. It is quite remarkable how often these clonic, facial spasms appear in traumatic hysteria. In this condition they are often voluntarily exaggerated. As a rule the individual contractions are quite uniform and limited to one half of the face. I have seen them exceptionally with regular intermissions even appear in both sides of the face.

Hysterical clonus of the muscles which rotate the head, therefore, a hysterical clonic torticollis, or, we might say, a hysterical tic impulsif of the head movements, is quite common. Many clonic forms of mental torticollis belong

to this category. Clonic spasm of the muscles of the tongue and jaw are less frequent.

Clonic spasm of the diaphragm, hysterical singultus, is a very common condition. Occasionally it appears in paroxysms, at other times it is almost continuous for days; it always ceases during sleep. It is frequently associated with hysterical disorders of respiration, which will be described more in detail later on, or it alternates with these. Not uncommonly is it accompanied with clonic contractions of the muscles which rotate the head and also by those of the muscles of the jaw. Clonic spasm of the abdominal muscles (hysterical *belly-dance*) is sometimes observed.

In addition to these *regional* clonic spasms there are clonic spasms resembling *coördinated movements.* It is usual no longer to employ the designation clonic to such spasmodic movements; we shall, therefore, refer briefly to localized coördinated spasms. These appear in any region of the body. I shall quote a few examples.

I am treating a lady who suffers from severe carcinomatous metastasis of the right brachial plexus. Palpation and the results of the neuro-pathologic examination leave no doubt of the diagnosis (partial DeR, etc.). For some time peculiar clonic, and, as she states herself, involuntary spasms have appeared in the right arm. She throws the right arm to and fro in a very complicated manner. These spasmodic, coördinated movements may be produced, arrested, and transformed by suggestion. Undoubtedly, as other characteristic hysterical symptoms, stigmata, are present, the condition is one of a serious organic disease which has been overshadowed by a hysterical symptom-complex.

In other cases these coördinated hysterical muscular spasms imitate some "occupation" movement; and, even more frequently, they are noted as the result of emotional expression or as a movement of resistance. Under these circumstances they cannot be sharply separated from the previously mentioned tics. Hysterical coughing and barking must also be included. The former may appear in paroxysms, sometimes almost continuous. Hysterical barking imitates the various cries of animals. A peculiar inspiratory stridor is also occasionally noted.

Besides, these coördinated spasms interfere with normal movements and particularly disturb the regular rhythm of the respiration. Most commonly there is a simple *acceleration of respiration* (*hysterical tachypnea*), but the rhythm of the respiration may be altered in a very much more complicated manner: thus there are sighing inspirations, concomitant distortions of the face and rotation of the head, etc. *Disturbance of the normal gait* by *accessory,* coördinate, spasmodic movements is even more common. The patients hop in a peculiar manner or they walk with a peculiar, bizarre gait (*gait-stuttering,* etc.). If we add that these spasmodic disturbances in gait may be associated, in the most varied manner, with hysterical paralyses and contractures of the legs, the enormous variety of hysterical disorders in walking, which we encounter clinically, will become manifest. It is not rare for hysterical abasia to run through some of these complicated changes in gait in the course of recovery.

Almost quite as common are analogous disturbances of normal *speech* from coördinative, spasmodic movements. Hysterical *stuttering* is most frequent. If we consider the acoustic aspect it resembles the genuine stuttering

neurosis, for it also consists of an interruption of the continuity of speech which is noted in individual letters or—much more common than in the true neurosis—in separate syllables. The stuttered letter or syllable is prolonged, at the same time there is an abortive effect, i. e., there is an incomplete, final articulation, but on the other hand it may be repeated several times successively. In hysterical stuttering, just as in the genuine form, certain letters are especially affected; as a rule the disturbance is more general than in the true neurosis. The processes of innervation, superficially considered, are almost identical to those of the true neurosis. Only a careful examination reveals differences. The hysterical stutterer shows the defect as a rule—provided there are no special suggestive influences—in whispering and in singing just as much as in ordinary speech, in contrast to the genuine stutterer. It is also significant that the repetition of the same word does not reveal the same favorable influence as in the true neurosis. The prolongation of the vowels does not favorably influence hysterical stuttering as is the case in the genuine variety. Common to both is the increase under excitement. The accompanying respiratory disturbances and the tic movements are sometimes quite similar.

Hysterical stuttering may occur in connection with all of the other hysterical symptoms after a longer or shorter period of latency, in connection with psychical shock or other accidental causes; sometimes analogous to the disturbances in gait, it is a transitional symptom which may be noted during convalescence from hysterical paralysis of speech. The inverse is also true, hysterical stuttering may be a prodrome of hysterical paralysis of speech.

Here also spastic, hysterical aphonia must be included: as soon as the patient attempts to talk a tonic spasm of the constrictors of the glottis appears which prevents the production of voice and even gives rise to dyspnea. Speech disturbances of higher coördination, such as *coprolalia*, are much less common. In one case of well developed hysteria I observed this symptom in the manner that each hysterical eructation—I shall recur to this spasmodic symptom of hysteria—was accompanied by the expression of some indecent word or phrase.

Another peculiar. motor, irritative symptom is hysterical

Tremor.

This symptom presents many varieties in hysteria. We may state at once that there is no form of tremor which is not occasionally imitated by the hysterical form. It occurs in any intensity and in all degrees of rapidity. Only a tremor of slight excursus and, on the other hand, of great rapidity as well as a very slow one is rarely noted. Upon an attempt at voluntary suppression there is often an increase. Sometimes the tremor is most pronounced during rest; at other times it is the static, then again the locomotor, tremor (the latter is also designated intention tremor) which is most marked. Accordingly the tremor of paralysis agitans, the tremor of chronic intoxication, the tremor of multiple sclerosis are imitated. In my experience the tremor is often combined with a mild but psychogenous tension of the affected extremity. Like the clonic muscular spasms, described above, the

tremor may be paroxysmal, at other times continuous, during the entire waking period. Sometimes it is limited—and this appears to me to be the most frequent variety—to an arm or both arms, at other times it attacks a leg or both legs. A tremor affecting only one or both legs is quite rare. A strict, regular rhythm is infrequent; on the contrary, it is usual for the tremor to be subject to great variations both as regards excursus as well as *tempo*. I have often noted that after a series of 5–10–15 marked and rapid movements there is a remission which is again succeeded by a new series of active tremor movements, just as if the patient were starting anew.

Remarkable combinations of the tremor with paralysis and contracture are noted. Here Charcot's *trepidante abasia* must be mentioned. Finally it must be stated that hysterical tremor upon an increase of the excursus may be transformed into the previously described clonic spasm. Thus hysterical tremor of the eyelids cannot be sharply separated from the blepharoclonus that has been mentioned, and even when the tremor arises in the extremities there is no well defined limit. Characteristic of all fórms of hysterical tremor is its dependence upon psychical conditions, particularly upon exaggerated emotional impressions and upon suggestion. Naturally in utilizing this criterium we must be particularly cautious in judging tremor, for non-hysterical varieties of tremor, even those due to organic lesions, are often greatly influenced by the emotions. The most certain method appears to me to consist in an intellectual and sensory diversion (listening to the ticking of a watch which is gradually removed to greater distances from the ear, having the patient spell his name backward, etc.), this method often proving valuable. If such diversions bring about a marked decrease of the tremor this favors the hysterical form. A single examination of the tremor is never conclusive, and all the other symptoms must be taken into consideration. For many years I have employed the graphic method in investigating the tremor, and I must emphasize that this process is not sufficient to decide with certainty whether the tremor is hysteric or not. Simulated tremor especially cannot be differentiated from the hysterical variety in this manner.

Hysterical chorea may easily be differentiated from hysterical tremor by the irregularity of the movements. Sometimes the choreiform movements are distributed over the entire body, at other times they are unilateral or are at least most prominent upon one side. Exceptionally they are increased to the severest jactitation.

Disturbances of Coördination

resembling ataxia are rare. When they are noted they are due almost entirely to sensory disturbance and will, therefore, be described later on. Only in one case did I observe an ataxia which was not due to sensory involvement; in another case the diagnosis—hysteria or a beginning cerebellar disease—remained doubtful. Naturally I do not here take into consideration the numerous cases in which, by special suggestion, such as the finger-nose test when the patient is not instructed with sufficient caution, an ataxia is directly provoked.

Not quite so varied but diagnostically just as important are the hysterical

SENSORY DISTURBANCES

I shall begin with the disorders of the contact and pain senses. According to their distribution we differentiate three main varieties:

(1) The areal or insular type;

(2) The unilateral type;

(3) The geometrically limited, or as I prefer, the regional [1] type.

Insular, sensory disturbance, in my experience, is the rarest variety. Sometimes there are hypesthetic, anesthetic or hyperesthetic islets; occasionally it is impossible to decide whether the condition of these areas, or the condition of the surrounding parts, is pathologic, whether we are dealing with a hyperesthetic area upon a normal base or with a normal islet upon a hyperesthetic base and *vice versâ.* Often a sensory alteration in the islet may be in contrast with the sensation of the neighboring parts. With relative frequency there is only a sensation of pain instead of an alteration in contact: then we are dealing with relative or absolute hypalgesia or analgesia, that is with hyperalgesic areas. Finally, contact and pain senses are altered to the same extent. The size of the islets varies within wide limits. In general the extent of a single islet increases proximally. In my experience the diameter varies between 1 and over 10 cm. The shape is sometimes almost circular, at other times elliptical. Occasionally the areas are close together, again they may be separated by some distance. The outline, the diameter as well as the situation, are nothing less than constant. Only when a structure of the surface of the body, such as the nipple, forms a starting point, is the islet more constant; in this case the areal sensory disturbance passes into the *regional form* which will soon be described. The influence of suggestion is very great upon this insular, sensory disturbance. We often gain an impression that only by a comparative examination of the sensation of neighboring areas, in the sense of a concealed suggestion, is the insular sensory disturbance produced. At times the areas are distributed over the entire body, at other times they are unilateral or are found merely upon the trunk, etc.

If the condition is not one of anesthesia but of hypesthesia, it is of interest to determine the errors in localization which the patient makes in indicating these areas. As a rule these are considerable, but it must be stated that in a large number of contacts, now and then, very slight errors occur, while in the case of simulation these mistakes are always very great.

Unilateral sensory disturbance is much more common. Here also the condition may be hemihypesthesia, hemianesthesia, hemihyperesthesia, that is, hemihyperalgesia, hemianalgesia, or hemihyperalgesia. Here also it is sometimes difficult to decide which half of the body shows the pathologic sensory disturbance: we can merely determine the contrast, only a relative sensory disturbance. I ordinarily compare pricks of a pin, punctiform contact with a fine head of a pin, strokes with the point and head, the comparison being made repeatedly upon the left and right sides of the body. A pair of compasses may be employed with advantage upon the trunk, in the face, upon the hands and feet, both points being placed upon the part simultaneously.

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[1] It is advisable in my opinion to reserve the designation "region" for the limitations here proposed by me.

Better results may even be obtained with the pendular esthesiometer. A unilateral sensory disturbance is commonly limited by the median line of the body, i. e., the limit varies as many centimeters from the median line as the error amounts to which the normal person would make if he were required to indicate the median line with his eyes closed. The reports, according to which the sensory disturbance is accurately defined by the median line belong to the realm of fable. The psychogenous origin of the hysterical sensory disturbance is revealed by this fact. Often the mucous membranes are involved in this unilateral disturbance, as well as the deeper parts, such as the fascia, muscles, periosteum, joints, etc. Occasionally some parts are excluded; thus a smaller or larger area adjacent to the median line, or the hairy scalp, may be exempt. On the other hand the sensory disturbance may extend beyond the median line. These limitations as well as extensions are included in the regional sensory derangement which will soon be described. A pure, unilateral, sensory disturbance is only a special instance of the regional form which we separate for purely practical reasons. Unilateral sensory derangement is somewhat more refractory to suggestion than the insular variety, but it is much more persistent. Apparently the idea of the "right or left side" of the body is a much firmer support than the conception of insular areas. Nevertheless the influence of suggestion may be demonstrated in many cases. Thus it is possible—quite apart from hypnotic suggestion—by placing a magnet or a watch, etc., or by stroking accompanied with suitable suggestion which need not always be verbal and explicit, to transfer the sensory disturbance from one side of the body to the other. This process is known as *transference,* and I must add that a similar transference is also possible with the previously described motor symptoms. The psychogenous origin of the unilateral, sensory disturbance occasionally shows itself when pin pricks, that are unexpectedly used for testing the hemianalgesic side, produce movements of resistance or flight. We may also often note that even unexpected pin pricks do not give rise to pain or that the pain sense is greatly decreased. It is interesting that—like most of the other unilateral symptoms of hysteria—the unilateral disturbance is found more often on the left than upon the right. This is probably explained from the fact that on account of a decreased employment the conception of non-use is the more obvious; it must also be considered that in fact it is represented by fewer motile, tactile and other conceptions in the cortex of the brain. Finally, it is possible that left-sided disturbances are less disagreeable than right-sided ones; there are actual facts which favor the view that factors of this nature are not without influence upon the localization and the choice of hysterical symptoms.

Regional, sensory disturbance essentially corresponds to regional paralysis. We are concerned with a sensory alteration, the limits of which are set by the naïve conceptions of the divisions of the human body. Thus an anesthetic region may be limited to the hand; then we refer to the glove-shaped form. Or it may be limited to the arm; if the hand is not involved we speak of the sleeve form of sensory change. Localization to the shoulder furnishes the epaulette variety, that to the upper trunk the waistcoat form, etc. Analogous limitations are found in the leg (the splint form, the bathing-tights form, etc.). The localization about a joint is apparently suggested by such a di-

visional conception; here we find the cuff-like, sensory alteration about the wrist, etc. The hat or hood variety is also common, therefore, a limitation of the sensory alteration to the hairy scalp perhaps including the forehead. Even greater and more special limitations have been noted. I have seen patients in whom the sensory disturbance was limited to a breast, or an eye, or even to the surrounding areas of the breast. The genital region, including the genital mucous membrane, may be the seat of this regional, sensory disturbance. These "*regions*" may be distributed in great number all over the body; on the other hand there may be but a single one. In the former instance they may be exclusively, or at least predominantly, limited to one side of the body and are then associated with the unilateral type. These regional, sensory alterations have been described as geometric.[1] In fact the individual region, as is to be expected from its psychogenous origin, is bounded by relatively simple figures; in the extremities by more or less incomplete circles the so-called amputation line; upon the trunk by almost straight lines. The designation "geometric" is improper in so far as there can be no question of mathematical accuracy, nor does the conception of geometric figures play the slightest rôle in the development of the disturbance. If the patients are examined more frequently and with greater care it will be observed that the limits are neither sharply defined nor are they at all constant and that they may vary to the same extent as do the popular conceptions of the limitations of the hand, foot, etc. It may often be shown directly that the limits may be displaced by suggestion or that the regions may be obliterated *in toto*. In regard to the relation of tactile and pain sensations in these areas the same is true as was stated of the insular and unilateral forms: sometimes there is merely esthesia, sometimes algesia, at other times both are disturbed. Hyperesthesia and hyperalgesia are much rarer than anesthesia, hypesthesia, and hypalgesia. The involvement of the mucous membranes and the deeper areas is not uncommon in the regional form. It is interesting to determine the relations of the boundaries of the different regions to one another. This is accomplished by a number of contacts going toward and away from the anesthetic zone. In the former instance, in the *zonipetal test*, a slowly approaching stroke of a pin from the normal areas is conducted to the anesthetic region and the patient is asked to report when contact or pain ceases. In the latter case, the *zonifugal test*, we start from the anesthetic region and the patient is asked to state when the tactile sense becomes apparent. It is in consonance with normal psychological laws that the anesthetic zone is found to be smaller upon zonipetal testing than with zonifugal. In my experience this may be most often demonstrated in hysteria—in contrast to simulation—but there are occasional exceptions.[2]

Among the regional hyperalgesias another group must be emphasized on account of its practical importance. These are the *arthritic hyperalgesias*.[3]

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[1] The designation "segmentary, sensory disturbance" is quite improper, because it is liable to produce confusion with the sensory disturbances dependent upon individual segments of the cord.

[2] These exceptions then, in part, correspond to the displacement turgescence in taking the field of vision described by Förster.

[3] The joints, in addition to their specific kinesthetic nerve terminations by which the specific sensations of movement and position are transmitted, also have nerve terminations, which, like those of the skin, transmit sensations of contact and pain.

In my experience they are usually monoarticular. The knee and ankle joint are most often involved. As these conditions are frequently associated with spontaneous pain and also with vasomotor disturbance they will receive consideration later on. These symptoms appear with every active or passive movement in the joint and sometimes even from the pressure of the bones upon one another in the same joint. The surrounding cutaneous region is often, but not invariably, involved in the hyperalgesia. Such patients naturally avoid any movement in the affected joint.

There may be absolutely no connection between the sensory disturbances and the motor phenomena previously described. In many cases, however, they are associated. Thus hysterical hemiplegia and contracture may be accompanied with hemianesthesia or hemihypesthesia, hemianalgesia or hemihypalgesia, etc. Only hysterical, functional paralyses are very often not associated with analogous sensory alterations.

In rare instances there is complete anesthesia or hypesthesia *distributed over the entire body.* Analgesia or hypalgesia is somewhat more common. Particularly in hysterical somnambulistic conditions is this met with quite as often as the other varieties of sensory disturbance. The nasal mucous membrane, which is especially sensitive in the normal person, may be involved in this hypalgesia or anesthesia. But it must be remembered that also in well persons, particularly those that are not hysterical, there may sometimes be a conspicuous lack of the pain sense distributed over the entire surface of the body—exceptionally even to the nasal mucous membrane as an individual peculiarity.

The hysterical *pressure points* show an especially circumscribed local area of sensitiveness. The most important of these are the jugular point (in the *incisura jugularis sterni*), the inframammillary point, the iliac point (about midway between the median line and the anterior spine of the ilium), the inguinal point, the epigastric point, the supraorbital and parietal points (sometimes at the center of the saggital suture, sometimes 1–2–3 cm. to the side of it). The sensitiveness of the iliac region was formerly designated as *ovarie*; we know now that this pressure point is not connected with the ovary. Occasionally there is a circumscribed point restricted to the ovary which is sensitive to pressure, but it can only be recognized by internal examination. An abnormal sensitiveness of the testicles to pressure is not infrequent. The intercostal spaces may present the same condition, but this sensitiveness to pressure by no means corresponds to the location of the intercostal nerves. This condition is often most marked in the mammillary and axillary lines. Pressure points in the hypochondrium, upon the sternum, and upon the mastoid process are much rarer. Sensitiveness of some few or of all of the spinous processes of the vertebræ and of the transverse processes of the cervical vertebræ is common. There are also characteristic pressure points in the course of the large joints. It is exceptional to find all of the pressure points in one and the same case; on the contrary we usually find but a limited number. It is very rare to find all of them absent. They differ from the analogous pressure points in neurasthenia, firstly, by the fact that they are limited or at least most marked upon one side of the body, and secondly, that pressure not only produces pain but also symptoms of an attack (oppression, etc.) or an actual attack, and as it is usually expressed have a "*hysterogenous*"

effect. Cutaneous sensation in the region of the pressure points is often normal. On the other hand there may be regional hyperalgesia which corresponds to the form previously described and may be used as a differential factor from neurasthenia. If there is hemianesthesia the pressure points are situated upon the non-anesthetic side, but I have found them often enough upon the same side. There is no satisfactory explanation for the peculiar localization of these pressure points. They rarely correspond to the larger nerve trunks although even these often show a greater degree of sensitiveness (not a sensation of stretching). Suggestive factors may be decisive.[1] Therefore, if the attention is diverted they may disappear.

Thermoanesthesia, as a rule, goes hand in hand with the contact and pain senses. If only the latter, as is often the case, is disturbed, thermoesthesia is intact; but exceptionally there is a parallelism between the temperature and pain senses. We must be prepared for all kinds of imaginable dissociations of sensation in the case of hysteria. More common than thermohypesthesia and thermoanesthesia is thermohypalgesia and thermoanalgesia.

The *kinesthetic, sensory disturbances* occupy a somewhat more substantive position. They are most frequently unilateral; they often accompany hysterical hemiplegia or hemiparesis and, therefore, are often combined with hemianesthesia. Occasionally I have noted the condition alone. It is very significant that in spite of the existence of severe kinanesthesia there is no, or but very slight, ataxia. In other cases the patient may present ataxia with the finger-nose and knee-heel tests upon the kinanesthetic side, but not during eating, buttoning their clothes, walking, etc. In still other cases hysterical kinanesthesia may produce disturbances in coördination which may involve all movements of the kinanesthetic members.

The functions of the special senses are often affected in hysteria. Most often we meet with hysterical

Disturbances of Sight.

The severest hysterical disturbance of sight is complete *bilateral amaurosis*. It is quite rare and occurs in connection with a hysterical attack or after severe emotion. Jean Paul has furnished us with a remarkable illustration in his "Titan." The pupils are oftener dilated than contracted. The reaction to light (upon careful examination) is always retained, but it may be slight, delayed and sluggish. Amaurosis may last but a few minutes, at other times it remains for hours or days; some authors have reported cases in which the condition persisted for a year. Often amaurosis is not complete; the patient maintains that smoke or fog dims the field of vision. Amaurosis may be limited to one eye.

In contrast to amaurosis, in other cases, there is a unilateral or—more frequently — bilateral *optic hyperesthesia*.[2] In severe cases there is optic hyperalgesia. Even feeble light dazzles and may actually be painful. If the eyes are illuminated there is continued blinking or an almost tonic contraction

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[1] In this connection it must be remembered that these are areas which even in the normal person are somewhat more sensitive to pressure.

[2] The designation "*hyperesthesia retinæ*" is entirely incorrect.

of the orbicularis oculi. Often the previously mentioned clonic or tonic blepharospasm is due to this optic hyperesthesia or hyperalgesia;[1] in these instances it disappears or at least lessens in the dark.

By far the most frequent hysterical disturbance in sight is *concentric limitation of the field of vision.*[2] This can only be demonstrated with absolute certainty by the perimeter; only in the severest cases is the finger test sufficient. The field should be determined for white as well as for colors. If we are dealing with colors it is necessary to instruct the patient that he is not to report the time when he sees the test object but only the moment when he recognizes the color. It is also advisable to have the colors tested in varied succession; not red at first in all meridians and then green in all meridians, etc. Each color must be tested at least 3 times in every meridian: with a single test an accidental error may give a very incorrect result, and besides, from the variation of the figures upon repeated tests, valuable diagnostic factors may often be obtained. If this causes the examination to be too lengthy, there should be an intermission, or several sittings may be necessary. Finally, it is necessary to test the field of vision of a normal person immediately before or after this examination for comparison. On account of the frequency of careless investigations of the fields of vision and on account of their difficulty and importance in hysteria these few remarks in regard to the method of examination do not appear to me to be superfluous. If the fields of vision for the individual colors, obtained after careful examination, are compared, it will be found that the limitation is often uniform; then the normal sequence of the color fields, arranged according to their extent (blue—red—green), is also retained. On the other hand occasionally the limitation for one color is greater than the others; thus the limitation for blue may be especially marked; I have repeatedly observed that the color field for green is larger than that for red. Usually the limitation may be demonstrated in both eyes. If the condition is associated with hemianesthesia, as a rule it is greater in the eye upon the hemianesthetic side. Occasionally in a case of this kind it is noted that the limitation in the right-sided half of the field of vision in right-sided hemianesthesia, in the left side in left-sided hemianesthesia in both eyes is especially marked. True hemianopsia—apart from the effect of very special suggestion—has never been observed in my experience.

It is quite remarkable that even in marked hysterical limitation of the field of vision orientation in space does not suffer. From this it is apparent that the peripheral areas of the field of vision, which are lacking upon examination with the perimeter, are by no means excluded completely. This may be demonstrated by the stereoscope. If the field of vision is projected at various distances upon a blackboard a similar "tubular field of vision" may be found, such as is otherwise only detected in malingerers. In contrast

[1] In some cases the causal relation is inverted; optic hyperesthesia forms an autosuggestive complement of the blepharospasm. In rare cases a blepharospasm, that has persisted for a year, may produce photophobia which may be regarded as physiologic on account of the long continued absence of light.

[2] An increase of the fields of vision beyond the normal limits does not occur. It must be remembered that also in normal persons there may occasionally be very wide fields of vision.

to neurasthenia the concentric field of limitation persists in hysteria independent of fatigue.

Central (macular) acuity of vision and central (macular) sensitiveness for colors, apart from the previously mentioned amaurosis, are rarely disturbed in hysteria. Central confusion of colors—only due to suggestion during the examination—is very infrequent.

Much less common are hysterical

Disturbances in Hearing.

Like those of sight, they may be unilateral and bilateral. Sometimes there is complete deafness, at other times only more or less difficulty in hearing. Occasionally there are definite, but never sharply, limited and complete breaks in Bezold's tone scale. Rinne's test in unilateral hysterical deafness —under the influence of special auto-suggestion—may, exceptionally, be negative; lateralization to the normal side may also take place with Weber's test. With the ordinary tests for hearing the hysterical patient may react just as a malingerer. If there is hemianesthesia we often find, especially upon the anesthetic side, a decrease in the acuity of hearing.

Acoustic hyperesthesia and hyperalgesia are not uncommon; they are usually bilateral.

Disturbances in Taste and Smell

occur in the same manner as do the other sensory disturbances of hysteria. We, therefore, recognize a unilateral and bilateral ageusia, hypogeusia, anosmia, hyposmia, hypergeusia and hyperosmia. Unilateral decrease of taste and smell ordinarily accompany hemianesthesia.

From these statements it will be observed that quite often hemianesthesia, in the restricted sense, i. e., unilateral cessation of the contact sense and the accompanying sensitiveness to pain, are combined with a unilateral arrest of the function of organs of special sense upon the same side. This entire symptom-complex has been designated "mixed sensory-sensorial hemianesthesia."

For the sake of completeness I must add that the *vestibular* functions are sometimes damaged in hysteria, usually (but not constantly!) bilaterally. Such patients have a gait which closely resembles the tottering walk of cerebellar disease. Tormenting, sometimes almost continuous, pseudo-movements of objects appear in these cases. Some of the brief or prolonged "attacks of vertigo" of hysteria may be explained in this manner and would, therefore, be analogous to paroxysmal amaurosis, etc., of hysteria.

Of specially practical importance are the sensory *disturbances of the sexual sphere.* Besides anesthesia and hyperesthesia of the mucous membrane (of the vulva and vagina) a decrease and increase of the normal sensations play a great rôle, therefore, sexual anhedonia and hyperhedonia.

There are also pathologic increase and decrease of *hunger, appetite, and thirst.* Alimia and bulimia, hysterical anorexia, adipsia and hyperdipsia must be mentioned. The latter may lead to polyuria which may resemble the severest varieties of polyuria from disorder of metabolism.

In any of the qualities of the special senses that have been discussed a positive or negative emotional phase may develop which may produce path-

ologic, sensory qualities in any one of them. Pitres has described haphalgesia, a condition in which contact with metals and other absolutely painless substances is accompanied by an intense feeling of pain. I know of quite a number of hysterical patients who maintain positively that the viewing of pointed articles, as the points of a compass, is associated with intense pain in the eyes. This aichmalgesia differs from aichmophobia by the complete absence of fear. Analogous conditions in the psychical life of normal individuals are closely associated. To this category belong many special forms of copiopia described by Förster. These "*selective hyperalgesias*," as they should be called, are especially conspicuous among the last mentioned functions of special sense: the idiosyncrasy of many hysterics to certain foods, the condition known as *pica* being here included. Even the preference of hysterics for certain colors, such as bright red, for certain odors, such as strong perfume, should not always be referred to hypesthesia of the organ of special sense as is done by Charcot, but more often to a selective, pathologic emotional condition, in the sense described above. I need hardly state that all of these *specialized*, or as Janet says, *systemized* pathological emotional impressions depend upon associations of impressions which are decisive for the selection or specialization. In aichmalgesia this association of impressions is apparent. In many other cases the proof of the selective association is very difficult to obtain. I must reiterate emphatically that in these "psychogenous" derangements of hysteria we are concerned with unconscious association processes. It is, therefore, readily understood that the patient is unable to give any reliable data in regard to the development of his idiosyncrasy; we are even compelled, in the endeavor to give an explanation, to content ourselves with hypotheses. Thus we may presume that the haphalgesia for metals is unconsciously dependent upon the association of the idea of a "weapon," the preference for bright red upon the association of the impression of the unusual or conspicuous. At all events these disturbances are intimately connected with the psychical sphere; in a restricted sense they are even more definitely psychogenous than the simple derangements with which we were first made familiar. But they are in apparent analogy to the special, motor, functional derangements. Their complete comprehension might be attained from the law of reflexion and irradiation of emotional conceptions, to which I must refer the reader.

Up to this point we have considered only the sensory disturbances of hysteria originating from stimulation outside of the nervous system. In addition to these there are also

Spontaneous Pains

the source of irritation for which we must seek exclusively within the nervous system itself. Often these are the symptoms which cause us the greatest difficulties in treatment. As regards distribution the *regional* type is by far the most common. I designate these regional pains as *topoalgia*.[1] The pain is frequently localized in a joint, especially in the hip or knee joint. Then we refer to *hysterical arthralgia*. Here must also be mentioned hysterical

[1] I give this term a broader meaning than was originally intended by Blocq with his topoalgia. Psychalgias have also been mentioned.

rachialgia, a regional, spontaneous pain, appearing along the entire extent of the vertebral column or localized in some of its divisions; further, coccygalgia or coccygodynia, sacralgia, mastalgia, etc. Hysterical cardialgia is also very common. Localization of the pain may sometimes imitate bladder or renal colic. I remember a case in which the symptoms so closely resembled hepatic colic that an operation was performed, but only normal relations were present. Limitation of the pain to a single extremity is also noted. In the head the pain is most frequent in the parietal region; more rarely it may have an occipital or temporal localization. It is also called *clavus.* Sometimes it is median, at other times unilateral. The *unilateral type, hysterical hemialgia,* is less common than the regional form. On the other hand pains are often observed which, upon superficial examination, appear to follow accurately the distribution of a nerve trunk, therefore, resembling true neuralgia. If these hysterical pseudoneuralgias are carefully examined it is found that the pain is not limited to the distribution of a peripheral nerve, but that they are modified by the manner in which the physician investigates the sensory condition or by questions giving rise to that suggestion.[1] Apparently these are, therefore, only regional, spontaneous pains of psychogenous origin, topoalgias in our sense of the term. Hysterical intercostal and trigeminal neuralgias are most frequent. In my experience these pseudoneuralgias are never accompanied with herpes. The trigeminal neuralgia may be limited to one branch. When the pains are situated in the cardiac region they may radiate to the left arm and resemble angina pectoris, particularly since the pulse in these attacks of pain is rapid and labile. In a similar manner there may be a resemblance to sciatica.

The first appearance of pain may be in connection with emotional shock or a spasmodic attack; sometimes it is actually due to some peripheral irritation. Thus I have repeatedly seen an exceedingly tenacious topoalgia of the leg in connection with some gynecological affection, such as a hematocele. The irradiation of pain due to the latter condition is apparently the cause of the pseudoneuralgia and determines its localization.

The pains are stated by the patient to be extremely intense. At times they are continuous, at other times paroxysmal. External irritation, such as cold, pressure, etc., which in true neuralgia and especially in neuritis, as a rule, produces an exacerbation, usually has no constant influence. But the significance of psychical factors is all the more noticeable. Diverting the attention is of decisive importance. I knew a married woman whose exceedingly tenacious neuralgia disappeared after she had procured a lover. Quite as characteristic is the influence of skilfully concealed suggestions.

Sensation may be entirely intact in the region of the topoalgia; more often there is regional hyperesthesia and especially regional hyperalgesia.[2] Clavus may be associated with a more or less extensive hyperalgesia of the scalp, hysterical coxalgia, i. e., hysterical arthralgia of the hip joint with hyperalgesia in the shape of "half a loin cloth" or with hyperalgesia of

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1 Even an insignificant, actually subsistent organic pain may act in this sense.

2 In those cases in which hysterical topoalgia, as mentioned above, occurs in connection with an organic disease of the internal organs, the hyperalgesic zone may correspond to Head's irradiation zones. In the main, however, the regional hyperalgesias of hysteria have nothing in common with Head's zones.

the capsule of the hip joint, etc. In addition there is often an additional pressure-point. Thus originates the triad: topoalgia, regional hyperalgesia and pressure-point that we meet with so often in practice.

Marked motor symptoms appear in addition to these spontaneous pains of hysteria. Among these is the avoidance of movement due to the fear of pain; this occurs especially in that part of the body that is the seat of the topoalgia. This fear of movement occurs especially in those cases in which the topoalgia is combined with hyperalgesia of the joint, each movement increasing the pain. Hyperalgesia of the joint without topoalgia is sufficient to produce this fear of motion. If these topoalgias or hyperalgesias are very much distributed they may cause an almost absolute immotility. This condition has been designated *acinesia algera.* In an analogous manner hysterical cardialgia, combined with hysterical hyperalgesia of the mucous membrane, may give rise to almost complete refusal of food (*aphagia algera*), etc. The conscious motive in the avoidance of pain need not always be present; there are also conditions in which motion is avoided, where it is necessary for us to postulate a topoalgia as an unconscious psychogenous factor.

In other cases the movements are not arrested, but are modified in a characteristic manner so as to protect the joint in which there is topoalgia. Thus in hysterical coxalgia and gonalgia there is limping, etc. The exceedingly numerous hysterical disturbances of gait are still further increased by this condition.

The motor, *irritative* symptoms which occur in connection with topoalgia are of great importance. Thus the clonic muscular spasms may develop secondarily as the result of topoalgia. In hysterical pseudoneuralgia of the fifth nerve there is a hysterical tic which may simulate tic convulsif such as accompanies true tic douloureux. Hysterical brachialgia may lead to peculiar clonic spasms of the arms, etc. Hyperalgesia often plays a similar part to topoalgia. Probably vomiting, which is often observed in hysterical cardialgia, must be embraced here. I include, further, among these secondary, motor, irritative symptoms the contractures which appear occasionally in the area of the topoalgia. Such hysterical contractures, the result of pain, are most often observed in arthritic topoalgia, therefore, in hysterical coxalgia and gonalgia. The extremity may assume the position which is characteristic of coxitis or gonitis. In some cases it has appeared to me that hysterical contracture and hysterical arthralgia have appeared simultaneously and that they are coördinated.

These spontaneous pains of hysteria are of additional importance from the fact that hysteria is often complicated with true *migraine.* I have already stated that among the spontaneous pains of hysteria there is also a unilateral "regional" headache. This is not, as a rule, associated with symptoms on the part of the sympathetic nerve nor with vomiting, and can, therefore, not be regarded as migraine. We only observe genuine attacks of migraine as a complication.

In addition to pain there are also other sensory and sensorial irritative symptoms which appear occasionally, such as paresthesia and, in so far as they are accompanied by pain, also paralgias; further pruritus, and on the part of the organs of special sense, tinnitus aurium, sparks before the eyes, etc. The attacks of vertigo of the hysteric sometimes present peculiarities

which may be due to an irritative condition in the vestibular distribution. All of these sensory symptoms may pass into true hallucinations. It will, therefore, not appear strange that—especially in regard to their psychogenous development—these, as well as the topoalgias, have been designated as "pain hallucinations."

In contrast to the manifold disturbances of motion and sensation which we have learned to recognize, the alterations on the part of the

REFLEXES

are of decidedly less importance.

In testing the *tendon reflexes* we often find an active, symmetrical increase in all of them. No special importance is to be attached to this, as the same condition is likely to occur in any of the other functional neuroses and in the neuropathic predisposition. In many cases the tendon reflexes are entirely normal. A decided decrease or the entire absence of the reflexes is exceedingly rare in uncomplicated hysteria. It is especially noteworthy that the tendon reflexes of the paralyzed members, in the course of hysterical paralysis, are not influenced. This is an important differential point from organic paralysis. An increase of the tendon reflexes is sometimes simulated, in that the patient develops more or less conscious movements of resistance to the reflex action. This is often easily recognized from the fact that the reflex movement occurs too soon or—what is more common—appears after a short pause. Even foot clonus may appear from such psychogenous additions, especially if the test is performed by the physician in a manner that may convey an idea of suggestion. A true, well developed foot clonus [1] has been noted by me as a transitory symptom in hysterical somnambulism. Hysterical anesthesia has as little influence upon the tendon reflexes as hysterical paralysis, even when the kinesthetic sensations have been completely abolished. It must be stated in addition that the tonus, as tested by passive movements of the joints, is never decidedly decreased or even abolished; only slight hypotonia and hypertonia is sometimes noted.

The *pupillary reflexes* are always retained. Especially rigidity to light, as I may state from my experience, is never observed. The isolated cases of hysterical rigidity to light of the pupils, mentioned in literature, are open to doubt; sometimes the test was not performed carefully enough, and at other times it was apparent that the patient had produced the condition artificially by atropin, etc.; finally, syphilitic disease of the brain, combined with hysteria, must not be forgotten. But it must be admitted that occasionally the reaction to light, in consequence of a spasm of the sphincter or of the dilator, may be slight or may appear very sluggish. Convergence is absent only when convergence and accommodation are absent from hysterical paralysis; there can be no question of an absence of reflex, for the reaction is lacking only because the stimulus is lacking, and in addition we are not dealing with a true reflex.

I must state in advance that in regard to the *cutaneous* and *mucous membrane reflexes* the condition, in our clinical examination, is by no means so simple as might seem from older views. The true reflex motion is, as a rule,

[1] Indications of a foot clonus are often found.

accompanied by a psychogenous double. This is best observed in the sole reflex. Upon tactile irritation of the skin of the sole the following movements may be observed:

(1) Plantar flexion of the toes.

(2) Contraction of the tensor fasciæ latæ, occasionally also of the quadriceps.

(3) Dorsal flexion of the foot.

(4) Flexion at the knee and hip joints.

Among these 4 components of the sole reflex the third and fourth reveal, in a conspicuous degree, the character of resistance and flight movements. The same is also true of other reflexes. Unfortunately we do not know in what portions of the central nervous system the point of origin of the individual components of these movements is to be sought. This much is, however, certain, that the individual components, in pathologic conditions, vary in their behavior to one another. In hysteria we note a more or less general decrease or even arrest of all cutaneous and mucous membrane reflexes. Especially the palate and retching reflexes, the conjunctival reflex (the palpebral as well as the episcleral) and the sole of the foot reflex are entirely abolished. It is not necessary that anesthesia accompany this condition. Great importance must not be attached to this absence of reflexes, as it may occur as an individual peculiarity in normal persons. Very much rarer is the absence of the epigastric reflex; naturally under the supposition that the test is made under conditions which permit the production of the reflex (flaccidity of the abdominal walls, etc.). It is also rare for the corneal reflex to be absent.

The behavior of the reflexes in hysterical hemiplegia and hysterical hemianesthesia is of greater importance. It is characteristic of the former that the Babinski reflex (dorsal flexion of the toes, especially of the great toe, instead of plantar flexion) and also Oppenheim's sign (dorsal flexion of the foot and of the toes upon stroking the medial surface of the lower leg) are never observed; but care must be exercised that psychogenous movements of resistance are not mistaken for the reflex. This latter condition is usually most distinct in the sole reflex, and I have found that, as a rule, the 4 components are most affected. The fact must not be concealed that other investigators, even recently—for example Crocq—have arrived at different results. In hysterical hemianesthesia a similar condition is noted; upon the anesthetic or hypesthetic half of the body the reflexes are decreased and especially in those components which—such as the movements of resistance—also under normal conditions are under psychical control. This permits us to comprehend that the winking reflex—and not only in complete amaurosis—is often decreased.

Perhaps more often a pathologic increase of the reflexes is noted in hysteria. These also affect particularly or even exclusively the complicated resistance components of the reflexes. These are patients who upon the slightest touch of the sole not only raise the leg but bend the entire body: upon illuminating the eye they not only close the organ tightly but also draw back, etc. These psychogenous pseudoreflex movements are not only called forth by the normal reflexogenous zones but in severe cases from any area of the skin. Very often there is an underlying general hyperalgesia or

hyperesthesia. It may often be noted that pressure upon one of the pressure-points gives rise to decided pseudoreflex movements. I am, however, familiar with positive cases in which hyperesthesia, hyperalgesia and sensitiveness to pressure over the pressure-points were emphatically denied, and nevertheless this exaggerated pseudoreflex irritability was present to a marked degree. Occasionally it may be unilateral and is then often associated with hemi-hyperalgesia. If the pseudoreflex irritability is greatly increased there may be pseudoreflex, clonic and tonic spasms, which throughout resemble the primary motor irritative symptoms that we have already learned to recognize.

We may be much briefer in regard to the symptoms of hysteria in the field of the

SYMPATHETIC NERVOUS SYSTEM

We shall begin with the *vasomotor phenomena*. A slight *tachycardia* is common. Usually it appears paroxysmally; severe attacks are rare. *Bradycardia* is even less common. The *surface temperature* in hysterical paralyses is frequently decreased in the paralyzed extremity, even if the paralysis has been present but a brief time. The characteristic initial rise of the surface temperature present in most organic paralyses I have never observed in hysterical paralysis. Blood pressure shows exceedingly variable conditions.

Peculiar forms of *edema* are very conspicuous. These are occasionally observed in hysteria. We may differentiate 3 forms:

(1) Quincke's circumscribed edema,
(2) Blue edema and
(3) White edema.

True circumscribed edema corresponds to the angioneurotic edema of Quincke and is very rare in hysteria. I believe it likely that when present it is chiefly a complication.

Blue edema, also called *Charcot's edema*, is somewhat more common. In this condition as in Quincke's edema there is no marked pitting upon pressure. The extent varies greatly, but it is usually not distributed over large areas. I have repeatedly seen the edema limited to the neighborhood of a joint, which, in itself, was not involved. The surface temperature in the area of the edema is decreased. It appears least often in the face. I have observed it most commonly in the hand and forearm, especially upon one side, but it is not rare in the upper arm and in the leg. It is frequently complicated with other local symptoms of hysteria, such as regional paralysis, regional contracture, regional anesthesia, analgesia or hyperesthesia and hyperalgesia; the last named is then commonly limited to the joint in the surrounding of which the edema has appeared. It is often accompanied with a regional, articular topoalgia. Sometimes it appears gradually, at other times acutely. Sometimes its development is connected with an insignificant trauma. Its course may be very chronic. Under psychical influence it may disappear very rapidly.

The white, or *Sydenham's edema* differs only by the pallor of the skin. As there are many transitional forms (apparently also an *œdème rouge*) I doubt very much whether the white and the blue edema are essentially different. The associated symptoms and the course are identical.

It has been stated that the mucous membranes are not involved by these

pseudo-edemas; nevertheless, in a case of severe hysteria, in which there had formerly been hysterical aphonia I observed an edema of the mucous membrane of the palate and of the false vocal cords, which persisted for a year. The case was watched by laryngologists and was regarded by them finally as neuropathic.

It is not known to what vasomotor disturbance this form of edema is due. It is generally supposed that it is the result of vasomotor spasm or the like.

Other cutaneous vasomotor affections have been described in hysteria, but in this connection I cannot enjoin sufficient caution. These are, as a rule, either accidental complications or—more frequently—injuries which the patients produce purposely. A tendency to hemorrhage resembling hemophilia appears exceptionally, but especially in this matter there should be great scepticism on the part of the physician. In the only case in which I was able to avoid an error by personal observation there were not only cutaneous but also intestinal and pulmonary hemorrhages. The patient perished from the continued hemorrhage. The autopsy revealed no sufficient cause for the hemorrhage. It is not unlikely that the insufficient and monotonous form of nourishment of the patient causing anorexia played an important rôle in the condition. On the other hand we are often astonished in hysterical patients at the slight bleeding after injury.

If we were to credit the reports of the patients, *secretory disturbances* would be very common. Thus the patients complain of lachrymation, rhinorrhea, sialorrhea, hidrorrhea (hyperidrosis), galactorrhea (from the breasts), etc. Most of these cases are found to be simulated. Nevertheless, some few disorders may be present. Colporrhea has been observed. It cannot be doubted that the excretion of urine is subject to great variation and this independent of the intake of fluid. Oliguria and polyuria may alternate. The influence of emotional disturbance is readily recognized in this. The cases of anuria lasting many days are deceptions.

Disturbances of metabolism do not occur in hysteria, at least not outside of the hysterical attack.

The so-called *hysterical fever* is almost always artificially produced. If the physicians who have described this condition would observe the matches at the bedside of the patient most of these cases would be unmasked. Only when the physician takes the temperature with his own thermometer in the axilla of the patient or in the rectum are we certain that there is no deception. Although we have often been told by the patient and by the family of temperatures up to 110° F. I have never found temperatures above 101.5° F.; naturally apart from cases in which there is no complication such as miliary tuberculosis I can only admit that under the influence of emotion, etc., more readily than in normal persons, slight rises in temperature may occur, but the designation "fever" is entirely misplaced when applied to this condition.

In regard to *menstruation* I believe that in hysteria—quite apart from any complication of the genital organs—irregularities are more common than under normal conditions and especially than in other nervous affections. Here also emotional disturbances appear to be of importance.

Disturbances in the innervation of the smooth muscles of the intestines have also been observed. At times these conditions are spasmodic, at other

times paralytic. Here, in addition to hysterical vomiting, which may be independent of cardialgia and hyperalgesia of the mucous membrane of the stomach, hysterical eructations, hysterical meteorism, hysterical borborygmi, etc., have been reported.

While the corporeal symptoms of hysteria are exceedingly variable the

PSYCHICAL PERMANENT SYMPTOMS

on the whole are much more uniform. We include these psychical permanent symptoms under the term "HYSTERICAL PSYCHOPATHIC CONSTITUTION." I may limit myself, in the description of this condition, to pointing out the connection with the corporeal symptoms and its influence upon the latter. The psychopathic constitution is characterized by the following factors: the variation in sensation and emotional conditions and especially the tendency to extremes, the pathologic absent-mindedness, the pathologic increase of phantasy, the inclination to paramnesia, the faculty of translating emotional impressions into hallucinations and even into "fixed ideas," the entire absence of objectivity and finally the abnormal suggestibility.

It is obvious that these psychical symptoms are in intimate relation and are all subject to the general principle which dominates the entire symptomatology of hysteria [1]: they may all be referred to the *abnormally increased activity of emotional impressions.* That the corporeal symptoms, in many cases, are also due to this is plain from our preceding description. It is self evident from this condition of affairs that the bodily symptoms of hysteria are in close relation to the psychical ones. I shall only indicate a few practical and important examples.

The intensity of the corporeal symptoms, in many cases, depends upon pathologic variations of the emotions; we have indicated this dependence several times. Many symptoms are first produced by this variation in the perceptions, many are increased by any emotional irritation. On the other hand a marked swaying of the emotional condition, in the sense of a diversion, may cause a tenacious hysterical symptom suddenly to disappear. Almost quite as significant is the rôle of the pathologic absent-mindedness for the corporeal symptoms. The hysteric is confused, i. e., inattentive in the sense of a defect of tenacity or concentration of attention, in so far as these emotional impressions are concerned. It must, therefore, not excite surprise if, in taking the field of vision, in dynamometric and ergographic investigations, etc., often very dissimilar results are obtained. Even the personality of the investigator may play a part. Janet and other authors have even attempted to refer the hysterical anesthesias and paralyses to pathologic inattention. Although an analogy exists between the hysterical symptoms of absence of function and physiologic inattention, Janet's view is entirely too one-sided. The active causative factor which also dominates the hysterical symptoms of absence of function, namely the importance of the conception of not being able to move, or to feel, etc., is overlooked. The tendency to abnormal imagination and to the transformation of memory impressions is

[1] Thus it happens that in the history of the teachings of hysteria almost every one of the psychical symptoms that have been mentioned has been regarded by some author as the underlying principle of all of the others.

particularly active in giving a report of the corporeal symptoms. The precise observation of the neurasthenic will not be seen in hysteria. Contradictions in subsequent reports and even in one and the same report are common. The tendency to hallucination processes we have already learned to recognize in considering hysterical topoalgias. We have seen that the latter are actually designated "pain hallucinations." In fact some authorities have gone further than this and have called the hysterical anesthesias "negative hallucinations." I need scarcely state that this is nothing more than an ingenious play upon words. Finally, the pathologic suggestibility in regard to the corporeal symptoms may be demonstrated everywhere. Often this is decisive for our diagnosis. We have seen that by "*transference*" it is possible to change the paralysis and the sensory disturbances from one side of the body to the other, that the result of the examination of the sensory and motor conditions is often dependent upon concealed suggestion or is altered by this influence, that frequently only by a concealed suggestion, contained in the manner of the examination, one or another symptoms is provoked. If we expand this conception of suggestion to the extent that it includes auto-suggestion, all or nearly all symptoms of hysteria become suggestive symptoms; in this enticing generalization I must, however, call attention to the considerations detailed at the beginning of this article.

PAROXYSMAL SYMPTOMS

We now turn to the paroxysmal symptoms of hysteria, therefore, to the

HYSTERICAL SPASMODIC ATTACK AND ITS VARIATIONS

We shall first describe the ordinary, hysterical, spasmodic attack in its severest and most complete phase, to a certain extent the *classical type*. A severe attack consists of the following phases:

(1) The remote prodromes;
(2) The immediate prodromes, the aura;
(3) The epileptoid phase;
(4) The phase of coördinated (in part mimical) spasms;
(5) The delirious phase.

The *remote prodromes*, as a rule, consist in a change of the disposition such as an increased irritability, with more or less insufficiency of motive for this depression. An indefinite fear is also frequent which shows its hysterical character from the association with corporeal accompanying symptoms, such as a sensation of constriction in the throat (*globus*) and oppression upon the chest; the patient frequently complains of numbness and formication in the hands and feet. There is often an increase of the previously described pseudoreflex irritability. Sometimes there is acceleration of the pulse or it is at least very labile, at other times there is merely a subjective sensation of cardiac palpitation. The stage of remote prodromes may last from a few hours to a few days.

The *immediate prodromes* consist of the hysterical *aura*. Sometimes this is merely a sudden increase of the sensations of fear, constriction and oppression just described. At other times the patient reports that a spasmodic sensation rises from the epigastrium, from the deeper parts of the abdomen.

from the region of the heart or from the extremities. "Everything becomes numb," "there is a spasm all over," "a ball rises to the head." In connection with the aura the patient SLIPS to the floor; sometimes there is an impression that he throws himself upon the ground. Rarely is there such an abrupt fall as in the epileptic attack. Injuries due to the fall are, therefore, extremely rare. Where these are noted in the hysterical attack they are almost always due to the powerful spasmodic contractions of the later stages.

The *epileptoid phase* consists of tonic and clonic spasms which resemble those of the epileptic attack. The tonic spasms often precede the clonic, as is the rule in the epileptic attack. The head is spasmodically thrown to and fro; the eyes are commonly spasmodically closed; the angle of the mouth is distorted by the spasm; arms and legs are in tonic extensor or flexor spasm; the jaws are pressed together, rarely is the mouth wide open; the muscles of the trunk often present tonic rigidity. In consequence of a transitory arrest of respiration (usually in the inspiratory position) there is occasionally slight cyanosis. After from 30 seconds to a minute this tonic spasm is replaced by clonic convulsions. These are at first apparently distributed over the entire body and, therefore, also at the first glance resemble epilepsy. Upon careful examination, however, significant differences are noted in regard to the tonic as well as the clonic convulsions. Above all, the muscle contractions are not so uniformly distributed over the entire body. There is a certain "choice" and "coördination" even in the epileptoid phase which is even more distinct in the succeeding stage. The tonic trunk and head contractions are more complicated than is generally noted in epilepsy. The tonic spasm of the extremities brings about postures which indicate that psychical, selective factors are operative. The "uniform" generality of the epileptic attack, in which, according to physiologic laws, the flexors predominate over, the extensors, is lacking. This choice is even more distinct in the clonic spasm. Many authors, therefore, include this with the succeeding stage. It is especially noteworthy that the hands and arms rather present a coördinated tremor than an incoördinated clonic spasm. The legs often show kicking and stamping movements which resemble voluntary movements or those due to emotion.

THE PHASE OF COÖRDINATED SPASMODIC MOVEMENTS

arises quite gradually from the epileptoid stage. I designate it briefly as the *stage of jactitation.* The patient now assumes complicated positions and performs complicated movements which distinctly betray coördination, that is, selection. Very frequently these resemble greatly exaggerated forms of expression and are, therefore, similar to those of the next stage. Some of these positions and movements have a special name, on account of their frequency or their conspicuousness. We speak of an *arc de cercle,* when the patient arches the trunk in opisthotonus to such an extent that finally only the tips of the toes and the occiput or—when the head is even more strongly retracted—even the forehead are in contact with the floor or the bed. Quite as common are the contortions of the trunk, sometimes winding movements, at other times a rigid lateral curvature. The head is thrown about, the hair flies around the head, sometimes following the rotations of the trunk.

The abdominal muscles are often involved in these spasmodic contractions (*hysterical belly-dance*). The pelvis often presents coitus-like movements. Sometimes the entire body is thrown into the air or turned to the side. The face is distorted by spasmodic grimaces. The eyes are usually wide open. The movements of the extremities are often very vigorous: the hands are balled into fists, then pronated and supinated to the extreme, the arms show the motions of striking, pushing, shaking or throwing, now they are pressed to the side or upon the chest, then extended high over the head or posteriorly, the legs stamp upon the bed or are thrown in the air, the thighs flexed upon the chest, sometimes extended to the extreme, widely separated or crossed. The movements are so violent that now and then injuries are produced. Often there are inarticulate sounds. Spasmodic crying or laughing may now be present. The respiration is accelerated, gasping and irregular. The totality of these movements has also been designated as "*grands mouvements.*"

The *delirious phase* is a continuation of the stage of coördinated and mimical spasmodic movements. The separation of these two stages is quite arbitrary and cannot always be accomplished. At all events it is characteristic that in the delirious phase the spasmodic movements gradually imitate definite emotions and impressions and consequently are better organized, in contrast to the irregular and indefinite irritation of the preceding stage. The patients laugh, cry, scream, bite, make grimaces, etc. One of my patients constantly inclines to the right side and with a furious expression in her face bites through her shirt into the right arm until blood flows, so that it is necessary to protect this member by a suitable bandage. In other cases religious exaltation may be noted in the facial expression or there may be the ecstasy of love or distraction. The patient assumes theatrical postures. There is forward bending or praying, kneeling and the like. The patient is no longer confined to the bed; he jumps up, runs away or attacks his surroundings. Sometimes he appears to listen or stares into space. Often scant expressions betray the psychical processes from which the peculiar gestures of the patient result. He utters threats or cries of fear and for help; or he prays or preaches; or there is murmuring, attempts to embrace those about him, with expressions of love. A patient of mine who had biting-attacks always stated in this stage "now I will bite off the head of my father-in-law." He was upon strained terms with the latter. In fact, the emotional conditions of the extraparoxysmal, relatively normal conditions, are often reflected in these deliria. Very frequently the gestures and expressions are in immediate connection with an emotional experience which may be very remote, but consciously or unconsciously is still active. An attempt at rape, a dispute with a neighbor, an insult or a punishment, an escape from danger often form the guiding motive. The patient re-enacts the experience in all of its details. We cannot escape the impression, from the actions of the patient, that there is a hallucination of his real experience. Besides, his exclamations indicate very distinctly that not only hallucinations and active imaginary conceptions but also illusions and delusions appear in the delirious stage. The patient speaks of men, animals, ghosts, etc., which he sees, occasionally also of voices that he hears. Therefore cases of mistaken identity are very common. Orientation as regards space and time are altered in a corresponding manner. The designation "delirious" for this stage is based

upon the existence of these illusions of the senses and of the hallucinatory conceptions in connection with the loss of orientation. The patient sometimes does not react to questions, at other times only in the sense of the delirious situation; rarely is the answer correct; often the delirium continues and while apparently unheeding the question, nevertheless, the patient shows by his subsequent behavior that he has understood. The delirium may often be greatly influenced by skillful suggestion.

While the epileptoid phase is very brief, the jactitory and the delirious phases may last for many hours. Very frequently there is a remittent course. After brief intervals the coördinated, spasmodic movements and the deliria reappear.

The termination of the attack is rarely abrupt; there is commonly a gradual decline. The patient either returns at once to his previous condition or for a longer or shorter time there is a sleep-like stage. Actual sleep is very rare. After the attack there is either complete or partial amnesia. As a rule the patients maintain that they remember nothing of the attack; but by skillful questions we may convince ourselves that at least some recollection is retained. Exceptionally there is even a complete and intact memory of the entire seizure. The partial amnesia occasionally reveals a remarkable "choice" of memory.

We shall now discuss a few of the accompanying symptoms of the typical attack. First the question arises: Is the patient actually unconscious during the paroxysm? The patient answers this question in the affirmative in most instances and probably on account of the amnesia. We must answer a decided NO to this question. The behavior during the delirious and jactitory stages permits of no doubt that in both of these phases psychical processes are in action; there can, therefore, be no question of loss of consciousness. But even in the epileptoid phase there are indications that stimulation of various kinds may modify the attack and, hence, that psychical factors are not lacking. We then have an important differential factor from true epilepsy in which loss of consciousness is characteristic.

The pupils which during the epileptic attack are most often non-responsive to light always react during the hysterical attack. It has been recently reported that exceptionally during the hysterical seizure there may be rigidity of the pupil to light. In the numerous instances in which I have had an opportunity of observing a hysterical seizure I have never observed a loss of the light reflex. The examination during the attack is so exceedingly difficult that the reports in regard to pupillary rigidity must be accepted with extreme caution. The cases in which experienced observers have reported pupillary rigidity were most likely cases of *hystero-epilepsy*. The pupil usually increases in size during the attack. The tendon reflexes are retained. Foot clonus occurs, the cutaneous and mucous membrane reflexes do not vary; they may be decreased but then the condition is symmetrical. Quite often during the attack, and then especially in the later stages there is a marked increase of the pseudoreflex irritability. The reaction to pin pricks may be absent, slight, or increased; at all events the susceptibility to pin pricks during the attack does not always correspond to the condition in the absence of attacks. Thus we may find that a patient who has marked hyperalgesia when free from attacks may not react to pin pricks even of the nasal

mucous membrane during the attack. Unilateral differences in sensation prior to the attack may disappear in the seizure; it is much rarer for these to appear during the attack. The relation of the pressure-points is also noteworthy. Pressure upon these occasionally increases the severity of the attack or may produce a new attack, or an increase of the symptoms in an attack that is just subsiding. In other cases pressure upon this or that point may abort an attack. Exceptionally in one and the same case pressure may either increase or control a seizure. Therefore we speak of *hysterogenic* and *hysterofrenic points.* The pain sense of these different pressure-points also varies greatly during the attack; sometimes it is decreased or disappears, at other times it is increased; nor is it rare for new points to appear either bilaterally or unilaterally. It is obvious that in all of these tests suggestion is a factor.

The severe or "*grand*" hysterical attack just described does not always pursue the course that has been indicated here. The remote prodromes may be absent and it is not rare for the aura to be lacking. Further the epileptoid phase may be ill-defined or absent. Finally the jactitory stage and the delirious may be combined; or these two stages may alternate. It is of importance to know that the two last stages may be much shortened so that the attack may be limited to the aura and the epileptoid phase; at most a rudimentary condition of the two last stages may be present consisting of spasmodic weeping, laughing or sobbing. Very often the entire attack may be abortive, i. e., it never passes beyond the aura. Upon the whole these mild, shortened, or abortive attacks are much more common than the severe ones. The duration of these incomplete attacks is also much briefer. I have seen seizures which were limited to a few seconds and these were often mistaken for syncope, vertigo or epileptic *petit mal.*

We must now call attention to some of the important *variations of the course.* The epileptoid phase may run its course under the picture of a Jacksonian attack or at least as a unilateral convulsive seizure. The reason for this unusual course is very apparent in some cases. I am treating, at the present time, a patient who had a tumor of the right motor region removed which was the cause of Jacksonian epilepsy. Subsequently the patient developed, in addition to other conspicuous hysterical symptoms, "automimical" spasmodic attacks which, to the inexperienced observer, bear great resemblance to Jacksonian epilepsy; nevertheless, on account of the great influence of suggestion upon the course and origin of the seizures they were unmistakably hysterical. In other instances seeing or hearing the account of a Jacksonian attack must be considered. In still other cases the unilateral persistence of the clonic and tonic spasms must be referred to the unilateral presence of definite permanent symptoms, such as a topoalgia. These incomplete motor attacks cannot be definitely differentiated from the localized (regional) muscular spasms due to permanent symptoms which, as we have seen, not infrequently are paroxysmally aggravated or in fact only appear in paroxysms.

More remote from the typical hysterical attack are the *cataleptic seizures.* Even in the course of an ordinary hysterical attack we sometimes note catalepsy, especially in the delirious phase; passive motion no longer meets with resistance, no more than in kneading wax (hence the designation "*flexibilitas cerea*"), and the limbs remain for a long time in the position given to them

by passive movements. These cataleptic conditions may be present alone almost unaccompanied by other hysterical symptoms of the attack. We then refer to hysterical "*cataleptic attacks.*" Exceptionally catalepsy may be unilateral or limited to one extremity. There is no or but very feeble reaction to pin pricks or calls. The eyes may be open or closed. The pupils do not show the characteristic miosis of sleep. The cutaneous and mucous membrane reflexes may be decreased. The tendon reflexes are not altered. After the patient awakens, which may sometimes be brought about artificially by powerful cutaneous irritation, memory is but slightly damaged: the patient reports that he has heard everything but was unable to speak or to move. Short cataleptic attacks are rare, prolonged cataleptic conditions are much more common in the course of hysterical psychoses.

Hysterical *sleeping attacks,* which are also called *hypnoid attacks,* are closely allied. Drowsiness commonly appears quite suddenly. If the patient, as was recently the case with one of mine, is unexpectedly taken with the seizure while he is out walking, he either falls asleep where he is or proceeds home in a staggering condition. The eyes are closed during the attack. A slight tremor of the eyelids sometimes shows that this closure is not passive as is the case in normal sleep. If the head is permitted to droop beyond the margin of the bed the eyelids, as a rule, remain closed. There is no reaction to calls or cutaneous irritation, but it may happen that a slight touch may awaken the patient, while he does not stir when called. Passive motion meets with no or but little resistance. Catalepsy is said sometimes to be present; I would include these cases with catalepsy as a sharp differentiation is impossible. The pupils are contracted; they react to light but are sluggish. The reflexes are the same as in the cataleptic attacks. Food, which is placed in the mouth, is sometimes swallowed and occasionally not. The duration—as in the case previously mentioned—is from 15–30 minutes, but it may be 16, 24, 48 hours or even longer. The sleep conditions lasting longer than this are accompaniments of hysterical psychoses. The short attacks have also been called *narcolepsy.*[1]

These sleeping attacks as well as natural sleep may be combined with *sleep wandering* (*somnambulism*).[2] One of my patients arose during his sleep, sat upon the window-sill so that his feet were hanging free, and was found in this condition. Others wander shorter or longer distances during sleep either with their eyes open or closed. The behavior of the patient often reveals that he is under the influence of illusions, delusions, or hallucinations. Suggestions are almost always effective in these somnambulistic states. The condition frequently resembles the delirious phase of the hysterical attack. In fact it differs only by its relations to sleep, that is, to a hypnoid condition.

We may regard the hysterical somnambulistic condition as a variant of the hysterical spasmodic attack; it is certainly possible to present an unbroken series between the complete spasmodic seizure and the hysterical somnambulistic state. The delirious phase may even be regarded as a brief post-paroxysmal

[1] The attempt to differentiate these "narcoleptic" attacks from the sleeping attacks, I believe to be futile. The designation "autohypnosis" for the sleeping attacks is improper because it is based upon an incomplete comparison.

[2] The term somnambulism should be strictly limited to conditions which can be demonstrated to be in relation to natural sleep, to the hypnoid condition or to hypnosis.

somnambulistic stage. In regard to the symptoms it does not differ at all from the ordinary hysterical somnambulistic state. The differentiation cannot be made from the unequal duration, for the duration of the ordinary hysterical somnambulistic condition varies within wide limits. There would remain then only the precedence of the earlier stages which might characterize the delirious stage from the hysterical somnambulistic condition. Little importance can be attached to this, for we have seen that any of these stages may be absent in the ordinary hysterical attack. If we consider, in addition, that in the course of the hysterical somnambulistic phase there are occasionally complete or incomplete spasmodic attacks, there must be general agreement that the hysterical spasmodic seizure and the hysterical somnambulistic condition are very closely related.

COURSE AND PROGNOSIS

At the very beginning of our discussion it is necessary to state that hysteria, like many another disease of the nervous system, is *delective* [1] in regard to its symptoms; by this I mean to express the fact that each individual case presents a choice of all of the symptoms which may arise in hysteria. This is the reason for the extraordinary multiplicity of the clinical pictures which we encounter in the course of hysteria.

Exceptionally this choice may go so far that at least for a time but a single prominent symptom, for example, a topoalgia, may be present and a careful examination is necessary to demonstrate other hysterical symptoms. Such mono-symptomatic cases are met with especially in the hysteria of children and also, as a rule, in the earliest stages of the affection.

The combination of symptoms due to this manner of selection does not remain the same in the further course of the disease, for, in addition to very tenacious and monotonous clinical pictures, we also observe an active change in the grouping of symptoms. In this respect there is no other disease of the nervous system that can compete with hysteria. This variation in the clinical picture is often associated with the spasmodic attack or with emotional conditions. Thus we may note a paralysis or tremor after a spasmodic attack which was not present previously. On the other hand it may be observed that a previous tenacious symptom may disappear after a seizure. The influence of emotional disturbances upon the development of new symptoms and complexus of symptoms has been mentioned so often that further discussion is unnecessary. In regard to accident cases it is of importance to know that the appearance of new symptoms after trauma or a severe emotional condition may show a certain period of latency. Thus after trauma the condition of the patient may be normal for several days and even weeks, and then merely a slight loss of power may appear which in a few hours develops into a well characterized paralysis.

The development and course of the affection must be regarded as chronic; on the other hand the appearance of a single conspicuous symptom is often acute and even peracute. The latter is especially true of the appearance of

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[1] I differentiate between "*delective*" and selective; the former designates a choice varying from case to case, the latter a constant choice.

the first manifest phenomena. In rare instances a decided tendency to periodicity has been noted in regard to some of the important symptoms.

If, nevertheless, it is thought desirable to single out a few of the individual pictures on account of their frequency and to give them a special designation, the following forms must be mentioned:

(1) The undeveloped cases;

(2) Those characterized by some striking feature (such as paralysis);

(3) Those characterized by frequent spasmodic attacks and

(4) Those in which severe psychical symptoms predominate.

I must repeat that these clinical pictures are often combined, or that they may alternate and that they by no means exhaust the abundance of the different varieties.

The **prognosis**,[1] in so far as complete and permanent cure is concerned, is unfavorable. Only in childhood have I seen a definite and complete cure, which in some special instances has been continued to 15 years and longer. Sometimes it is possible, in favorable cases, to bring about an intermission. The prognosis of the individual hysterical symptom is much better. As a rule even the severest hysterical symptom finally yields to suitable treatment or to fortunate chance. *Permanent "invalidism,"* in the legal sense, is exceedingly rare as the result of the corporeal symptoms of hysteria. Nevertheless, now and then, some symptoms are met with which do not yield to treatment, or, after they have disappeared, return anew. Some of the hysterical topoalgias belong to this category.

A fatal issue, apart from the hysterical psychoses and the attempts at suicide, is extremely rare. But the vomiting, anorexia, and some of the other symptoms may endanger life and aid in causing death. Some authors have reported fatal cases, the result of a hysterical spasm of the glottis. Personally I am not familiar with such a case.

DIFFERENTIAL DIAGNOSIS

The importance of the individual symptoms, from the standpoint of differential diagnosis, has already been considered in the symptomatology. We are now concerned with the differential diagnosis of special diseases. In this connection the following affections must be taken into consideration:

(a) Epilepsy.—Only the absolute loss of consciousness is decisive between the epileptic and the hysterical attack; and this is present only in epilepsy. Caution must be enjoined in taking the patient's statement of loss of consciousness during the attack. The amnesia of the hysteric frequently simulates loss of consciousness which, in reality, is not present. In these instances those that have seen the seizure must be thoroughly questioned as to whether the patient reacts to any form of stimulation, shows no complicated or mimical movements, and is, therefore, actually unconscious.

Often involuntary evacuation of urine, biting the tongue and self inflicted injuries are stated as the factors of the epileptic character of the attack, and in general this is true. But it must be added that the absence of involuntary evacuation, biting the tongue and injuries during the seizure do not exclude

[1] Hysterical psychoses are not considered here.

epilepsy. There are many epileptics in whom these three conditions never occur. Therefore only the positive factors and not the negative may be utilized in diagnosis. Besides it must not be forgotten that exceptionally involuntary evacuations of urine and especially biting the tongue (biting the lips somewhat more commonly) also occur in hysterical attacks. In regard to self-inflicted injuries only those that occur as the result of the fall come under consideration. Self-inflicted wounds in the later course of the seizure are quite common in hysteria while they are exceedingly rare as the result of the primary fall.

If the physician has an opportunity of noting an attack or if the report of a reliable eye witness can be obtained the nature of the convulsions should be carefully investigated. Mimical spasmodic movements are foreign to the epileptic attack, coördinated convulsions are very rare, and even should they be present they are exceedingly monotonous and simple (rubbing, scratching, striking, etc.[1] In the hysterical seizure these mimical and complicated convulsions are never entirely lacking. This is also the reason why the hysterical spasm is much more variable in the same patient than is the epileptic seizure. I do not attach great importance to the reaction of the pupil for the reasons previously stated. The transitory presence of Babinski's sign, immediately after the attack, is in favor of epilepsy.

Examination during the interval free from attacks often reveals important points of support. The characteristic corporeal and psychical factors of hysteria, the hysterical stigmata, are never entirely absent during the interval, while in epilepsy analogous symptoms are never present. Only pressure-points are occasionally found in epilepsy, and exceptionally, especially in dementia epileptica, general analgesia. Among the psychical symptoms an acquired, progressive defect in intelligence is always in favor of epilepsy; only we must be quite certain that this defect in intelligence is truly acquired and that it is progressive. A stabile, congenital lack of intelligence may be present in hysteria; the latter affection not infrequently develops as the result of congenital weakness of mind, especially of debility.

Finally it must be stated that sometimes, in the course of genuine epilepsy, hysterical attacks and other hysterical symptoms and, *vice versâ*, in the course of genuine hysteria, typical epileptic attacks may occur. A gradual *transformation*, for example, an infantile epilepsy into hysteria may also take place in exceedingly rare cases. In all of these combined and transformed cases we refer to HYSTERO-EPILEPSY.

(*b*) **Chorea.**—The choreiform movements of hysteria may very closely resemble those of chorea minor. Hysterical choreiform movements are more often unilateral than those of chorea minor, but this is not sufficient for a differential diagnosis as the movements may also be most prominent upon one side; I have even met with pure unilateral cases. Increase of the choreiform movements upon intended innervation may often be demonstrated in both forms. It is by no means constant in chorea minor. Increase of the movements under emotional conditions is common to both affections. Of more importance is the influence of open or concealed suggestion (pressure upon the pressure-points) in the differential diagnosis: as a rule, this is slight

[1] Post-epileptic somnambulistic conditions are not included here.

in chorea, in hysterical choreiform movements it is often marked. Of more importance is the type of the movement upon accurate observation: in chorea in addition to combined movements there are also isolated ones as of a toe, finger, etc.; the movements also take place in much greater variation without connection in the most varied parts of the body; in hysterical chorea there is a tendency to an almost rhythmical repetition of the same movements and especially of combinations of these. In the history, a preceding infectious disease, or an acute endocarditis, is in favor of chorea. The association with an emotional disturbance is not absolutely in favor of hysteria, as this may also be an accidental factor in chorea minor. Imitative development decidedly points to hysteria. The demonstration of other hysterical symptoms is naturally of great significance, but it must always be borne in mind that chorea is particularly liable to develop in hysterical persons.

(*c*) **Neurasthenia.**—Well developed cases of both diseases cannot be confounded with one another. Apart from the pressure-points, which appear in both affections, all of the symptoms, especially if they are well developed, may be easily differentiated. Caution must be exercised so as not to confound the rare hypochondriac attacks due to fear and hypochondriacal impressions, which occasionally arise in severe forms of neurasthenia, with hysteria; for, here, sometimes very bizarre movements appear which resemble the hysterical attack. On the other hand the differential diagnosis may be exceedingly difficult in the undeveloped cases. It can only be stated that regional sensory disturbances (but not regional topoalgias), unilateral pressure-points, marked sensations of constriction and oppression, and a great variation in the mood favor hysteria; pathologic exhaustion and pathologic irritability favor neurasthenia. It must, however, be admitted that there are many transitional forms which have been called *hysteroneurasthenia.* Many cases of traumatic neurosis belong to this category.

(*d*) **With Organic Diseases of the Nervous System.**—In regard to this differential diagnosis I must state that the question should never be put: organic disease or hysteria? but always: organic disease, hysteria, or organic disease + hysteria? It is very common for hysterical symptoms to be "superimposed" upon an organic disease of the nervous system.[1] This possibility must always be considered in the diagnosis. The demonstration of a few symptoms of a hysterical nature (their capability of being influenced by suggestion) is never sufficient to make a diagnosis of hysteria, but a complete examination of the nervous system must be undertaken and each symptom investigated as to its hysterical nature. Very often, in this manner, reflex pupillary rigidity, chocked disk or optic neuritis, the absence of a tendon reflex, etc., will show that in addition to hysteria a severe organic disease is present.

The following organic diseases often bear a resemblance to hysteria:

Multiple Sclerosis.—Exaggerated tendon reflexes are present in both affections. The contractures of multiple sclerosis resemble those of hysteria, but develop more gradually. Foot clonus is sometimes simulated in hysteria as well as patella clonus. The intention tremor of multiple sclerosis is occa-

[1] These hysterical symptoms may have been present prior to the development of the organic disease, but the latter is often the causative agent of the former (thus in hereditary cases, but by no means exclusively).

sionally present in hysteria so that confusion is very likely to occur. If there is an undoubted Babinski reflex—I understand by this, that upon stroking the sole of the foot a dorsal flexion of the great toe, which must not be too rapid, without dorsal flexion of the entire foot and of the other toes takes place—is decidedly in favor of multiple sclerosis. The examination of the eye-ground is of particular importance, for optic neuritis is often present in multiple sclerosis; in hysteria the examination is negative; exceptionally a pseudo-optic neuritis may produce difficulties. The presence of dissociated paralyses of different nerves is very important: this is greatly in favor of multiple sclerosis. This is especially true of isolated paralyses of the ocular muscles. A history of genuine diplopia or its presence upon examination, therefore, favors multiple sclerosis, while in hysteria there is monocular diplopia and polyopia but not true diplopia. Feebleness of convergence may produce diplopia in hysteria, but this diplopia differs according to Parinaud from the organic variety in that it does not increase upon lateral movements. Well developed acquired nystagmus is present only in multiple sclerosis although a resemblance to the symptom may sometimes be noted in hysteria.

The same differentio-diagnostic considerations are important in *gumma of brain syphilis*. Here we must search for cranial nerve paralyses, pupillary rigidity and changes in the eye-ground. The history of a syphilitic infection is not sufficient to make a diagnosis of cerebral syphilis. In regard to the differential diagnosis from *dementia paralytica* I shall only remark that in dementia paralytica there are sometimes well developed superimposed hysterical symptoms; thus the paralytic disturbance in speech may act as the *agent provocateur* for hysterical stuttering, etc.[1] To obtain a positive decision, in some few cases, between cerebral syphilis and hysteria and between dementia paralytica and hysteria, I found it necessary to resort to lumbar puncture: both organic diseases—at least at the onset—show a pathologic lymphocytosis.

The differential diagnosis from *brain tumor* depends upon the ophthalmascopic examination of every case "no matter how functional the condition may appear." This is obviously not sufficient in those cases which do not cause chocked disk. But these tumors are characterized by the severity and tenacious nature of the headache in addition to heaviness and insomnia. Slow pulse is also in favor of tumor and against hysteria.

Focal diseases of the medullary layer and of the internal capsule may present difficulties in diagnosis. In this connection I must reiterate that a strict, typical hemianopsia does not occur in hysteria. In regard to hemiplegia and hemianesthesia we have already learned to recognize the factors which permit of a differentiation whether they are organic or of hysteric nature. In addition I must add the following: if an apoplexy, followed by hemiplegia, occurs in connection with an emotional disturbance, such as fright, hysterical hemiplegia should be thought of first, but I must remark that occasionally, although very much more rarely, fright may produce cerebral hemorrhage and cerebral thrombosis; I have seen a number of undoubted cases of this kind. The paralysis betrays its organic origin by the

[1] The development of dementia paralytica in a hysterical person is by no means uncommon. Naturally there is no connection between these affections but it may be readily noted how the hysterical symptoms are gradually replaced by the organic ones and finally substituted for them.

involvement of the so-called muscles of predilection, exaggerated tendon reflexes, Babinski's sign, modification of the patella tendon reflex, and finally by the gradual development of characteristic contracture and the implication of the muscles of the mouth supplied by the facial nerve. The sources of error and the exceptions have already been indicated. Somewhat more difficult is the differentiation of organic from hysterical hemianesthesia. The former usually increases distally. The peculiarities of the boundaries of hysterical hemianesthesia must also be considered. Hemialgia, so as to make the point quite clear, also occurs in organic hemianesthesia, such as is due to foci of softening in the posterior areas of the internal capsule.

Among the *affections of the spinal cord, dorsal transverse myelitis* is the one which is most likely to be mistaken for *hysterical paraplegia.* The condition of the tendon reflexes cannot, very often, be utilized, as the comparison with the opposite side gives us no points of support and a bilateral increase is often present in hysteria. Babinski's reflex is decisive and in favor of myelitis. Determining the upper limit of the sensory disturbance often gives us valuable aid. In lumbar myelitis the absence of the knee jerk and of the Achilles tendon reflex is sufficient for a diagnosis.

Great difficulties may arise in the case of *syringomyelia,* if, as sometimes happens, paralyses and muscle atrophy, which would at once reveal the organic nature of the malady, are absent as well as trophic disturbances. A thorough investigation of the sensory condition is then necessary. The more the limitation of the sensory disturbance corresponds to the areas of the spinal cord roots or the spinal cord segments, the more likely is syringomyelia. A very marked dissociation of sensation—abolition of the pain and temperature senses with retained contact sense—is in favor of syringomyelia, but exceptionally very similar dissociation is present in hysteria.

The characteristic limitation of the sensory disturbance is decisive in the case of *peripheral neuritis,* provided the diagnosis cannot be made from the involvement of the motor fibers, the degenerative paralysis, and decrease or absence of the tendon reflexes; besides in hysteria the characteristic sensitiveness upon stretching the nerve trunks (*Lasègue's symptom*) is lacking. We have already considered the differentiation of *true neuralgia* from hysterical pseudoneuralgia.

(*e*) **With Diseases in the Vicinity of the Central Nervous System.**—*Meningitis* and its varieties must be considered. Among the symptoms of the latter affection the rigidity of the neck, the headache, the vomiting, the delirium, and the convulsions, may be imitated by hysteria. The absence of fever and an irregularity of the pulse as well as paralyses of the basal cranial nerves is sufficient for a differential diagnosis. In addition the hysterical delirium differs in being more connected and also by its emotional admixture. As a final diagnostic resort lumbar puncture may be employed.

Among diseases of the spinal cord care must be exercised in differentiating *diseases of the vertebræ,* such as the *tuberculous* with hysterical *rachialgia.* The pain in the latter is even more severe than in the former. Contact and passive motion, which do not involve the vertebræ that are affected, are associated in hysterical rachialgia with active pain. On the other hand in hysterical rachialgia the characteristic girdle root pain is absent. The regional

hyperalgias, which often accompany hysterical rachalgia, must not be forgotten.

(*f*) **With Diseases of Other Organs.**—The regional topoalgias give sufficient cause for mistakes of this kind. First the *articular topoalgias* must be considered. I advocate a minute surgical examination in all of these cases. If there be any doubt an X-ray examination is necessary. If there be any suspicion of tuberculous joint inflammation careful temperature records are necessary. I am in doubt whether there are a greater number of reckless operations performed on account of an inconsiderate diagnosis when pure hysterical topoalgia is present or whether the necessary operation is omitted more frequently from a hasty diagnosis of hysterical topoalgia. For this reason alone a minute surgical examination is required. If no surgical affection is present the well known characteristics of hysterical topoalgia, as a rule, are sufficient for a correct diagnosis. Other hysterical topoalgias may simulate gall stone disease, a gastric affection, angina pectoris, a peritonitis, etc. In these instances all of the diagnostic aids known to internal medicine are to be invoked, and only then should a diagnosis of hysterical topoalgia be made by exclusion. I cannot enter upon a minute differential diagnosis of these conditions, they will be found in the respective chapters of this work dealing with the separate maladies.

There is often the greatest difficulty in the differentiation of hysteria from **malingering**. The hysteric impression of "not being able to walk" cannot be so readily discerned from the "not being willing to walk" of the malingerer, as it might appear. It finally depends upon this: whether or not in the occurrence of the symptom under consideration a conscious conception has been active from which advantage is to be expected. Where this is the case an attempt at aggravation is the motive. If the conscious conception which produces the symptom, from which an advantage is expected, is the only cause of its presence, the case is one of simple malingering. Practically when we are considering the question "simulation or hysteria" we must investigate whether the report of the patient is contradicted by the actual psychic condition present, for example, whether he maintains, against his better knowledge, that he cannot walk, etc. I cannot describe the different methods which are in use to obtain this knowledge. Most of those in vogue, to unmask simulated disturbances in sight and hearing, cannot be employed in this condition. If we determine, in the well known manner, that a person who simulates right-sided deafness nevertheless hears upon the right side while we make him believe that he is hearing with the left ear, whereas the sound wave actually strikes the right ear, this argument would not be applicable in hysteria, for here the entire disturbance is dependent upon impression: a hysteric who suffers from hysterical deafness upon the right side must, if he is able to appreciate sound waves in the right ear, hear these, although he may believe that only the left ear is capable of appreciating sounds. It is necessary to point out that simulation of the entire pathologic picture can never be demonstrated from the proof of simulation of a few symptoms. Exaggeration and aggravation are so common in hysteria that we must always reckon upon a combination of actual and simulated symptoms or with an exaggeration of actual symptoms. We may only speak of complete malingering when we are able to demonstrate that each separate

symptom is simulated. Sometimes the exaggeration itself has a pathologic character. I need only refer to the pathologic distorted simulation in Ganser's hysterical somnambulistic condition.

Let us assume that we have made a diagnosis of "hysteria," it now becomes necessary to develop a plan of treatment.

TREATMENT

The therapy of hysteria is dominated by two main principles, the *etiologic* and the *psychotherapeutic.*

The *etiologic* principle requires counteraction of the causes of hysteria which is, at the same time, the method of prophylaxis. This primarily requires, as the hereditary predisposition as such cannot be eliminated, a *proper education* in the threatened, but not yet affected, child and often enough also in the diseased adult, so as to obliterate deleterious influences. Above everything the child must become accustomed to control its feelings and emotions, especially the negative ones. This is in consonance with our general interpretation of hysteria, of which, according to our description, the abnormal action of exaggerated emotional conceptions is characteristic. Translated for practical purposes this simply means that the child must not be indulged, that its little pains and aches should be ignored, that it must be callous to such irritants as slight pain, hunger, cold; it must be taught to subjugate its will and convenience to that of others and learn to observe foreign interests. Even in married adults there is too great neglect of this requirement, and an elimination of these deleterious influences of education, that is, the behavior of those in contact with the patient, is a prerequisite, in these cases, for therapeutic success. It is self-evident that this process of "*hardening*" must not be overdone, above all, it must be methodic and uniform. It is of the greatest importance that the parents set an example to the child in the control of their emotions. From this point of view it will be readily understood that such children are ordered cool ablutions, etc. The essential element is the control of the emotional state, not the hardening of the body. Gymnastics, swimming, skating, etc., have a similar purpose: the child must learn to control its fears of slight danger.

In another direction also education is capable of removing an etiologic, deleterious factor. It is very significant that not emotional sensations, but above all exaggerated, emotional impressions are of pathologic influence in hysteria. Pathologically increased activity of the imagination, we have seen, is a chief symptom of the hysterical, psychopathic constitution. Here also education must step in. A child, that is threatened or already attacked by hysteria, should not be permitted to read books that stimulate the imagination to any considerable extent, whether these be fairy tales or Indian stories. The hysterical adult, for the same reason, should not be permitted to read exciting novels. On the other hand, it is very good practice to accustom the child to observe phenomena of nature. It may build with blocks, draw, collect stamps, stones, plants, insects, etc.; cultivate the garden, have an aquarium and so on. These pastimes may also be followed by adults. All spare time is to be devoted either to rest or to *objective* observation. Hand-work is not suitable for it allows the patient to lose himself in phantasies and broodings.

Translating and abstracting is much better employment. Play and association with others is very effective protection against the egocentric limitation of the hysterical life of impressions. Cultivation of the habit of nature observation upon walks is particularly important, for upon such occasions there is most likely but little opportunity for the patient to indulge in useless dreamings. The theatre is to be prohibited. It is also obvious that public schools, in general, are to be preferred to private instruction, for the former gives a much better opportunity for association and the necessary process of hardening.

Among the other etiologic factors of hysteria there are none that require special treatment. Therefore we may be brief, and but a few points need be discussed. If there is a severe, nutritive disturbance Weir Mitchell's treatment may be employed. Should a chronic intoxication be an etiologic factor this must naturally be combated. The question will often arise in the female whether the treatment of a genital affection is not indicated from the stand-point of etiologic therapy. In the discussion of the etiology it was expressly stated that gynecologic affections are not of essential importance as factors in the etiology. This coincides with the experience that only very rarely does gynecologic treatment produce an essential or lasting improvement not to speak of a cure in hysteria. Even in the few instances where improvement has followed operation it is probable that the operation merely had a suggestive effect. On the other hand, there are many cases in which gynecologic treatment, especially if long continued (such as internal massage), has materially increased the hysterical symptoms. Urgent indications for gynecologic management must of course be fulfilled in spite of the existence of hysteria, but hysteria neither indicates castration nor any other gynecologic treatment; on the contrary, caution is necessary and no gynecologic measures that are not urgent should be attempted. Only when it has been determined, after rigid investigation, that the gynecologic symptoms are regularly and markedly aggravated from the presence of some genital malady (such as retroflexio uteri) is it advisable to treat this malady even without urgent indications. Not with the expectation of curing the hysteria, but only with the view of relieving a factor which might hinder the treatment. If at all possible a rapid method should be employed, perhaps even operation (such as ventrofixation of the uterus), for the irritative effects of chronic treatment are not without serious consequences. There should be no unnecessary gynecologic examinations, particularly in the case of virgins, for this very often brings about a decided aggravation of the symptoms.

The question often arises whether emotional conditions, brought about by occupation and the affairs of daily life having a tendency to keep up the malady, cannot be removed. Sexual relations, dissatisfaction in the occupation, marital difficulties, etc., come under this head. The "process of hardening," to which we referred previously, is too late for these cases. Here the physician must decide each individual case according to its merits, and after a minute investigation and after having exhausted all other methods of treatment must explain to the patient and his friends the chances of a change in occupation, etc. Only from my own experience I should advise against a too sanguine opinion in this respect.

In this connection the very important question arises whether hysterical

persons "may" marry. It is an undoubted fact that marriage often has an unfavorable effect, that the hysterical symptoms increase and that the hystero-psychopathic constitution leads to the greatest marital misfortunes. It is rare that a favorable influence is noted. From this condition of affairs it is not justifiable to advise all hysterical persons against marriage. In fact the physician is not to "advise," but to state quite objectively the dangers of marriage for the patient and for the possible offspring. In weighing these dangers not merely the severity of the symptoms but also the entire external situation in the contemplated marriage, the personality of the husband, etc., are to be taken into consideration.

If the outbreak of the manifest, severe symptoms is associated with a special emotional shock, such as relates to the sexual sphere, which acts in the sense of a latent, exaggerated, memory picture, the patient should be permitted, at the first examination, to explain the entire emotional cause as fully as she desires. This is sometimes an absolute requirement for a cure. By continued questioning and a repetition of the same answers, as advised by Freud and Breuer, so that all of these latent memories are refreshed, very little good can be gained; I have repeatedly observed that the "hyper-imagination" of the patient constantly enlarges the original experience, sometimes even in the most dangerous manner, without decreasing the emotional emphasis of the memory and without improving the other hysterical symptoms.

After these etiologic therapeutic requirements have been fulfilled and all deleterious factors have been removed in so far as is possible, treatment in a positive sense is to be instituted. These positive measures of treatment are almost exclusively *psychotherapeutic*. In our previous discussion, in regard to the removal of deleterious factors in the rearing of the child, we have discussed a few of these curative factors. The first practical question which must be put in every case is whether the patient may remain in his previous surroundings. Very often the reply to this question must be in the negative. The patient must be placed under favorable psychical conditions. For this purpose a visit to trustworthy relatives or friends, or—in the case of youthful hysterics—a suitable *pension*. In severe cases the patient should be sent to an institution, so that the psychotherapy may daily and hourly be under the direction of a specialist in nervous diseases. Such institutions are private and public hospitals for nervous disease, psychiatric and nervous clinics; but general hospitals are available only in so far as the directing physician is familiar with nervous diseases and psychotherapy. In rare cases, under especially favorable circumstances, isolation may be attempted at home, but my experience in this direction is not very favorable. In many instances the change of residence cannot be considered for pecuniary reasons. Secondly, the question must be considered whether the attending physician should carry out the treatment himself. The answer to this question depends upon whether the physician is conversant with nervous disease and psychotherapy and whether he possesses sufficient authority in the special case. It is undoubted that the family physician often has less influence, because the patient is accustomed to him, than a strange physician. Very often incidental qualities of the physician are of the greatest importance in obtaining this psychical influence over the patient. It would be a great mistake to attempt to carry

the treatment on in a severe case in the absence of this personal influence. It is quite remarkable how often a change in physician brings about surprising improvement in the hysteric. The "*cedo meliori*" in such cases is nothing of which the physician need be ashamed. Thirdly, in severe cases the nurse is of the greatest importance, for the personality of the latter is often of vital importance upon the result of treatment. Naturally the individual circumstances are decisive in the choice of a nurse, but in general it may be stated that above all absolute conscientiousness in carrying out the orders of the physician is the first requisite. Nurses who allow themselves to be controlled by the patient and who become intimate with their charges are usually not to be trusted.

After these practical, preliminary questions of psychotherapy have been settled a proper manner of living is ordered for the patient in which all of the etiologic indications are considered so far as possible. In severe cases it is best to give a *written, hourly plan*; regular hours for bodily and mental occupation and for rest. In the choice of employment the principles developed above are decisive. Very often under this plan of treatment the malady improves greatly without other curative measures. But it is often necessary to treat special symptoms, and this treatment is largely suggestive. Whether the vehicle of suggestion be a drug, a mechanotherapeutic, electrotherapeutic, or hydrotherapeutic procedure, it remains the same in principle. It depends merely upon the suggestive factor and this again is entirely dependent upon the suggestive authority of the physician who orders the drug or the process. It is self-evident that a vehicle is chosen that has not already been employed unsuccessfully in the special case. The more educated the patient the more it is necessary to employ concealed suggestion. Naturally a pill may be ordered or the electric current or massage employed and the physician declare in an authoritative tone: "this will cure the paralysis"; however, such a crude suggestion will not be effective in the case of an educated person. Therefore, a more cautious method must be utilized; for example, without previous preparation the paralyzed muscles are caused to contract by the electric current, and then very casually the action of the current should be emphasized. If there is no actual paralysis but merely paresis, skillful exercise treatment, in the sense of suggestion, should be employed. It is of advantage to test the muscular power by the aid of the dynamometer from time to time without concealing the differences from the patient. In paresis of the legs and abasia "walking-frames" may be employed. I have often used these exercises with success even in absolute paralysis. In such instances I have previously stated to the patient: "Naturally your exertions will not show immediate motion, but it is of great advantage if the paralyzed muscles are stimulated at all; they gradually become strengthened and some day motion in the part will appear." Passive gymnastics and resistance gymnastics are often serviceable in the sense of suggestive therapy. Contractures with and without accompanying paralysis often present great difficulties in suggestive treatment. Here passive gymnastics as well as electricity and massage of the antagonists of the contracted muscles is of use. Prolonged baths with exercises (in the bath) are sometimes valuable. Forced overpowering of the contracture and immobilization in a new position should not be practiced as bandages of this kind often produce contractures in

hysterical persons and we merely risk a new contracture in another position. The artificial production of a hysterical spasmodic attack—in the hope that after the attack the contracture may disappear—I utilize only in the most desperate cases.

Inhibitive exercises are of use in combating the various forms of spasm. It would be quite superfluous to indicate the suggestive remedy for each individual hysterical symptom. In the text-books and special monographs countless drugs, electric, hydriatic, and other measures will be found. The vehicle of suggestion in itself is never decisive. I shall explain this by an example of hysterical vomiting. Sometimes the use of a drug will relieve the condition. Whether this be sugar, chloral, bismuth or an ice pill is immaterial if success is attained. If the medicine fails relief may be obtained by suitable diet. If this also proves useless, massage, the thermophore or faradization of the gastric region may be tried. Gastric lavage used once or several times is sometimes effective. Pretended operations, which have occasionally been used for this or that hysterical symptom, had better be avoided. In some very tenacious cases I have prohibited food until 1 o'clock in the afternoon, and then, at 1 o'clock, when a—very scant—meal was given the patient it was not vomited. In another patient the vomiting ceased when I had her fed in the bath.

In addition to the suggestive therapeutic methods that have been described there is also the so-called "*harsh*" *method.* This consists in the physician suddenly giving an energetic command to the patient which relieves the symptom at once. For instance, the abasic patient is commanded, in a harsh tone, to walk. On the whole I have had very little success with this method. It is perhaps most suited to the hysteria of childhood.

Another method, in a certain sense the direct contrast to the previously mentioned one, is more effective: deliberately ignoring the symptom that is to be treated, a "*purposeful neglect.*" I utilize this method by first examining the patient very carefully, then give a plan of treatment for each hour and then ignoring all of the symptoms for weeks. At the onset I usually state to the patient "the treatment must now continue for some weeks without change in the manner just outlined, in spite of any of the symptoms; rapid improvement is not to be expected, but only this method will prove of benefit; frequent examinations and complaints are, therefore, without purpose." Exceptionally I may add that the patient should aid in ignoring the symptoms until the treatment begins to be effective. It is rarely advisable to go further than this and to reject all forms of treatment, at most to give a plan for each hour or directions as to employment and the manner of life, ignoring all of the other details. It is self-evident that this method must also be followed by all of the relatives, friends and nurses and—in the hospital—by the other patients, otherwise there will be absolute failure. In the hospital I generally isolate these patients. Déjérine has even developed this "isolation method" still further.[1] This *process of ignoring* must also be required of those about the patient in cases in which the physician employs the suggestive method and not the medical method of ignoring. Exag-

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[1] Here other curative factors also come into consideration, such as avoiding irritation, etc.

gerated sympathy and care on the part of parents often serves to increase the pathologic, hysteric impressions. We have, therefore, seen that removal of the patient to other surroundings is a necessary measure for cure. Now the method has been described in the treatment of those cases in which this requirement cannot be fulfilled; we must then require of those about the patient that the process of ignoring be strictly maintained. This need not bear the character of lack of sympathy: brief, not too frequent, sympathetic, but quiet inquiry of the patient's condition may continue; I often permit the relatives to say to the patient about as follows: "the physician has assured us that there is no danger, but he has said that we must be prepared for a long illness and that it will take some time before the first signs of improvement appear; he has, therefore, forbidden us to inquire daily in regard to your condition; he will inform us himself." It is understood that this treatment of ignoring on the part of the physician and the friends must be continued even if there be at first an increase of the symptoms, spasmodic attacks, etc., after the method has been tried for a short time.

This method unquestionably very often bears good fruit. The question, therefore, arises when should the suggestive method be used and when the method of ignoring? If there is actual danger of suicide or a very conspicuous tendency to hyperphantastic illusions there is an absolute contraindication to the latter method. In fact, in the case of a very pronounced hysterical, psychopathic constitution or with a fully developed hysterical psychosis, we must be exceedingly cautious with treatment by the method of ignoring; it must be either dispensed with entirely or at most greatly modified. But the treatment may be employed in the milder form, previously described, in all cases in which the suggestive method does not produce immediate results. In these milder forms the method of ignoring is constantly associated with suggestive factors, and the plan of treatment in regard to hourly employment is persisted in, which on its part depends entirely upon suggestive effects. The only difference consists in this, that the suggestive effects of the hourly plan are not constantly urged, but on the contrary are combined with the psychical influence of the purposeful ignoring. I believe, therefore, that the methods do not exclude one another, but according to the varying conditions in the individual case must be combined.

Up to this time I have purposely avoided the mention of the special treatment of the hysterical, spasmodic attack. Here also all of the therapeutic factors which we have mentioned as valuable for the permanent symptoms are in place. Exceptionally the attack may be surpassed by gruffness. A suitable suggestion has a better effect. This, as we have seen previously, may be connected with one or the other hysterofrenic pressure-points. Cool ablutions or the faradic brush may be tried in the same sense; but here the gruffness is more important than the suggestive factor. These measures must never have the appearance of punishment. In most instances ignoring the attack is at the same time the best form of treatment. We only interfere if in the jactitory or delirious stage the patient threatens to injure himself, and after having reassured the friends as to the harmlessness of the attack, they are sent into another room. It is unnecessary to administer drugs. Hypodermics of morphin are absolutely contraindicated.

In case these general measures in treatment are insufficient to bring about

the disappearance of the attacks, I believe it to be good practice to relieve the emotional irritability, which favors the appearance of attacks, by suitable drugs. The *bromids* are especially useful in this respect, given in small doses. Large doses, such as are administered in epilepsy, are not merely superfluous,[1] but are likely to produce bad results, for hysterical persons not infrequently react to large doses of the bromid with delirium. Doses of 1–2 grams of sodium bromid are given to reduce the emotional irritability, which is very likely to control the attacks and thus aid the other curative measures. Morphin and other narcotics are absolutely prohibited, even in a prophylactic sense, for hysterical persons are exceedingly apt to become habitués.[2]

Finally, at the conclusion of our therapeutic considerations it will be asked whether *hypnotism,* that is suggestion during hypnosis, is to be employed in treatment, as is maintained by so many professional hypnotizers and unfortunately also by some physicians.[3] I must state, briefly, that hypnotism should only be considered as an *ultimum refugium.* Hypnotism, no matter what the professional hypnotizers may state, is often directly harmful. The special directions which are supposed to render hypnotism harmless are to be regarded as humbug. In children hypnotism is not to be permitted under any circumstances. Only after all of the other methods of treatment with which we have become familiar, after conscious employment have failed, may hypnotic suggestion be considered; it is then urgently necessary to state to the friends that the method is by no means harmless, that mental disturbance or new symptoms may arise in connection with the hypnotic treatment. The more I see of the treatment of hysteria the more I am convinced that hypnotism may be dispensed with in the treatment of the affection. Hypnotism does not bring about what must be considered as the chief aim in the treatment of the malady corresponding to the underlying condition of the hysterical change: *restitution of the psychic equilibrium by the removal of the tendency to pathologic, active, hyperemotional impressions.*

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[1] It is characteristic of hysteria, in contrast to epilepsy, that large doses of the bromids do not decrease the number of attacks, at least not more so than do small doses.

[2] For the same reason I prohibit the use of alcohol entirely.

[3] These persons always maintain that "their" hypnotism is never harmful and has never injured anyone.

TRAUMATIC NEUROSES

BY P. SCHUSTER, BERLIN

I. HISTORY

AT the beginning of this article a definition of the term traumatic neuroses may, as a matter of course, be expected. But, if we review the historic data which mark the development of our pathologic conception of traumatic neuroses, it soon becomes evident that a definition cannot be given in a few words, therefore we at once turn to the historic facts. A study of the history of traumatic neuroses cannot be omitted, being both interesting and instructive; interesting because the development of the pathologic picture of traumatic neuroses largely represents the total development of neurological science within the last decades; instructive because the development of differential diagnosis in almost every individual case is a miniature reflection of all development of pathologic conceptions. Therefore in the diagnostic ontogenesis of any case the phylogenesis of the pathologic *conception* is always kept in view.

In a work, "Upon Injuries of the Central Portiòn of the Nervous System," etc., published in London in 1866, Erichsen first called the attention of physicians to certain pathologic conditions following injury, especially after railroad accidents. Erichsen regarded the symptom-pictures as organic, and believed the seat of the pathologic processes to be located in the spinal cord. A few years later he expressed the same view in his book "On Concussion of the Spine." To Erichsen, therefore, the designation "railway spine" is to be attributed.

It was quite natural that those physicians, who, following Erichsen and stimulated by his publications, made a specialty of diseases occurring after accidents, should share and retain the views of the originator. Among his followers in Germany were Leyden, Erb, Bernhardt, Westphal, and Rigler (Ueber die Folgen von Verletzungen auf Eisenbahnen, Berlin, 1879), who devoted themselves to the study of the newly described pathologic picture, and accepted Erichsen's views.

Moeli's report in 1881 led to the adoption of a more advanced theory in place of the original pathological conception. This report, which deals with "psychical disturbances following railroad accidents," was the first to introduce a *psychical* element into the pathology of traumatic affections. Another step in the direction indicated was taken by Wilks (*Guy's Hospital Reports*, 1883) and Walton (*Arch. of Med.*, 1883), who described hemianesthesia as a symptom of trauma, as a rule functionally produced. In the two articles upon sensory disturbances which he published in 1884 (*Berliner klin.*

Wochenschr. and *Arch. f. Psychiatrie*) Oppenheim attempted to base the pathologic picture upon the sensory disturbances observed at the onset. In the latter of these articles Oppenheim (to whom Germany is indebted for the great advance in the understanding of traumatic nervous disease) absolutely rejected Erichsen's view of the spinal nature of the pathologic picture, and maintained its cerebral localization; he also brought out strongly the many functional features of the pathologic picture. But in 1886 Charcot took the most important and final step in advancing our knowledge of the disease by stating that the entire pathologic picture was functional and psychical. In 1883 Page, an English author, in the first edition of his book "Injuries of the Spine and Spinal Cord and Nervous Shock," expressed views which extraordinarily resemble those of Charcot. Although Charcot's idea of the nature of traumatic diseases to-day unquestionably molds the views of most neurologists, yet the history of the development of our pathologic conception was not completed with Charcot's teaching. Subsequently the view of the functional nature of the disease was generally adopted, but there was a want of unanimity concerning questions of secondary importance. An originally calm and purely scientific consideration of the question gave way to a remarkably warm polemic, which was not always confined to the subject. The discussion began about 1890, and the most important point was no longer the general medical one, but the special neurologic cause of the pathologic symptoms.

Charcot in his "New Lectures upon Diseases of the Nervous System" considered the nervous symptoms arising after trauma as purely hysterical. He therefore did *not* assign to these symptoms any special nosologic importance. Oppenheim's celebrated treatise, which appeared in 1889, was based upon a diametrically opposite view. In this he absolutely maintained the functional nature of the pathologic picture, but he objected to the opinion that the "traumatic neurosis"—as he called the clinical condition—should be regarded as hysteria nowise different from other forms of hysteria except in its traumatic origin. Oppenheim's theory that the traumatic neurosis was a peculiar neurosis rapidly found acceptance among general practitioners, but there was much opposition on the part of our most celebrated neurologists. Schultze, Jolly, Eisenlohr, Mendel, and others denied the specificity of traumatic neurosis as a disease *sui generis,* and claimed that, instead of the schematic designation "traumatic neurosis" for the existing symptom-complex, the designations traumatic hysteria, traumatic neurasthenia, or traumatic hypochondria, etc., according to the type, should be substituted. Oppenheim did not absolutely deny that there was truth in the objections of his opponents, but he strongly emphasized that the peculiarity of his pathologic picture of traumatic neurosis was the peculiar admixture of symptoms which originated entirely from this neurosis. On this point complete unanimity of opinion has never been attained. I believe, however, that the views enumerated above are not so diametrically opposed as discussion would sometimes make them appear to be. Undoubtedly we must agree with Oppenheim that many cases of the affection may be differentiated from non-traumatic cases of hysteria, hypochondriasis, etc., by certain common symptoms, but these differentiating factors are non-essential, and they depend less upon the *intimate nature* of the neuroses than on external circumstances.

To illustrate this, we may briefly refer to accident insurance under the control of the State. The immediate and mediate *psychical* effects of the filing of claims for damages on the part of the injured, and the practical insistence on these claims, is primarily, although perhaps not wholly, the factor which gives to the clinical physiognomy of accident neuroses their definite stamp. One of the most important results of these psychical effects is the "conception of indemnity" (for damages) which Strümpell brought prominently into the discussion. Besides the question of the specificity of traumatic neuroses, which is of more scientific than practical importance, in the last 15 years practical and significant issues have been discussed; namely, the objectivity of the pathologic phenomena and the question of malingering. In the discussion of the objectivity of symptoms, sensory disturbances have filled an undeservedly wide space.

In regard to simulation, which some observers regarded as unlikely or rare, and others as extremely common, in the course of time we have arrived at unanimity. To-day nearly all authors maintain the importance and frequency of exaggeration, while they admit that a close simulation of the entire pathologic picture is rare.

The conviction is steadily gaining ground that the psychical genesis of most of the symptoms explains why there are so few strictly objective phenomena. Among those independent of the psychical sphere are the numerous symptoms on the part of the vessels, conditions which—strictly speaking—are not actually symptoms but complications of traumatic neurosis. In 1889 Sperling and Kronthal were the first to call attention to arteriosclerotic phenomena in the pathological picture of traumatic neuroses, and to demonstrate them by anatomical proofs.

Friedmann (1891–93) referred a number of symptoms (marked vertigo, etc.) to vasomotor disturbance in the interior of the skull, and proved that these conditions were due to changes in the small cranial vessels and their surroundings. Subsequent investigations upon an experimental basis were made by other authors, and these made it appear likely that other symptoms of traumatic neuroses which are not observed in non-traumatic hysteria, neurasthenia, or hypochondriasis, may be regarded as organic. By an animal experiment performed in 1896 Schmaus showed that shock to the vertebral column produced small foci of degeneration in the spinal cord and changes in the axis cylinders. There is nothing to prevent the same demonstration in human pathology. It cannot have escaped attention that in applying Schmaus's experiment to human pathology, the same conditions are produced as exist in so-called *commotio cerebri* and *commotio medullæ spinalis*. Therefore Schmaus's experiment has illuminated the clinical and pathologico-anatomical border-land between cerebral shock and traumatic neuroses.

In the discussions of the Wiesbaden Congress of Internal Medicine in 1893 there was an apparent tendency to regard certain severe traumatic neuroses as also due to organic change (which might be assumed from Schmaus's experiments); subsequently the same inclination was noted in other authors, especially Strümpell, Vibert, and Stepp.

After this, opinion apparently drifted back to where it stood in 1868. In reality our comprehension of the nature of traumatic neuroses has made great advance in the last thirty years, as I have previously attempted to

prove from the valuable historical data referred to. It is true our pathologic conception is not yet perfect. It may not be irrelevant to mention Hoche's opinion that the pathology of rare electric injuries may give us a further insight into the nature of traumatic neuroses.

On reviewing the history of these affections, we will more minutely define our pathologic conception, and will state that by the designation traumatic neuroses we mean pathologic pictures which are either cases of pure hysteria, neurasthenia, or hypochondriasis due to trauma, or cases which, besides showing these neuroses, also exhibit slight organic changes, or, finally, cases which reveal the same combination of symptoms of hysteria, neurasthenia, and hypochondriasis which is peculiar to some of the non-traumatic neuroses.

II. ETIOLOGY AND PATHOGENESIS

In discussing the etiology and pathogenesis of traumatic neuroses we shall consider only the purely functional pathologic picture. The etiology of the phenomena due to organic changes (changes in the circulatory system, hemorrhages into the central organs and their membranes, destruction of nerve substance, etc.) does not come within the scope of this article.

As in all other diseases, in the pathogenesis of traumatic neuroses there are a number of causative factors, and all those predisposing agents which play a rôle in the development of other nervous diseases must here be considered. Chief among them are chronic *abuse of alcohol,* chronic *metallic poisoning* (above all, lead poisoning), and a number of *occupation injuries* such as those due to working in intense heat, near noisy machinery, and those in which the duties are combined with great responsibility or in which there is rigid discipline. In addition there are a few other predisposing factors which are especially significant in the cases with a purely psychical pathogenesis; namely, the mental or *intellectual inferiority* of the injured, as well as the baneful influence of those about the patient, and the evil effects of instituting a claim for damages.

As is evident from its name, the most significant factor in the etiology of traumatic neuroses is trauma; the more serious the accident, and the more extensively the central nervous organs or their coverings are implicated, the sooner will traumatic neurosis develop, and the more severe will the symptoms *usually* be. I emphasize "usually." For the etiology of traumatic neuroses depends not so much upon the *objective* severity of the accident as upon its subjective consideration by the injured person. This statement is based upon the fact, repeatedly emphasized, that the development of traumatic neurosis is generally psychogenous, but it by no means indicates that the pathogenesis is the same or even similar in all cases.

Sometimes the disease develops *acutely* after emotions which appear and disappear suddenly, such as fright, fear, anxiety, and, more rarely, anger. These emotional conditions either accompany the trauma or immediately precede it. Etiologically they become most active when they are simultaneously experienced by many persons, as in great theatre fires, in railroad accidents, in earthquakes, in school panics, etc., in which circumstances severe traumatic neuroses frequently arise.

That after such violent emotions psychical shock and not bodily injury

is most active, and that not objective trauma but its subjective reflection in the psychical life of the injured one causes the development of the neurosis, is proven by hundreds of experiences. For instance, I observed the motorman of an electric street car who became seriously ill after being struck on the head by the conducting wire, which, it transpired later, belonged to a "live" wire, but no current was then on. We may thus understand that the etiology does not wholly depend upon actual external trauma, for this may apparently be absent, but that a purely psychical trauma, like psychical shock, may sometimes be sufficient to produce a traumatic neurosis. In addition to acute traumatic neurosis from psychical causes, there is also a *chronic genesis* for this affection, which is undoubtedly more common. It may appear alone, or may be combined with the former mode of development.

When the disease develops gradually and completely, the beginning of the process—namely, the trauma—is acute. At the onset, there may be neither emotion nor psychical irritation. Pain, more or less impairment of function, and the inconvenience thereby produced, now cause the mind of the injured person continually to dwell upon the local injury, the accident itself, and everything connected with it. Sleepless nights following the accident, as well as the first days of incapacity for work, are spent by the injured person in meditating upon his position. As recovery is not so rapid as he expected he gradually becomes anxious about the future. At this period, in which a favorable psychical influence is usually lacking, relatives and friends come forward with diagnostic and prognostic utterances, and, with the love of the uneducated for sensational tales of injury, identical and similar cases are related to the injured person in pessimistic strain. Each individual case "in which externally nothing was to be seen," but in which there must have been "severe internal injuries," is magnified until finally the injured person demands indemnity for his injury.

The moment the question of damages arises, the wife of the injured man usually seems to exert upon him an extraordinary and baneful influence. She has listened to conversations between her husband and his friends and fellow-workmen, has enlarged her understanding of the case by discussing it with her neighbors, and now invariably persuades her husband to demand indemnity for the injury he has sustained. As a rule, neither the wife nor the injured man has in mind compensation merely for the time during which it was impossible for him to work. On the contrary, a sense of justice impels them to seek redress.

The time arrives when the physician in attendance pronounces the man well and advises him to return to his work. The injured goes to his place of employment after being cautioned by his friends and relatives to take care of himself and not to attempt to do too much. As a rule, on the same day, or, at the latest, after a few days, he returns saying: "It is impossible, I cannot work." He seeks lighter employment—as a rule without success; if he obtains it, he soon abandons it. He does not attempt to overcome a slight disability by continuing his work, but clings to his own idea that he is unable to work. He feels himself justified when he declares that he is absolutely unable to work; therefore he demands damages. Subsequent professional examinations, the inquiries and communications of his coöperative associations and of the courts (which, even under normal circumstances,

are somewhat exciting to the workman) highly inflame the mind of the injured man, and he and his adherents regard them merely as attempts to deprive him of his just deserts. His mind becomes embittered, he is quarrelsome, he devotes special attention to the results of his accident, and nourishes these as proof of his rights. In describing his case in the courts or before his labor union, all the details are now grossly exaggerated. He speaks of "dreadful pains," "absolute weakness of memory, and of mania," "whole nights without sleep," "great nervous depression," etc. As soon as he has made these statements in court or lodge, either verbally or in writing, he becomes absolutely convinced of their truthfulness.

The strongest confirmation of his views is the patient's distress because he cannot work, distress not due to consideration and thought, but to want of money which now makes itself felt. If the unfortunate man, because not working, has suffered from hunger (i. e., if he has once suffered from the idea which possesses him), the constant thought of his disease and the sense of his inability to work cannot be removed by argument or by persuasion; nothing will dispossess him of this idea, not even the granting of the full damages claimed. A pathologic mental state has gradually supervened, a distressing form of hypochondriasis.

That this picture may not be too one-sided and distorted, and to avoid injustice, I must remark that the injured person sometimes more strongly resists the development of the disease, and from a sense of duty and native energy he sometimes masters the threatening affection. But these cases are rare.

In the opinion of some authors, the *acute and chronic traumatic neuroses* show no other mode of development than the psychical, which has just been described. As opposed to this, attention must be called to special forms of traumatic neurosis without discussing their psychical or non-psychical pathogenesis. Cases occur in which the injuries are purely peripheral and either severe or moderately severe without general shock to the body or to the central organs, and in which no emotion of any kind, especially fright and psychical shock, had appeared prior to the accident, and in which the slowly acting psychical deleterious change just described could not be demonstrated.

In such cases (in which, according to existing views, there is no justification for speaking of a neurosis due to autosuggestion or to any similar psychical agency), motor, sensory, and even trophic changes are often observed which are strictly limited to the region of the trauma. In these cases the injury or shock to which the peripheral nervous apparatus has been subjected almost invariably appears to have been propagated by the nerve tract to the spinal and cerebral centers, or has reflexly produced a pathologic change in the centers of the affected peripheral tracts. Cases of the kind, which closely resemble typical hysteria, are commonly observed after electric injuries. Whether, as suggested above, their pathogenesis is chiefly a purely somatic process of unknown "molecular" nature, or whether in spite of the apparent absence of any psychical shock psychical processes are active, cannot now be determined. At all events the condition must be attributed to a psychical genesis if the accident caused absolutely no conscious psychical shock. For an external and subsequent psychical effect is possible even when there has been no *conscious* emotion at the time of the accident. We are

accustomed to think of psychical effects only when the patient *has suffered* from fright, shock, or other emotion. But experience with hypnotism, with conditions during sleep, and also in states of subconsciousness, has shown that an external factor like trauma may under some circumstances subsequently produce psychical effects of most complicated nature and extent in cases in which there is absolutely no question of a *conscious* psychical impression of the trauma or of a *conscious* beginning of the psychical process it produces. Here I must call attention to another condition which is not only most significant in the psychogenesis of the cases last described, but in the psychogenesis of all traumatic neuroses, and in my opinion is not sufficiently appreciated.

As shown by superficial observation every sensory impression produces not only a quantitative but also a qualitative effect, which varies even in the same individual. This effect may at different times be differently experienced, and may lead to absolutely unlike emotions. For the effect does not depend upon the nature of the stimulation, but is determined to some extent by the nature of the area stimulated; i. e., it is determined by the existing functional condition of the brain. In other words, the perception and distribution of every sensory irritation depend to a great extent upon the condition of the psychical functions at the time the irritation was conveyed. Obviously it makes a difference whether the irritation is expected and thus is met by a brain prepared for it, or whether it is received unexpectedly by an absolutely unprepared brain. The condition of "expectation" or "non-expectation," as stated, furnishes a crude illustration of the extremely varying psychical conditions which may exist at the time of the trauma. Just as the prick of a needle may be differently experienced, according to whether the attention of the person is withdrawn or not, and whether or not the prick is expected, so will the effect upon the mind of trauma vary, according to whether it is expected and dreaded, or comes as a complete surprise.

Now it is more than likely that not only the *conscious* psychical state of the person, but perhaps still more so those psychical processes which run their course with a wave of consciousness, has an important influence upon the further distribution and the ultimate action of those irritations which affect the sensory sphere. There are laws for the development of sensory irritations in different psychical fundamental conditions, but such laws are unknown for conditions within the wave of consciousness, as well as for unconscious psychical states. *We must, therefore, not be greatly surprised if in one case trauma leaves no visible signs, in spite of the fact that a marked psychical impression is made upon the injured, while in another case sensory and motor changes appear in an affected member notwithstanding the fact that the injured person has experienced neither fright, fear, nor actual pain.* It is to be hoped that the foregoing will somewhat broaden our conception of what is generally understood by "psychical genesis."

We have thus far considered the traumatic cases which, without recognizable psychical change, show symptoms resembling hysteria. In view of the preceding survey we do not maintain that in all of these cases the pathogenesis is absolutely psychical and not somatic. But I believe I have made this clear—that in those cases which apparently are not psychically affected (nevertheless interesting) there may be a psychical pathogenesis. Moreover,

if we enlarge the circle of somatic pathogenesis as we do the psychical, we must expect that these two circles will finally meet, and somatic genesis be merged in the psychical.

III. SYMPTOMATOLOGY

As the clinical picture of the affection under discussion is not uniform, but as we are dealing with a number of related neuroses, the symptomatology must be so presented that the majority of the phenomena appearing in the neuroses, as well as the most common ones, and those which deviate from type, will be considered.

General Impression and Mental Symptoms.—In so far as the general impression is apparent in the constitution, the bony structure, the muscles, and the subcutaneous fatty tissue, traumatic neuroses reveal nothing peculiar. In many cases the *facial expression* and the general posture of the injured person is so characteristic that these alone tell the tale of suffering. There is a slight, but persistent, longitudinal wrinkling of the forehead, the eyebrows are highly arched, and the angle of the mouth is drawn downward. The expression of these patients changes but little while speaking; on the contrary they look dissatisfied, anxious, careworn, and often frightened. The posture of the body and its movements correspond to the physiognomy. The head and trunk are often bent forward (except in those cases known as "spinal cord neuroses" in which there is an abnormal extension of the body); all movements of the trunk, all changes of position are sluggish, and are made as if the patient were tired, dissatisfied, or as if under constant protest.

The *mental condition* is frequently what might be expected from the appearance of the patients, all of whom feel much more ill than they actually are, and believe themselves unfit for any kind of work. As a rule, they detail no special hypochondriacal symptoms, but only hypochondriacal general states, declaring that "the entire nervous system is shattered and ruined, the whole body is useless, cure or even improvement is absolutely impossible." In consequence of this underlying hypochondriasis and the struggle for damages, most patients become irritable and embittered, and this engenders a nervous condition, feelings of anger, opposition to the physician, sometimes even thoughts of suicide. The injured talk themselves into such a state that they become utterly unreasonable and unmanageable. The physician, who has their welfare at heart, explains to them their condition and why the obtaining of heavy damages under present circumstances is impossible, tells them that light work will be beneficial, that this is recommended also for nervous patients who have not suffered from accident, etc.; the patient not only obviously but in reality fails absolutely to understand this well meant and conscientious explanation. This is largely to be attributed to the persistent psychical depression resulting from the condition, partly, however, to the influence of friends and fellow workmen, from whose ranks most of these patients are recruited, and who are incapable of understanding this objective logic.

As to *intellection*, the patients often complain of "defective memory," "incapacity for thought," etc., and often attempt to simulate mental weak-

ness; usually, however, there is merely a limited power of psychical concentration, due to the hypochondriasis, as well as to want of effort without actual loss of memory. In the serious cases which must be included with severe forms of hysteria or melancholia, there may actually be a marked psychical inhibition which gives us the impression of dementia.

That these patients manifest great loss of energy and an inherent "weakness of will" need not be specially emphasized when we remember that traumatic neuroses are really hysteria, neurasthenia, or hypochondriasis, or combinations of these affections. Their conversation often voices their mental depression. Their speech is soft, monotonous, or whining, and they become animated only when discussing the accident or the question of indemnity. In some markedly hysterical cases I have repeatedly noticed speech which closely simulated stuttering, and in exceptional instances have observed the appearance of the falsetto voice.

Constitutional Condition and the Vegetative Functions.—As already stated, persons of most varied constitution are liable to traumatic neurosis. It cannot be stated that the poorly nourished or those with weak frame are more readily attacked than the robust and well fed. Varying degrees of muscular development, all types of complexion, and differences in the general nutrition, will therefore be observed in these patients. When the disease is prolonged, the general constitution it naturally more or less affected. Certainly this is true of some forms of traumatic neurosis; combined with psychical depression it gradually produces a constitutional *invalidism*. We often observe patients who suffer so greatly from excitement and psychical unrest that they emaciate; but true cachexia is hardly ever seen. It has always seemed to me remarkable that, in contrast to the foregoing observation, many of our patients who complain of numerous corporeal and psychical ills, maintain their weight and occasionally even grow stout. Perhaps alcoholism, evident in so many of these patients, here plays a rôle.

The *appetite* is more or less capricious. The patients eat enough, and their metabolism is usually in equilibrium, but—as they often tell us—"there is no pleasure in eating." This loss of appetite is probably in part due to their inactivity and refusal or inability to work. The function of the bowels is not greatly disturbed; when there is constipation this is often due to a spastic state of the muscles of the colon.

In general remarks upon the nutrition, the appetite, and the posture of the body, I have referred only to certain groups of traumatic neuroses; but what is said of sleep will apply to nearly all cases. The majority of these patients are poor sleepers, they complain of retarded falling to sleep, as well as of its being restless and frequently interrupted. If we carefully question them we often find these complaints to be justified, although the absolute insomnia often emphasized does not exist. Yet the reports of absolute inability to sleep are, as a rule, not consciously false; for the patients do not sleep soundly, therefore while asleep are still alive to certain sensory impressions. Moreover, in health sleep is followed by a sensation of rest and rejuvenation which is absent in these cases, hence the patients feel as if they had not slept at all, but were constantly awake and "had heard every hour strike." Nocturnal rest is frequently disturbed by dreams, filled with unpleasant sensations of fear (of persecution, falling, corpses, or disputes).

Dreams in which the accident is lived over again are rare, while those of a pleasant nature have never been reported to me.

Motion.—It is not remarkable that the muscular system which directly depends upon the nervous system should manifest great disturbance. True degenerative *muscular atrophy* does not follow traumatic neuroses; but we frequently note atrophy of the extremities—the so-called inactivity and disuse atrophy—the extremities being kept immotile by the patient from pain or other cause. Insufficient muscular nutrition is revealed by *idiomuscular contraction.* This shows itself by the appearance of a small transverse prominence whenever the muscle is sharply tapped with the percussion hammer. In these patients true fibrillary muscular contractions are more rare, while muscular waves which are closely allied to fibrillary muscular contractions are somewhat common.

Among the numerous symptoms on the part of the muscular system we observe all grades of tremor. The very fine tremor seen in Graves' disease, a coarse tremor, and the physiological trembling from cold which resembles exhaustion tremor are noted; even the grotesque tremor which visibly increases with intended movements, and which is best designated as shaking tremor. The most common localization of this is as follows: Tremor of the eyelid on closure, tremor of the muscles of the face during speech, tremor of the extended hands, tremor of the legs when lifted high, and tremor of the muscles of the trunk.

Passive movement in many cases is impeded. Not that actual muscular spasms such as occasionally appear in hysteria—for instance, in pseudospastic paresis with tremor (Nonne)—are so common in these patients, but peculiar muscular contractions are readily produced in a painful extremity or in its vicinity, and these at first are perhaps voluntary, but in the course of time, if the pain continues or if the patient believes it to his advantage to make the affected extremity appear immotile, these gradually become uncontrollable and permanent. Among such contractions belong the *muscular rigidity* which follows injury to the joints, the so-called Ehret's contracture after injury to the foot, and curvature of the vertebral column and changes in the position of the pelvis accompanied by pain in the region of the sciatic nerve after injuries to the legs and back.

Here I must briefly describe a group of injuries to the back which have been referred to as "spinal cord neuroses." In these cases the back, especially the lumbar portion of the vertebral column, is persistently extended far beyond the physiologic limit, and the erector trunci muscles project at either side of the lumbar vertebral column like two contracted wooden bands.

In describing pathologic muscular tension we have so far considered merely the conditions resembling contracture. Excessive contractions may readily be recognized, but they are quite rare. The simple and more common *muscular tension* which is often detected by passive movement of the injured extremity is more difficult to diagnosticate. Here a thorough understanding of the condition, practice, and care, are necessary in order to differentiate an actual spastic, reflex, or involuntary tension from a voluntary one intentionally produced. Of course, in the conditions under discussion it is presupposed that there is no evidence of local organic change, especially of disease of the joint. But even if this be present the differential diagnosis

is sometimes extremely difficult because hysterical arthralgias may so closely resemble organic joint affections—certainly in regard to fixation and position of the member and in the pain—as to make mistakes extremely likely.

Hypotonia of the muscles, actual decrease and flaccidity of the muscular tonus, is seldom observed in these patients. The flaccidity of the muscles after injury to the joints is generally due to inactivity.

We must now consider active motility in these patients, and what has been said in regard to the difficulty in the diagnosis of muscular tension may here be repeated. Whether there is *complete* and real paralysis of an extremity—for the condition may be due to hysteria—cannot always be determined by a single examination, but after repeated investigations this should not be difficult. Whether, however, there is merely a ***diminution in the power*** of certain movements, or of an entire extremity, or—which often happens—this is simulated by the injured person, is one of the most difficult problems which the neurologist is called upon to decide. True "adynamia" or "hypodynamia" is more easily diagnosticated when it implicates the entire motor apparatus of the body than when the power of only a ***single member*** is diminished.

General adynamia, which is more conspicuously shown by the abnormally great exhaustion than by the slight production of power, accompanies hysteria as well as neurasthenia and hypochondriasis. Local hypodynamia is decidedly more common in neurasthenia and hypochondriasis than in pure hysteria. It is to some extent characteristic of general adynamia that in the upper extremities the grasp of the hand in particular appears weak while the shoulder- and elbow-joints are still powerful. Inversely, in the lower extremities the movements of the foot are quite forcible while those of the hip- and knee-joints are hypodynamic. Hypodynamic conditions in the course of the cranial nerves are not rare in these patients. They are revealed by an inability to wrinkle the forehead, to open the eyes widely, to rotate them outwardly, and to protrude the tongue. The movements of the eyes and, even more so, of the tongue are occasionally impeded by hysterical spasms.

In the same frequency with which the power of motion is decreased *coördination* is disturbed. Undoubtedly ataxic movements take place, but only rarely and in certain forms of hysteria. A symptom which is usually regarded as a sign of coördinative disturbance is that on closing the eyes and placing the feet close together the patients sway or even fall. Sometimes this swaying occurs when the feet are in juxtaposition with open eyes. This is not the true Romberg's symptom, but is a psychical phenomenon, for swaying with closed eyes and feet close together is also noted in amaurosis.

The disturbances of the *gait* in traumatic neuroses are many and various. Some of the patients when walking exhibit only such sluggishness and flaccidity as corresponds to the general motor weakness; others, and this is by far more common, simply drag the injured leg and try to spare it. The leg is then slowly abducted and extended at the knee-joint, probably at first voluntarily although often unconsciously, in the subsequent course of the disease from habit and unconsciously; these disturbances in the gait are usually observed in neurasthenics and hypochondriacs.

More manifold and interesting are hysterical abnormalities of gait. Here

the hemiplegic form, the sweeping gait (Todd) with a mere dragging of the leg which hangs as if dead, as well as hundreds of variations of the hysterical dysbasic gait, with spastic or ataxic features, or with these combined. Frequently a cane or crutch is used in walking when this is unnecessary, and this is done merely to awaken sympathy or because the patient desires to appear as ill as possible. The staggering gait is most common with malingerers, and—as is quite evident to the injured person—this resembles the gait of one under the influence of alcohol.

Just as in walking, motor sluggishness and flaccidity are often apparent in other movements of the trunk—in sitting down and standing erect, in rising from the recumbent posture, as well as in bending forward and again assuming the erect posture. Some patients groan and complain of vertigo when they perform this last movement; others, who suffer from so-called spinal cord neuroses, cannot stoop at all but let themselves down by flexing the knee while holding the back stiff.

Sensory Functions and Those of the Special Senses.—Disturbance in the function of the special senses is noted in all forms of traumatic neurosis. Usually there are subjective phenomena: specks before the eyes, blurring of the vision, photophobia, tinnitus aurium, sensitiveness to noise, a bad taste, etc. Compared with the frequency of these subjective disturbances, actual diminution of sensory function is extremely rare, and is almost peculiar to hysteria.

In the eyes this impairment of function does not appear as a decrease in the central acuity of vision but as a concentric limitation of the optical field. The concentrically limited field (a typical sign of hysteria) is usually bilateral. It betrays its psychical genesis by the fact that it remains narrowed, unchanged, and uniform, whether its projection is in the immediate vicinity of the eye or far removed from it (tubular form of the field of vision). If we carefully examine the optical field in these patients without asking them any suggestive questions, and do not depend merely upon the results of a single examination, we will conclude that the frequency of a limitation in the field of vision in traumatic neurosis is greatly exaggerated.

Much less familiar to us, and more obscure than in the eye, are the conditions found in the ear. Here, chiefly after injuries to the head, and with an absolutely negative finding by the speculum, we detect a unilateral or bilateral defect in hearing especially impairing the perception of the high tones of the tuning-fork. Whether this defect, designated by otologists as "nervous difficulty in hearing" or "labyrinthine shock," is to be regarded as purely neurotic or the pathologic conception of a neurosis must at this point be abandoned is not yet certain. True hysterical deafness or difficulty in hearing is much more rare than "*commotio labyrinthi.*" Although it occasionally disappears suddenly or passes to the other side which has thus far been normal—it is, as a rule, extremely tenacious, and the prognosis is grave. The diagnosis of a hysterical defect in hearing is extremely perplexing; but, in addition to sudden changes in the power of hearing, it is facilitated by the fact that there may be other disturbances of sensation in the parts surrounding the ear or in the ear itself.

A decrease in the *senses of smell and taste* (a common complaint of these accident patients is that "everything tastes alike") can neither be confirmed

nor absolutely disproved since we are unable to test these senses. In hysteria there may be complete anosmia or ageusia.

Unilateral decrease or even abolition of all the special senses is a well-known hysterical stigma, and is usually combined with a loss or decrease of the tactile and pain senses on one side of the body. Occasionally the *sensation of the deeper parts* is involved in this hemianesthesia, so that the patient is unconscious of a passive change in the position of his members, and even the stereognostic sense is lost.

Among the sensory disturbances of a positive hysterical nature which are observed in these patients, *general anesthesia* affecting all of the sensory qualities of the skin must be mentioned; this is occasionally accompanied by a decrease of the pain sense in the muscles, so that muscular contractions produced by a powerful faradic current are not painful. Moreover, we not infrequently observe a decrease and loss of the pain sense with an unimpaired contact sense, therefore a dissociated sensory disturbance; the temperature sense is rarely disturbed. Partial sensory disturbance limited to a certain area of the body, an extremity or a portion of one, is more common than disturbance extending throughout the body. On investigating sensation in a member that has been injured, the patients feel, as a rule, the prick of a needle as dull or blunt, or perhaps not at all. We would greatly err in regard to these sensory disturbances if we should at once consider them as hysterical. Quite a large proportion of these patients undoubtedly and consciously make false reports; in others these are due to autosuggestion. The injured person cannot believe that in a member which is so painful and so useless, one function, that of cutaneous sensation, remains unimpaired. If he is simulating, and we express a suspicion or utter a warning, the apparent sensory disturbance soon disappears while true hysterical sensory disturbance will persist. In hysterical monoplegia localized anesthesia is most apparent. Hysterical anesthesias are not always—as is stated—sharply demarcated from their surroundings, but they often gradually merge with areas in which sensation is normal. I do not doubt that some of the sensory disturbances noted in our patients are due to frequent, consequently somewhat careless, examinations, and are thus cultivated. Therefore too great value should not be attached to them, and it is unnecessary to resort to all kinds of expedients in order to learn whether or not there is a sensory disturbance. The diagnosis may often be made without including the sensory conditions, and in deciding whether or not the patient is able to work existing anesthesia plays but an insignificant rôle.

Much more essential in regard to capacity for work is *hyperesthesia*. This may appear at the point of trauma, also in other portions of the body which are not injured, in the head, the back, etc. The preceding statements in regard to the reality of anesthesia are specially applicable to hyperesthesia. The mental condition of traumatic neurotics is usually such that the patients complain of great pain on pressure at the point at which the trauma occurred, and even of sensitiveness on superficial contact. In the injured extremities passive motion of the joints, sometimes even the mere contact of the limbs, is said to cause pain. Whether this hyperesthesia is simulated or not can be decided only by *successive* investigations. If by diverting the patient's attention we can repeatedly make firm pressure upon the area said

to be hyperesthetic without his manifesting pain, while on direct questioning he reports extreme pain, it is usually a case of malingering. If, in a single examination, there is a contradiction between the condition with deflected attention and that upon direct questioning we must not at once assume simulation.

Hyperesthesia of the head and back also follows injury of these areas, but may occur even when these parts are uninjured; if in the head, it is particularly noted over the forehead and the crown; if in the back, it is usually in the vertebral column. The lower portions of the vertebral column are generally most sensitive. Spinal hyperesthesia is peculiar in that the areas most sensitive to pressure or to percussion vary. If patients with this symptom are examined several times in succession, and at each examination we mark the vertebra most sensitive to pressure, we may find that a different vertebra is each time designated as the most painful one. This variation in the report by no means indicates malingering. I have often seen the same condition in patients with neurasthenia not due to injury. Other hyperesthetic areas are the following: the *arch of the ribs*, the *hypogastrium* (ovarian pressure points), and the superficially situated nerve trunks, the last being most marked in women. We must be cautious in assuming a special sensitiveness of the nerve trunks to pressure. Many a reported pressure point would be omitted from the history of the patient if the investigator had by repeated examinations informed himself concerning sensitiveness to pressure in the areas surrounding the so-called pressure point. For there are numerous cases in which pressure at any point of the body (in so far as pressure of the soft parts against the periosteum and the bone is possible) is asserted to be painful.

Spontaneous hyperesthesia and *paresthesia* are more common than hyperesthesia to contact and pressure. Headache, a sense of pressure or drawing pains in the head, stitches in the head, reeling sensations, vertigo either persistent or paroxysmal, and both upon directing the glance upward and in bending forward, formication at the top of the head, a sensation of heat in the head, a feeling as though the brain "would burst," or as if it moved in the head, or as though the head would burst, are a few of the common complaints. Among thousands of paresthetic sensations in the extremities only a few will be mentioned: a general feeling of paralysis, "the legs feel as if dead," heaviness of the members, formication in the hands and feet, a sensation as if the tendons were too short or that "everything is tense," as if the bones would break, etc. In the trunk we note pains in the back, in the lumbar region, between the shoulder-blades, in the side, pressure in the cardiac region, and feelings of constriction. The so-called *globus hystericus* is exceedingly rare among the hyperesthesias of these patients.

Reflexes.—The most important of the tendon reflexes, the *patella tendon reflex,* is always increased in traumatic neuroses, whether they are due to hysteria, hypochondriasis, or neurasthenia. In the cases of most decided increase, a single light tap over the quadriceps tendon will repeatedly cause the extension of both legs in rapid succession. This extension is often accompanied by a general contraction of the body, which is occasionally produced when the quadriceps tendon itself is not touched but a neighboring point—for instance, the border of the tibia. The *Achilles tendon reflex* is not so invariably increased as the patella reflex.

True *patella clonus* and *foot clonus* occur in the hysterical forms. A condition resembling true clonus, in which the clonic movements cease with the cessation of the psychical stimulation of the patient or as soon as his attention is diverted from the clonus, will often be observed in the neurasthenic and hypochondriac forms. True clonus which accompanies organic diseases is very persistent. The pseudo-clonus just described is an inconstant sign.

The *triceps reflex*, the *jaw reflex*, and other *tendon reflexes* play no rôle in the symptomatology. But attention must be given to the cutaneous and mucous membrane reflexes.

It is well to bear in mind that the action of motions of resistance which are closely related to those *cutaneous reflexes* following painful cutaneous irritation do not always agree with the patient's reports of his cutaneous sensation. But it does not absolutely prove simulation on the part of the injured if a member which has been reported as analgesic is withdrawn on being unexpectedly pricked with a pin. The apparent absence of a cutaneous reflex (for example, the abdominal reflex) with normal sensation usually depends upon an improper position of the patient and consequent muscular tension. In connection with cutaneous reflexes I must devote a few words to the so-called *goose-flesh reflex*. After stroking the skin of the chest or back with the handle of a percussion hammer we not infrequently perceive that the papillæ of the skin in the irritated area come into prominence from reflex action of the *erectores pilorum*.

Of the *mucous membrane reflexes* only those innervated by the cranial nerves need be considered. In rare cases the conjunctival reflex is absent when there is hysterical anesthesia, as well as the reflex of the nasal mucous membrane, and those which originate from the posterior pharyngeal wall and the region of the soft palate. The last two reflexes may be absent without simultaneous sensory disturbance, and in some of the cases this depends upon chronic irritation of the mucous membrane such as is observed in smokers or alcoholics. Of course, the absence of the retching reflex is not the rule in chronic alcoholism; but, on the contrary, a decided increase of the reflexes originating in the pharyngeal mucous membrane.

The internal reflexes, the *pupillary light reflex*, the reflexes for the evacuation of urine and feces, and the sexual reflexes, are generally undisturbed in these patients, although even here some deviations and peculiarities are noted. In hysteria and neurasthenia a rapid and intense pupillary reaction to light will sometimes appear, and the so-called *hippus* of the pupil is often observed. This hippus is shown by the fact that, without any variation in the brightness of the light, changes in the width of the pupil take place in rapid succession.

Rigidity to light does not appear in traumatic neuroses, but the reaction is sometimes sluggish and retarded. Strictly speaking, sluggish pupillary reaction to light is sufficient to relegate those cases in which it occurs to a realm beyond these neuroses. The cases that I have in mind were usually those with severe injuries to the head, with profound psychical depression and general mental dulness.

Among disturbances of the *bladder* and *rectum* I must mention inability to void urine when the attention is directed to it, also spastic constipation

due to similar processes in the smooth muscles of the intestine; these conditions are found in all traumatic neuroses. Abnormally frequent micturition, pollakiuria, is also common. Very rare but of undoubted occurrence is the occasional involuntary discharge of urine. This is sometimes found in hysterical astasia abasia. The *sexual reflexes* of erection and ejaculation also show severe disturbance. In certain cases the power of erection absolutely ceases.

Vasomotor and Trophic Disturbances.—Derangement of the circulation is very common. Conspicuous reddening or pallor of the face, livid discoloration with coolness of the hands, more rarely of the feet, bluish discoloration of the knees, and extreme redness of the face on stooping are often observed. A number of vasomotor disturbances are not confined to a definite portion of the body but are found over its entire surface. Among these is a peculiar bluish red *mottling* of the skin as well as irregular map-like red *flakes* upon the chest and back. Some very interesting vasomotor phenomena may be evoked. If the physician makes firm pressure upon the skin with the whole hand—for example, on the chest of the patient—after removing the hand a white impression of it with redness of the surrounding areas will be seen for a few seconds. Furthermore, if we stroke the skin firmly with a hard, somewhat pointed object—for example, the tip of the percussion hammer—a red line appears in these areas, which often swells for a few minutes and becomes elevated, then turns pale, and remains so for some time (*dermographia*). Instead of a sharply demarcated line, in some cases irregular red flakes appear in the parts that have been stroked.

A phenomenon which may be noticed during the test of sensation is the *appearance* of small *nodules* in the areas subjected to the needle; but these also are a form of dermographia. Colorless or pale *edema* is noted as simple stasis edema, occasionally in organic as well as in functional paralysis of the extremities.

Besides this stasis edema we must mention so-called *œdème bleu,* which accompanies hysterical paralyses, and the so-called "*flying edema.*" The latter appears to be closely related to hysterical conditions. It reveals itself by edematous swelling which is most marked in the face, which appears suddenly around the eyes, remains for hours, and then disappears without leaving any disturbance.

The *hair* often shows trophic disturbance. The hair and beard suddenly or very rapidly turn gray; this may affect strands or uniformly implicate the entire hairy scalp. Besides grayness of the hair we must consider *alopecia* which follows the distribution of the fifth nerve, while the region of the occipital nerve is usually exempt.

Frequently there is a derangement of the *secretion of sweat*; the secretion is usually profuse. In spite of the fact that the patients do not feel warm but are often even cold, there is copious perspiration in the hands and axilla. The opposite condition—an abnormally slight secretion of sweat and consequently unnatural dryness of the skin—is much more rare.

The structures beneath the skin undergo little, if any, atrophy. *Muscular atrophy* is not peculiar to the nervous pathologic process as such; on the contrary atrophy may be of the so-called inactivity or reflex form (in arthritic diseases). Consequently it may be found in entire extremities, or in portions

which are the seat of pain, in joints which are stiff or which for any other reason are but little used.

Under the trophic and vasomotor disturbances I must again call attention to certain difficulties from which the patients suffer. Congestion of the head, vertigo, headache which appears and disappears suddenly, therefore symptoms which are common in these cases, cannot always be regarded as purely functional, but are in all probability often the expression of organic vasomotor disturbance in the interior of the skull. Friedmann, who has closely studied these disorders which do not strictly belong among the neuroses, found them to be accompanied by marked dilatation of the cerebral vessels, extravasation into the surroundings of the smaller vessels, hyaline degeneration of the vascular walls, and similar changes. If such symptoms appear after severe injuries to the head the changes in the cerebral vessels which Friedmann observed should be borne in mind.

Spasmodic Conditions.—Although spasm is not an invariable symptom yet it is by no means rare. As a rule, local or general spasm justifies us in regarding the case as hysteria. *Local spasms* are never accompanied by loss of consciousness. They may be tonic, clonic, or even of a mixed nature, and usually implicate the limb or side of the body which is affected by trauma. Local hysterical spasm is more common, and may occur many times during the day. It is caused by certain external or psychical irritations, and in many cases may be evoked voluntarily by the patients. Moreover they often possess the power to suppress such an attack at its onset, but for obvious reasons make no use of it. General convulsions—such as those of hysteria—are manifold. Consciousness is little or but slightly disturbed, or affected by hallucinations or illusions. Complete unconsciousness like that of epilepsy is never seen in the spasms of traumatic neurosis, and if during the attack the patient does not respond to calling, and seems insensible to all about him, nevertheless we may easily prove that sensory irritations are both experienced and appreciated. If we suddenly grasp the patient who is convulsed, or press the fist deeply into the hypogastric region, dash cold water into the face, or the like, the character of the spasm as well as its intensity will change, which sufficiently demonstrates that the patient was conscious of the irritation.

The character of the contractions varies as greatly as do the voluntary movements; for in its nature a hysterical spasm resembles voluntary movement. Entire movement complexes are executed, and only very rarely do we see simple flexion and extension of the member or similar motions which are in the lowest plane of coördination. The movements most often observed in these spasmodic conditions are a tight closure of the eyelids, drumming movements with the fore-arm, oscillations of the head, rotation of the body upon its longitudinal axis, pulling off the clothes, and tearing out of hair. Of tonic movements the well-known *arc en cercle* or its more common abortive form, mere extension of the back, is common. No mention need be made of the other characteristics of hysterical spasm, except that there is sometimes a sluggish reaction to light, more rarely even the absence of pupillary reaction to light. As a rule the spasm is preceded by certain prodromes: severe pain in a certain area, a sensation of fear, spasmodic crying and the like. After the spasm has ceased the patient often feels

perfectly well; sometimes, however, there is exhaustion which lasts several hours.

In addition to pure hysterical spasms there may be other spasmodic attacks which present some of the characteristics of epilepsy, but these cases are usually *alcoholic.* Here the picture of the neurosis is not pure, but comprises also organic symptoms. Besides attacks of spasm there is "vertigo" which plays a great rôle in the complaints of the injured. This usually embraces a variety of subjective phenomena which the patient groups under the name of vertigo. But there may be actual objective attacks of vertigo, in which the patients suddenly experience a sensation of dizziness, lose their equilibrium, and sway. If these occur during rest and without external cause, they are usually due to severe injuries of the head combined with circulatory disturbances of the kind described by Friedmann, such as arteriosclerotic changes, or there are organic elements in the pathologic picture. If by stooping or rising suddenly or by passive movements of the head it is possible to produce such a variation in the intracranial circulation as to cause swaying or similar objective signs of vertigo, organic complications are not necessarily present, but we may have a purely pathologic picture unaltered by organic changes.

Symptoms on the Part of the Internal Organs.—In many cases there may be symptoms attributable to the internal organs, although usually of merely functional nature.

First, the *heart* must be mentioned. Persistent increase of cardiac activity and abnormal irritability are not rare. While the normal pulse after slight exertion is accelerated only a few beats per minute, and then returns to its original pace, in these patients after slight exertion the pulse is often greatly accelerated (20 to 30 beats per minute), and this rapid action persists for several minutes; or we observe that even during rest the pulse may become accelerated, and be much more rapid than on bodily motion. Simultaneously the apex beat is often heaving without a positive hypertrophy of the left ventricle being discernible. In some cases there is a conspicuous acceleration of the heart beat if we make pressure upon any existing pain points (*Mannkopf-Rumpf's sign*). This, however, is so inconstant that we cannot regard its absence as a reliable indication—as some authors do—of malingering.

Irregularity or intermittency of the pulse, signs which appear without organic disease of the heart and which are not rare in neurasthenia, may also be noted in neuroses of traumatic etiology, although somewhat less often.

A common and important symptom on the part of the circulatory apparatus is *arteriosclerosis,* which is of organic nature and appears to be genetically connected with cardiac activity. The occurrence of arteriosclerosis in traumatic neuroses without simultaneous and chronic alcoholism, syphilis, or any of the other well-known vascular poisons, was at first doubted, but must now be regarded as a fact. I believe the genesis of arteriosclerosis and its connection with neuroses must be explained by the fact that the principal damage to the vascular wall consists in the extremely frequent and intense variation of its vascular lumen. This abnormal distention of the arteries may be primary and due to a damage of the general vasomotor center, or it may be secondary and attributable to a pathological increase of emotional conditions of various kinds or to similar psychical processes. Both modes

of development are possible in traumatic neuroses. It is obvious that the normal physiologic stimulation of the interior of the vessels by the circulating blood must be changed by the abnormal variation in the caliber of the arteries. The reaction of the vascular wall to this peculiar internal irritation is expressed by a loss of elasticity in the vessel followed by a thickening of its walls, and finally by a calcareous deposit; briefly, the phenomena of beginning and complete arteriosclerosis. As already indicated, arteriosclerosis is an exceedingly important, and the most frequent, organic accompaniment of traumatic neuroses.

The respiratory organs, especially the *lungs*, less often show changes. Sometimes the *respiratory type* is altered, an acceleration being observed which may be quite excessive. It may be paroxysmal or persistent. Of course, changes in the lungs and respiratory passages are not the cause of the increased respiration in traumatic neurosis. Hysterical *cough* is as rare as hysterical *tachypnea*; it is usually distinguished by its peculiar barking character, and here another symptom may be mentioned—hysterical *hemoptysis*. In most cases this is due to hemorrhage from the mucous membrane of the mouth, the gums, or the pharynx, produced by the hysterical mental state of the patient, or purely from malingering. In isolated cases there are said to be spontaneous hysterical hemorrhages from the respiratory organs.

Of the abdominal organs only the *kidneys* show disturbance in function. Here must be mentioned the excretion of enormous amounts of urine and *alimentary glycosuria*—i. e., a temporary excretion of sugar after the ingestion of large quantities of carbohydrates.

IV. DIAGNOSIS; SIMULATION

In the diagnosis of traumatic neurosis we must decide three questions: 1. Are we dealing with a neurosis? 2. Is the neurosis traumatic—i. e., can it be traced to an accident? 3. What form of traumatic neurosis is present?

The first question—whether a neurosis is present—is generally more easily answered than the other two. It is true only the most important neuroses form such characteristic pictures that non-recognition is impossible and that the diagnosis can be made at sight. Difficulties arise only in the less fully developed forms, the so-called *formes frustes* (undeveloped forms) of general neuroses, and in certain local mono-symptomatic neuroses, such as functional monoplegia and diplegia, paraplegia of the legs, etc.

In the differential diagnosis of pathologic conditions manifested by general complaints and general phenomena, we must first consider the following organic diseases: Progressive paralysis, obscure focal diseases of the brain, multiple sclerosis, general arteriosclerosis, certain chronic intoxications, such as saturnism and alcoholism, also a series of systemic diseases such as chronic disease of the kidney, diabetes, etc.

A diagnostic danger pointed out by many authors is that *indefinite* pathologic conditions of nervous nature are liable to be regarded as "traumatic neuroses" simply because of their appearance after trauma; here only the most careful investigation will preserve us from error. It must be borne in mind that the presence of one single symptom due to organic cause vitiates the diagnosis of neurosis. If we observe sluggish pupils, rigidity of the

pupils, pallor of the optic disc, absence of the patella reflex, degenerative muscular atrophy, a lead line, sugar or albumin in the urine, extreme arteriosclerosis, or similar conditions, the most obvious inference is that no neurosis is present. Of course traumatic neuroses are somewhat more apt to be accompanied by organic disease than the non-traumatic—as I tried to explain when discussing the symptomatology—and only when the complicated organic symptoms are subordinate to the entire pathologic picture may we conclude that a neurosis is present. This is the case when there is slight arteriosclerosis after injury in a person who shows chiefly the general objective and subjective symptoms of the neurosis or similar conditions.

If the organic symptom present is pupillary rigidity or something of like importance, we must have other and cogent reasons for believing a neurosis to be present before we can make such diagnosis, or can believe that symptoms of two different natures, one of a neurosis and one of organic disease, coexist. In practice this generally occurs when the organic sequels of chronic alcoholism (disturbance of pupillary reaction or optic atrophy) or of an old syphilitic infection are joined to neurotic symptoms.

Among the organic diseases of the nervous system which must be considered in the differential diagnosis are a few which, especially at the onset of the affection, may completely simulate a neurosis. Chief among these are progressive paralysis and brain tumor. A case of the latter proved most instructive to me.

The patient was a man with a severe injury of the head. He presented marked symptoms of a neurosis, but under any psychical influence there was such a conspicuous variation in his condition and complaints (uncertainty in gait) that we seriously considered a diagnosis of hysteria, and thought certain symptoms to be feigned. While under observation, however, objective signs rapidly appeared which permitted the diagnosis of brain tumor, and autopsy revealed a tumor in the anterior portion of the brain.

In a pathologic condition like that just described the decision whether the affection is functional or organic may not always at first be possible; nevertheless, after an examination, especially after several such or after clinical observation, it can be made with great certainty. But there are conditions in which such a diagnosis, or even a differential diagnosis, cannot be made; I refer to the cases in which, following severe injuries to the head, there is mental depression, inhibition, general psychical dulness, vertigo, headache, general adynamia, etc. This symptom-complex, as Friedmann demonstrated, is often due to the multiple but slight organic changes in the cerebral vascular apparatus and its surroundings. It is no doubt true that Friedmann's findings are observed in only a few of the cases with this clinical picture, and that others with exactly the same clinical phenomena are purely functional. But Friedmann's investigations and similar ones make it appear likely that in the future many other symptom-pictures following trauma, which have so far been regarded as functional, will be proven to be due to an underlying organic condition.

Assuming that examination has revealed no *organic* change in our patient, even this does not certainly prove a neurosis, and our object has been only partially attained. For in the preceding consideration we always assumed the presence of distinctly *objective* pathologic symptoms. If the

injured person presents no noteworthy symptoms but, on the other hand, there are many subjective phenomena and complaints, purely in theory the existence of a neurosis becomes likely. In practice, as a rule, *another factor* is added which doubles or trebles the diagnostic difficulties: *simulation.* Here I believe it wise to discontinue our discussion of the diagnosis, and briefly consider simulation.

The term *simulation* should be employed only when the person examined consciously and purposely attempts to deceive us, representing pathologic signs to be present which are not so, or when he wilfully exaggerates existing conditions. *Therefore the attempt to deceive is an integral constituent of our conception of simulation.* This is decidedly modified when we consider what physicians commonly call feigning. Primarily we must differentiate those cases in which a pathologic state of mind or of sensation leads to false reports, and among these we include most of the prominent forms of hypochondriasis and many cases of hysteria. This by no means implies that hysterics or hypochondriacs do not occasionally and consciously make false reports, and may therefore be malingerers.

Secondly, we cannot group among malingerers those injured persons who, while not hypochondriacs or hysterics, actually do misrepresent facts, but who are not sufficiently intelligent to appreciate that what they state (usually by the advice of others) in order to obtain damages is not the truth. Such people are often imbeciles from birth, and not possessed of even the most rudimentary education.

Between the latter and the hysterical is a third group of patients who are difficult to handle. These are patients who actually have sustained certain injuries. For years past they have made innumerable reports, verbal as well as written, of their condition. To appear consistent, ailments which have gradually passed away or become secondary are continually included. They have observed that judges and physicians believed their reports. Without any special predisposition to nervous affections—except a congenital want of discernment—and from constantly brooding over the injuries in question a mental condition has gradually developed which impairs the injured one's faculty of criticism, nor does he desire to use this faculty. Such torpid mental appreciation, which for any psychological reason may gradually and completely be removed from criticism, which has become a "matter of faith"—this latter, however, being more or less true of all persons—by no means implies an aberration from the normal mental state. This approximates what we moderns have called "autosuggestion," and has become the more firmly rooted the more sensibility and emotion have aided in its development.

The chief difficulty in the clinical discrimination of these cases is on the one hand their similarity to hypochondriasis and a paranoic condition, and on the other hand to malingering. This perplexity is enhanced by the fact that the same case is at various times judged differently. What may to-day be regarded as simulation (according to the definition just given) may a year from now appear otherwise, and, just as in a previous examination, may be unconsciously simulated, therefore no longer feigned. No general rules can be formulated, but every case must be individually investigated and judged.

The perfect *simulation* of an entire *symptom-complex,* none of the symptoms actually existing, is so exceedingly rare as to be seen but a few times among several hundred cases. For obvious reasons *a few symptoms* are often simulated, and these are interwoven with difficulties actually existing, therefore etched into a real pathologic condition. Prominent among these is an apparent *diminution* or absence of *sensory functions* and those of the *special senses.* Hemianesthesia which actually exists in no small proportion of these cases is known to many patients (perhaps owing to the indiscretion of the physicians during their frequent examinations, their accompanying questions not being always cautiously framed) as a sign of disease, hence is often simulated. Among motor symptoms those *adynamias* and pareses are simulated which are generally limited to an extremity or a portion of an extremity. *Arthritic* and *muscular rigidity* are often feigned.

In discussing *Romberg's sign* I called attention to the fact that swaying on closure of the eyes is a favorite method by which the accident patient attempts to prove the severity of his disease, but the movements of *stooping and subsequent rising* also furnish an opportunity for exaggeration, the injured either declaring themselves absolutely unable to stoop or that on doing so they sway or even fall.

In eliciting the *patella tendon reflexes* we occasionally note that the patients try to increase the jerk by their own efforts, or they produce all sorts of muscular contractions such as drawing up or drawing away the legs and the like, in order to show the active reflex effect upon the nervous system. In the course of objective investigation, attempted simulation in regard to *sleep, appetite,* and other *subjective difficulties* is much more common.

After having enumerated the principal factors which incite the injured to feign, I must state that the recognition of simulation is one of the most difficult parts of the investigation of these patients. For, since actual simulation as well as hysterical and hypochondriacal exaggeration originates psychically from the pathologic nature of the disease, and is therefore a psychically congenital phenomenon, it is not remarkable that simulation and pathologic exaggeration are often closely akin. Only a most experienced physician who understands the nature and the phenomena of the great neuroses will attempt to diagnosticate simulation from any of the aforesaid symptoms, and cases occur which even baffle an expert, as has been repeatedly emphasized by Strümpell and other authors.

The *demonstration of simulation* is usually of greater practical than scientific import; and in practice one law must chiefly be our guide. We must always prove that the *actual difficulty,* the definite symptom-complex, or the individual symptom which has always been paramount, is simulated. Therefore evidence that the patient has a *general tendency,* even in other matters, to simulate does not warrant us in declaring that a suspicious *individual* symptom is feigned. For example, if a patient has paralysis of the arm which is difficult to explain, and we find upon investigating sensation or testing his visual field that he makes incorrect reports (therefore shows a tendency to feign) this is not sufficient to prove that the paralysis of the arm is also feigned. The fact that the patient at every investigation tries to deceive us will naturally strengthen our suspicions that the paralysis of the arm is simulated, but this presumption becomes certainty only when by some method

of examination—or even without this—it is proven that the patient can *voluntarily* move the arm which he exhibits as paralyzed.

The foregoing postulate of a *special* proof of simulation determined in each case is based upon the experience that accident patients, who are usually of little education, attempt to make an actual infirmity, pain or the like, appear more plausible to the physician; perhaps because they have previously been met with suspicion. For this reason they are anxious to show that other portions of the body are affected as well as that which was injured, and for this they invent symptoms. Although these patients are really at fault, yet if the investigator condemns this tendency to simulation as malingering, his judgment of the general pathologic picture is neither scientific nor just. Aside from this reason there is another; we should be very cautious in attributing a symptom to simulation, for among the methods which are calculated to discredit the symptoms of the patient there are many which, as I have already intimated, in the hands of one not an expert may lead to most erroneous conclusions. I have called attention to the similarity of conditions in malingerers and in hysterics. This similarity is markedly apparent when we test the accuracy of the patient's statements by a method based upon spontaneous sensory impressions or those of the special senses. For this reason I advise caution in the use of a number of tests advised in text-books and employed to unmask malingerers. If the patient answers affirmatively certain absurd and suggestive questions as to whether "he sees double early in the morning," whether "both thumbs become numb," or similar ones advised in literature, the significance of these answers is purely problematical. The same care must be exercised in putting our questions in regard to pain upon pressure and the like, and even in the opposite direction skepticism should not carry us too far. Sometimes the physician makes pressure over a cicatrix upon the head, and the patient reports pain in the area touched, but does not state that the surroundings are also sensitive; shortly afterward the physician tells him that the cicatrix is in another area (very often the patient is unable minutely to localize the cicatrix upon the top of his head if he is prevented from touching it with his finger) and the patient now reports tenderness at a second place, the simulated cicatrix, while the actual cicatrix is no longer painful. In the great majority of cases this is regarded as conscious simulation. But it may be that, even here, we are dealing with a markedly hysterical person, and that the suggestion was conveyed by the physician.

I must here call attention to another localization of pressure pain in discriminating which I believe errors are often made. *Pain in the spine*, which is frequently produced in these patients by pressure on or tapping the spinous processes of the vertebræ, sometimes varies so much in the course of a single examination that nearly all of the spinous processes may be declared to be the ones most sensitive to pressure. Those not familiar with this condition may readily suppose the patient to be feigning. But in patients with functional nervous diseases, those who have not suffered from an accident, and who therefore have usually no incentive for simulating this condition, we often note the same phenomenon.

What has been stated sufficiently indicates the difficulty of proving malingering. Hence the question arises: How can we prove that a definite

symptom or complaint is simulated? And here it is much easier to state how we should *not* proceed than how we should.

Only general rules can be given; above all, it is desirable that the method employed to determine simulation should be most simple. The more complicated it becomes and the greater the time it consumes the more it taxes the intelligence and good-will of the patient, and the more practice and familiarity with the condition it necessitates on the part of the investigator. I strongly object to the employment of complicated apparatus. If the patients have the slightest degree of intelligence, in most of them such apparatus is calculated to produce immediately a certain psychical irritation which proves a source of error. If the examination makes simulation appear likely, it is wise to make this diagnosis positive by giving the person an opportunity to make an "honorable" and apparently non-compromising withdrawal, the suspicious symptom for one reason or another seeming to disappear. Under long observation the patient sometimes realizes that he cannot keep up his rôle for any length of time, and is anxious to avail himself of the chance offered. It is inadvisable to use powerful agents, such as strong faradic currents, to prove malingering. Such methods are cruel, and the results attained are not always uniform.

After this somewhat lengthy but necessary digression and the description of simulation, we return to our proper theme—the diagnosis. Before discussing simulation, we had assumed that the diagnosis had reached that point at which an organic affection could be excluded. The description of simulation became necessary for, after making a diagnosis, and after excluding *organic disease,* the affection is proven to be *functional.* Now we must decide two questions—whether the neurosis is traumatic, and what form of traumatic neurosis it presents. In answering the first, we must distinguish between the scientific and the practical proof that the disease is of traumatic genesis. Scientifically its accidental origin may often be proven from the fact that we knew the patient prior to the accident. Those forms are exceptions which bear the stamp of traumatic genesis, because of either a typical ensemble of somatic and psychical disturbances, or the presence of hysterical paralysis or something similar at the seat of trauma. In practice the proof of a traumatic origin of the disease is easily demonstrated. As a rule it may be considered certain if no other etiology for the neurosis than that of trauma is apparent, and if the trauma from its nature and severity seems calculated to produce a nervous affection. Practically it suffices if we can prove the trauma to have been an essential or partial cause of the disease, or that the affection was aggravated by the accident.

The last point to be decided in making the diagnosis is the *accurate determination* of the nature of the traumatic neurosis present. It has already been stated that by the designation "traumatic neuroses" we mean cases only of hysteria, hypochondriasis, neurasthenia, or combinations of these affections; but not those other neuroses which occasionally develop after trauma, such as chorea, epilepsy, etc. Therefore the decision as to the nature of the traumatic neurosis will chiefly depend upon a differential diagnosis between these three great psycho-neuroses.

The *differential diagnosis* is facilitated when there are conspicuous symptoms which point to the fundamental hysterical character of the neurosis;

for instance, monoparesis of an extremity, astasia abasia, sensory hemianesthesia, contractures in an extremity, or in the muscles of the tongue or face. These signs, or similar ones, generally justify us in regarding the functional symptom-complex as of hysteric nature. If there are no conspicuous, somatic, hysterical symptoms, but psychical ones are most prominent, if there is anxiety, metaphysical mania, or mental depression, the neurosis is usually hypochondriasis. When the psychical depression is less marked, or when it is only slight while adynamia and abnormal exhaustion are extreme, the pathologic condition must be regarded as *neurasthenia*. Sensory disturbances contraindicate the hypochondriacal or neurasthenic character of the affection. Certain somatic symptoms, such as increase of the tendon reflexes, dermographia, irritability of the heart and acceleration of the pulse, tremor of the tongue, hands, and eyelids, swaying upon closing the eyes, are found in all of these three neuroses and, as a rule, do not aid us in the differential diagnosis.

The majority of the somatic symptoms enumerated as common to the three neuroses lead us to the conclusion that the differential diagnosis may often be exceedingly difficult, and in many cases it really is impossible to reconcile the pathologic picture as a whole with any of the forms of pure neurosis. This difficulty is enhanced by the fact that the psychical conditions appertaining to these three neuroses have many features in common: the tendency to excitement and irritability, fear, unrest, and distressing dreams. To these psychical conditions which belong to the picture of hysteria, neurasthenia, and hypochondriasis of non-traumatic genesis are finally added those psychical peculiarities which may be attributed to the claim for damages. Since a suit for damages represents a mighty psychologic influence which often preponderates over the genuine psychical effects of the neurosis, we can easily understand why most cases of traumatic neurosis wear a similar psychical mask, and why the peculiar and original physiognomy of the neurosis can be recognized only by close study of the case. As, however, the marked psychical influence of the desire to "obtain damages" finally reacts upon the somatic signs of the disease, it does not appear strange that in the differential diagnosis of neuroses of *traumatic* origin we do not advance very far before we perceive that we are dealing with a combination of three great neuroses. Nevertheless, even here we must act according to the fundamental law, "*nominatio fit a potiori,*" and designate the disease according to the symptoms which dominate the clinical picture.

V. PROGNOSIS AND COURSE

The prognosis of traumatic neurosis in regard to life, provided there are no special complications or sequels, is absolutely favorable. Among complications I include general invalidism and suicide. In rare cases, especially where there is profound psychical depression, but also in cases of Friedmann's vasomotor type, we note a gradual loss of strength which so lessens the power of resistance of the organism that even mild intercurrent diseases may cause death. Nevertheless, such cases are exceptional.

Somewhat more frequently severe traumatic hypochondriasis, hysteria, and the mixed forms terminate by self-destruction. This is most often the

case when chronic alcoholism plays a rôle. Despair caused by inability to support a family, anger over presumable or actual injustice, and similar emotions, are usually the cause of suicide.

The prognosis as regards cure is much more unfavorable than in regard to life. Here it may be laid down as a law that if there are claims for damages, no matter of what nature, the prognosis is decidedly more grave. This law needs no elaboration after what has been stated of the psychical genesis of traumatic neurosis. For it is to be hoped that the connection between corporeal and psychical phenomena has been so clearly outlined that what I have said may not be construed as meaning that the patient who makes a claim for damages purposely, and as a rule, prolongs the period of convalescence. Cases of deliberate prolongation of convalescence are certainly rare. On the other hand, the dominating thought of obtaining material damages for the injury, at the time, but only for the time, is such a dominating psychical factor that, without straining conscience, it stimulates the development and continuance of a hypochondriacal mental state quite distinct from the injurious psychical effect due to the excitement of court investigation of the claims.

But even if no such claims are made, the prognosis is by no means good. Psychical and somatic factors may prolong the purely functional affection for many years, even when the patient is most anxious to recover. The individual traumatic neuroses show distinct differences in *prognosis*; it is best in the mild or moderately severe cases of neurasthenic type following simple peripheral injuries or slight head trauma and in those with not too severe, purely hysterical symptoms. When pain is persistent—as, for example, with rigidity of the back and hysterical symptoms—the prognosis is more serious. In those hysterical cases combined with cardiac and respiratory symptoms or with conspicuous tremor, there is little tendency to improvement. In opposition to the foregoing statement, the prognosis is often somewhat better in hysteria accompanied by spasmodic attacks. Those cases in which the neurosis is based upon imbecility are exceedingly unfavorable, as are also all true traumatic cases of hypochondriasis. The gradual transition of the latter into paranoia is not rare, and this renders the prognosis absolutely hopeless.

The *duration* of the disease is never to be reckoned by days or weeks, but usually by months, and sometimes by years. As a rule, the pathologic picture of cases with a bad prognosis remains not only qualitatively but quantitatively unchanged. Remissions are rare; on the contrary, transitory exacerbations are common, usually occurring after excitement or the like. Intercurrent disease may temporarily make the symptoms of traumatic neurosis secondary. After this intercurrent affection has run its course, the nervous phenomena appear unchanged, prominent, and sometimes even increased by the addition of symptoms which are nervous "transitional" forms of the sequelæ of the intercurrent disease.

The prognosis of traumatic neurosis is generally the same in men and in women; children occupy a special position.[1] The prognosis in children is

[1] Compare Paul Schuster and Kurt Mendel: "Traumatische Nervenkrankheiten bei Kindern." *Monatsschrift für Unfallheilkunde,* 1899, Nr. 6.

usually not worse, as might be supposed, but better than in adults; while the effect upon the course of the disease or filing claims for damages is more conspicuous in children than in adults.

VI. PROPHYLAXIS AND TREATMENT

There are few affections the treatment of which is so unsatisfactory to the physician as traumatic neuroses. An ideal plan of treatment is not difficult to devise, but its carrying out will always be Utopian; for traumatic neuroses are more dependent upon external conditions than any other disease. In prophylaxis—by far the most important part of treatment—our best endeavors are thwarted by difficulties due to external circumstances. As was shown at the beginning of this article, traumatic neuroses are most prone to develop in that period immediately following the accident. This nascent stage is the one in which conceptions and influences conducive to the production of the disease are most dangerous, and at this time the influence of the physician is most powerful. Therefore, in the first few days after an accident, an injured person should be under the care of a physician who will take sufficient time to gain the confidence of his patient, and thereby secure a psychical influence over him. Moreover the physician who first treats the case should be possessed of sufficient psychiatric and neurologic acumen to recognize the first sign of a neurosis, and thus to control it. By a careful and full examination—which alone often quiets a nervous patient—and by telling him not to fear any further consequences, the physician may often relieve the mind of the injured person, and at the same time counteract the psychical shock which the patient has suffered by showing him that he regards the accident as of slight importance and a secondary matter. Furthermore, if possible, the physician should attribute the nervous disturbances resulting from the accident to some other cause. Inability to work should be spoken of as merely temporary, so that if possible the patient's anxiety for the future should be at once allayed. Briefly, the physician who first treats the patient should invariably and continuously manifest interest in the patient and give him attention and care, and should also try in every way to divert his mind from the accident, and eradicate its memory.

In reality how are these indications usually fulfilled? After an accident the injured person is generally treated by a dispensary physician or is taken to a drug-store, and given cursory treatment by a passing physician. In either case the physician usually sees the injured person but once, and has no opportunity to acquire an influence over him. At this time the injured man hears from his fellow workmen or from some of the hospital nurses how serious and how prolonged such injuries usually are, etc. After receiving first aid the patient is usually left to the care of a dispensary assistant (in Germany of a Kassenarzt). This physician is under such pressure of work that, as a rule, he treats only the local (surgical) symptoms of the accident without concerning himself with the mental state of his patient. If, during this time, general disturbances are noted for which no apparent reason can at once be assigned, or if they appear to be due to chronic alcoholism, arteriosclerosis, or other condition, the physician, with the hope of consoling the injured, will as a rule ascribe these difficulties to the accident,

saying that they will disappear with the gradual amelioration of the other sequelæ. This method of ascribing all of the patient's difficulties to the accident has a very serious effect on the further progress of the case, and it is strengthened under the following circumstances: if the patient's case is pending in court, or (in Germany, when there are sick benefits, Krankenkasse) if the question arises whether the pathologic condition is the result of an accident or an independent disease in no way connected with the accident. The lodge or society, which in doubtful cases is anxious that the existing condition be attributed to the accident, sometimes brings pressure to bear upon the physician. The firm sued for damages does not share this opinion, and thus the patient is pulled to and fro with conflicting opinions, his mind constantly busy with the origin of his condition. Of course, the knowledge that it will be better for him to be awarded substantial damages than to receive a certain sum of money from his society or lodge, will lead the attending physician to concur in the opinion of the physician of such beneficial society; therefore he will frequently ascribe the difficulties to the accident. Thus the external conditions are directly opposed to any rational prophylaxis.

From the foregoing it might be deemed advisable to relegate the first treatment of the injured to specialists who are most familiar with accident cases, in order to secure the best mental effect upon the patients. But such a proposition is impracticable; for, in the event that the physicians had been appointed by the firm for whom the patient had worked, this would arouse prejudice and opposition on his part. The prophylaxis of traumatic neurosis is therefore not very encouraging, and even if some slight result is attained this is immediately more than counterbalanced by the adverse opinions and the conversation of the patient's friends and fellow workmen.

What measures are at our command when a traumatic neurosis develops in the injured? First, those measures which aid us in psychical treatment must here be employed. How these act has already been explained in discussing the prophylaxis, but even without such explanation, and merely from his knowledge of the genesis of traumatic neurosis, the physician should understand how to treat a patient with this affection. The details of this knowledge cannot be imparted, but spring from the physician's professional tact and general knowledge of human nature.

Secondly, we have at command all those physical remedies which are employed in the treatment of nervous patients in general: hydrotherapy in the form of baths, lukewarm plunge and partial baths, warm or cool ablutions, general or local manual massage, vibratory massage, and faradic and galvanic treatment. Drugs cannot be entirely dispensed with: bromid and valerian, and for many cases opium, are necessary. In patients not wholly degenerate these and similar measures sometimes bring about the improvement of one or several symptoms, or even the entire condition, but this improvement usually lasts only as long as the treatment is continued.

On account of the difficulty of carrying out this therapy, it has been suggested that these patients be treated in a hospital, and in different places institutions have been equipped chiefly for the treatment of traumatic neuroses. These offer the advantages of clinical observation and treatment of the patient by specially trained physicians. Nevertheless, the practical suc-

cess of these hospitals for accident cases appears to be neither great nor encouraging. This is because the injured entertain a prejudice against accident clinics and similar institutions to which I have called attention, and have a strong disinclination to being treated in such. Moreover, the bringing together of many injured persons in one hospital has the disadvantage that the patients, by constant intercourse with one another and from the fact that they continually see the professional examinations of their associates, often acquire an astonishing and most undesirable education in the symptomatology of traumatic neuroses, and subsequently (consciously or unconsciously) make use of this knowledge. Finally, as I know from years of experience, there is engendered in the patients (not only in those suffering from injury but from general neurologic conditions) a certain passive resistance and mute opposition which makes treatment exceedingly difficult, and calls for the greatest patience on the part of the nurses and physicians. Yet if it is desirable for these traumatic neurotics to have the advantage of clinical treatment, nothing remains but institutions for the treatment of internal or nervous diseases or sanatoria. Now, as a rule, such hospitals refuse admission to patients of this kind, for reasons easily understood, and treatment in a sanatorium is usually beyond the patient's means.

As the result of these considerations I can only repeat what I implied at the beginning—that the treatment of traumatic neurosis is in the majority of cases "love's labor lost." Its intractability to therapeutic measures is only slightly due to the fact that hysteria, hypochondriasis, neurasthenia, and related neuroses are all likewise insusceptible to therapy. It is largely because, for most of these patients, the prolongation of the disease secures material advantages without working for them. And although there are many cases in which the affection persists in spite of obtaining damages, nevertheless the chief obstacles in the way of carrying out a rational therapy are a claim for damages and refusal to work.

Yet a method must be devised by which to relieve the injured from material care for his future, and simultaneously to prevent him from directly transferring his injury into cash without an intervening attempt at cure. It should be made impossible for the person partially incapacitated to obtain indemnity unless he makes some effort toward recovery. *It might be enacted that a person who is only partially disabled should receive indemnity in proportion to his disability only provided he is willing to exert himself and to work so far as his condition permits.* Only the one absolutely incapacitated should at once receive damages. In order actually to utilize his remaining capacity for work, the injured first of all needs an opportunity. The meeting of this want should be the object of large manufacturing concerns or, still better, of trades unions and the like. The majority of those partially disabled cannot utilize their powers because employers will not hire such persons when better material is to be obtained. *Therefore coöperative associations should provide employment with wages corresponding to the capacity for work, and thus make actual opportunities for work—only in this way can a claim for damages be justified.* It is clear that the finding of such employment would be an arduous task for the coöperative associations, but such remedial measures may be forced upon us if we are to reduce the present number of suits for damages. It is to-day everywhere recognized that the

salient point in the treatment of traumatic neuroses consists in getting the patients to work, and that compared with this all mechanotherapy and similar measures are far behind.

Recognizing these facts a society has been formed in Berlin which attempts to secure this action on the part of the coöperative associations. The "Society for Accident Cases" in Berlin will aid persons suffering from accident by giving them an opportunity to work, or does this through their own societies. The Society is still young and inexperienced, but there is no doubt of its beneficent work. Some large industrial establishments—for instance, a large brewery in Berlin—have adopted similar methods of aiding their injured workmen.

The furnishing of suitable work to those only partially disabled I must designate as the chief thing to be provided for in the readjustment of accident insurance. Another but less important change would be to modify the regulations concerning work so that only complete disability and *three or four grades* of partial disability should be recognized. Small weekly payments should be altogether tabooed, and indemnity should consist of a single payment.

Experience seems to indicate that by these or similar changes much of the baneful excitement which now attends the payment of weekly indemnity for accidents might be avoided. More might be done in this direction if coöperative institutions were to discard some of their irritating officialdom, and conduct affairs in a more business-like way. Even trivial things must be considered in establishing a new law; its enforcement and beneficent effect depend in a high degree upon psychical coaction.

VASOMOTOR TROPHIC NEUROSES

By R. CASSIRER, Berlin

The realm of *vasomotor trophic neuroses* which we shall review is exceedingly vast. We find here a rich chain of pathologic pictures which sometimes are substantive, and sometimes merge into other nervous symptom-complexes. Among these we include *acroparesthesia, Raynaud's disease, erythromelalgia, sclerodactylia, and scleroderma.* Somewhat apart from these is *angioneurotic edema,* but its relations to the other forms mentioned are so intimate that we shall describe it with this group. In this realm, we often meet with symptom-complexes which can be strictly classified only with the utmost difficulty, which in one respect resemble this, in another respect that, clinical picture, and which unite into every possible mixed or transitional form. If hereby the strict lines of our system are somewhat obliterated, yet certain lines of demarcation are absolutely necessary. The realm of these affections is so vast, and there are such differences between the terminal links of the chain, that a separation into groups appears to be absolutely necessary.

As the name indicates, the symptoms which play the main rôle are chiefly those of *disturbed vasomotility* and *trophic* disturbance. To these are added *sensory* and *secretory* disturbances, while true motor and irritative symptoms are of subordinate importance. Individual symptoms of different kinds may develop. In those of vasomotor nature, we differentiate between a decrease (*local anemia*) and an increase (*local or regional hyperemia*) of the local amount of blood. The first may be subdivided into *local syncope* and *local asphyxia.* The clinical appearances will later become obvious. Change in sensation mostly appears as *paresthesia* and *pain.* Very frequently thermoparesthesia, or objective disturbance of sensation, is of less importance, and this more often appears as hyperesthesia than as anesthesia. The secretory symptoms present themselves as *anhidrosis* and *hyperhidrosis.* But the *trophic* disturbances are of greater significance. When we speak in this way of trophic disorders, we refer chiefly to those which depend upon definite disturbances of some portion of the peripheral or central nervous system. The question as to how this trophic influence arises, what tracts it utilizes, what is its action, has of course been by no means decided, and is still open to discussion. Even to define its outlines would be beyond the scope of this article.[1] I can only state that experimental as well as clinical experience points to the great influence which the nervous system exerts upon the nutrition of tissue; as a rule it appears to be a pathologic change in innervation rather than its complete cessation which produces trophic disturbances. Each

[1] See my monograph: "Die vasomotorisch-trophischen Neurosen," Berlin, 1901, S. Karger.

tissue and cell is nourished by attracting to itself from the nutritive fluids those products which it requires for its nutritive integrity. This function of the cell is subject to nervous influence, but the arrest of the trophic nervous influence does not cause a suspension of nutrition and consequently of growth and new formation, but only decreases and modifies these. "The necessity for nutrition," says Samuel,[1] "lies in the cells, the measure of trophic influence comes from the nervous system." Without these there would be, as we might say, a *vita minima* of the tissue; when there is no special exercise of its activity, its structure may remain undamaged, but any great exertion may change it. The tissue becomes "more sensitive." Thus, when a patient suffers from myelitis, slight pressure on the skin may cause necrosis, a pressure which will be borne without damage by the healthy skin. The slightest external damage to a cornea from which the nerve has been removed produces a severe inflammation. It has often been supposed that such immediate and external proof of damage by a nutritive disturbance sufficiently demonstrated that we were dealing with a trophoneurotic lesion. But this is not correct. The normally nourished tissue of the body has a power of resistance to external and internal damage, and is able to maintain its trophic integrity provided the damage does not go beycnd a certain extent. The slightest irritation which causes destruction will disturb the trophic condition, and the cause of this disturbance is a pathologic change in the nervous influence which is probably conducted by sensory and vasomotor tracts.

We therefore find such trophic alterations as important features of the pathologic pictures which we are here considering. We meet them in the form of local tissue necrosis or *gangrene*, or as *abnormally marked* or *abnormally decreased* growth, also as a special nutritive disturbance which we designate *scleroderma*, and which we shall discuss later.

In addition to the *nature of the symptoms*, we find another common peculiarity; namely, that the *projecting portions of the body* are almost invariably *the seat* of the pathologic change. The fingers and toes, the nose, the ears, and the chin are preferably attacked, and there is a conspicuous tendency to *symmetry*.

We are constantly dealing with *neuroses*, that is, we consider the seat of the affection to be in the nervous system, yet there is no pathologico-anatomical foundation for this hypothesis. Therefore we must assume the existence of "functional" diseases of the nervous system, and the vasomotor apparatus is probably the chief seat of these conditions. The fact that it extends throughout the entire body is one of the chief objections to this minute localization of the pathologic seat. The vasomotor system consists of numerous branches one above the other, and dependent upon each other. A vasomotor center appears to be located in the cortex of the cerebrum. Thence the vasomotor tracts, probably close to the sensory, pass to the great subcortical ganglia where their continuity is most likely interrupted by cell groups. They then pass on through the pons to the chief vasomotor center in the medulla, thence through the lateral divisions of the medulla spinalis to the various heights of the spinal cord. Here they split around the cells of the gray middle substance. From these spinal vasomotor centers new

[1] Samuel, "Die trophischen Nerven," Leipzig, 1860.

fibers arise which pass as white communicating branches to the boundary column of the sympathetic and to the periphery. Here again ganglion cell groups are intercalated, some in ganglions of the boundary column, some nearer the periphery, even in the walls of the vessels.

Physiologically we differentiate two kinds of centers and tracts: *Vaso-constrictors* and *vaso-dilators.* The immensity of the tracts and centers is of the greatest physiologic and pathologic importance. It permits a rapid compensation of disturbance, which is further favored by the fact that the subordinate centers, in contrast to the more important ones, are relatively substantive. This naturally makes a local diagnosis difficult, particularly since we have no means of concluding from the nature of the lesion its seat, while in the motor system, for example, the nature of paralysis, whether spastic or of an atrophic degenerative kind, at once betrays the seat of the disease. Thus all the factors combine to render the pathogenesis and pathology of these pathologic pictures very obscure, and much is left for investigation.

Acroparesthesia.—The symptom-complex which we most frequently meet is acroparesthesia. The following is the history of a case:

A woman, aged 34, came to the Clinic because three weeks previously she suddenly felt pain in her arms, particularly in the right one, which still persisted. She described the pain as deep-seated, of a stabbing and burning character, passing along the bone, and radiating from the elbow-joint to the hand and fingers. The pain was greater at night, the hands and fingers then becoming numb so that she could neither touch nor hold anything, and there was formication. These unpleasant sensations were so marked that they prevented the patient from sleeping. All these symptoms were said to have appeared for the first time three weeks previously after a day of washing. The patient stated that she had lately done a great deal of laundry work. Of former diseases she could report but little. According to her statements, she had quite a severe attack of syncope two years ago, and gastric ulcer fourteen years ago. From time to time she suffers from headache, but has not recently had it.

Examination of this powerfully built, and not anemic, woman revealed no pathologic condition in the internal organs, and objectively, in the nervous system, only that sensation was decreased in the fingers of both hands, the tips of all the fingers being insensitive to pin pricks. In the vola manus this intense analgesia extended to the middle phalanges. In the same region, strong brush contact was not experienced, and sensibility to heat and cold was decidedly disturbed. The nerve trunks were not sensitive to pressure, but in the region of the cervical sympathetic there was a very unpleasant sensation, especially upon the right side. The pulse was not increased, and was the same upon both sides. Vasomotor stimulation of the skin showed no change. There was no disturbance of motion in the hands, the muscles were not atrophic. The skin of the fingers was somewhat remarkable, being extraordinarily hard and dry, and traversed by numerous fissures. These changes were more marked than usual, even in persons of the laboring class.

This case is a typical example of a conspicuous form of acroparesthesia. It occurred in a woman in the fourth decade of life. Women are more frequently attacked than men; among 90 cases which I observed, only 10 occurred in men. I have an impression that a more intense damage is necessary to produce this affection in men than in women. People of a certain age are particularly liable to it. Among 184 cases (my own statistics and those of Frankl-Hochwart,[1] 150 were between 30 and 60 years of age, but

[1] Frankl-Hochwart, "Die Akroparästhesien," *Nothnagel's Spec. Pathol. u. Therapie*, 1898, XI, 2.

I once saw a child aged 7, and at another time a lady of over 70, who presented the same symptoms. Our patient stated that the first symptoms appeared after washing clothes. This is in conformity with other experiences. Occupations which necessitate the use of much water, particularly cold water, are important factors in the development of the disease. Other forms of hard manual labor, also over-use of the hand, may predispose to it. In women there are additional etiologic conditions in the sexual life, the influence of the climacterium, the puerperium, pregnancy, etc. It is especially interesting to note, as I did in four or five cases, that acroparesthesia occurred after operative removal of the uterus and the ovaries with and without other symptoms of *climax præcox.* Several of my male patients were alcoholics. Sometimes trauma may be the cause, compression of the fingers, or a fall upon the elbow as in one of my patients. Sommer also describes a case with traumatic etiology.

The symptoms in my patient were unpleasant or even painful sensations in the fingers, hands and forearms. These are the main symptoms of acroparesthesia. There is a sensation of formication, of itching, burning, or numbness, a feeling as if the finger or the entire hand were swollen. But there may be every transitional stage between these unpleasant sensations and actual pain, which, as in the history just quoted, may disturb the rest at night.

Usually the paresthesia and pain are not continuous, but occur in attacks which are most marked toward morning. Then the fingers become stiff and clumsy, and they can be used only after rubbing, squeezing, or patting them. As a rule, the patients cannot sharply define their sensations. Sometimes the fingers of both hands, sometimes of one hand only, and occasionally the individual fingers, are said to be painful or numb. Exceptionally the upper arm is implicated as high as the shoulder. In my patient there was a conspicuous disturbance of sensation affecting the terminal and middle phalanges of all the fingers of both hands uniformly including their sensory qualities. Here, as elsewhere, the hyperesthesia was by no means limited to a peripheral nerve or its root area. This is important, for both Déjérine and Egger have recently reported cases in which the objective sensory disturbances were limited to the tract of certain posterior roots, and Pick had formerly observed a similar distribution of subjective disturbances. According to my experience, this is by no means the rule. Objective sensory disturbances may be entirely absent, and they are generally neither so frequent nor so well developed and extensive as in the patient whose history was just related.

In many cases the only symptoms are these objective and subjective phenomena. Other cases, however, may show the following symptoms: *Pallor of the fingers and of the hands* simultaneously with the previously mentioned paresthesia, in which sensations of cold frequently predominate. The history of a patient presenting this type, which unquestionably is more rare than the first, is the following:

A servant girl, aged 16, was compelled to work in a cold kitchen. For eight weeks she complained of distress in her hands, numbness, a loss of sensation, and burning of the fingers, especially in the morning and after carrying heavy loads. Frequently the fingers became deathly pale and so stiff that she could not move them. This condition lasted several hours, formication extending to the shoulder.

Examination revealed absolutely normal conditions except for the paroxysmal numbness of the fingers.

Here, in addition to paresthesia, we note **local syncope.** This form of the clinical picture, as already remarked, is rare, and should be differentiated from the first mentioned variety; it forms a transitional stage into one of the incomplete forms of Raynaud's disease.

The affection usually begins with slight attacks and develops gradually, but an acute onset has also been observed, and this was the case with our first patient. When the disease has appeared it usually proves very intractable.

This acroparesthesia is not uncommon in persons who present other symptoms of a general neuropathic condition. The patients are easily irritated or frightened, they are anxious, they cry readily, and are often bad-tempered. Especially do they show the signs of the so-called *neurasthenia vasomotoria,* namely, a tendency to flushes and heat in the face, to vertigo, palpitation, and syncope; I, as well as others, have seen this neuropathic state combined with migraine; on the contrary it must be stated that such a neuropathic diathesis is not invariably present.

I shall not discuss the pathogenesis of the disease. In my opinion we are dealing with a *sensory* or *vasomotor sensory neurosis,* and, as a rule, the peripheral, sensory, cutaneous, and vascular nerve terminations, also the peripheral vaso-constrictor nerves and ganglia are the seat of the disease.

The *diagnostic* difficulties are not great; but we must be careful not to overlook other severe pathologic centers than those referred to by the patient, for acroparesthesia may be a partial symptom of severe organic affections of the nervous system. Not infrequently have I listened to the complaints of patients in whom investigation revealed the signs of tabes, usually of beginning tabes. Sometimes these proved to be (a fact which I can confirm) the early symptoms of acromegalia, a disease first described by Sternberg. Beginning polyneuritis may at the onset also develop similar symptoms. Latent cases of tetany may cause confusion. Most difficult is the differentiation from occupation neurosis. I recently saw a cigarmaker who for weeks had suffered from paresthesia of the tips of his fingers. His occupation obliged him to roll wet tobacco. Suspension of his work in a short time brought about a cure, and this beneficial effect of rest is certainly in the majority of cases a valuable differential guide. Positive differentiation is not always possible, for some occupations are very influential in the development of true acroparesthesia. The avoidance of these deleterious pursuits must often be advised in *therapeutics,* particularly the prolonged use of the hands in cold water should be interdicted; on the contrary, warm water in the form of local baths with or without additions (salt, various extracts) often has a beneficial effect. The symptoms of the patient are sometimes relieved by the use of the faradic or galvanic hand bath.

Raynaud's Disease.—The pathologic picture next to be discussed, Raynaud's disease, is much more rare. This was first reported in 1862[1] under the name of "local asphyxia and symmetrical gangrene of the extremities." The chief symptoms and the course of this remarkable disease are briefly as

[1] Raynaud, "De l'asphyxie locale et de la gangrène symétrique des extrémités." Paris, 1862.

follows: Usually in neuropathically predisposed individuals, but occasionally also in others sensory and vasomotor disturbances gradually develop which are localized principally to the peripheral portions of the body. These are manifested by local syncope, by local asphyxia, occasionally by a regional redness accompanied by severe pain. This redness appears paroxysmally in blotches showing symmetrical arrangement, and may dominate the pathologic picture for days and weeks. In many cases, however, especially in the severe ones, trophic disturbance of the implicated areas appears in the course of the first weeks in the form of an almost symmetrical, superficial, dry gangrene.

The disease may exhaust itself with a single attack of gangrene, or renewed attacks may occur for years. I shall relate the clinical history of a case which presents all the important features of this pathologic picture.

A man, aged 29, formerly a typesetter, and as such coming into contact with lead, had again, as a tavern-keeper, been exposed to alcoholic toxic influences. His preceding history shows that in 1892–3 he suffered from severe attacks of pain in the abdomen which were diagnosticated as neuralgia of the nerves of the liver, and were treated with morphin.

Although he relinquished his occupation as a typesetter, the attacks did not at first cease, but after persisting for five years they disappeared. In the autumn of 1900 new symptoms appeared: After bathing, the index finger of the left hand always lost sensation, it became numb, and as white as if it belonged to a corpse. A few months later a circumscribed black discoloration appeared upon the middle finger, and with great pain a part of the tip of the finger sloughed off. This bluish black discoloration paroxysmally appeared in other portions of the middle finger of the left hand and in the second finger of the right hand. The pains were intense, particularly at night; in the index finger of the left hand a vesicle occasionally appeared, and attacks of local syncope were noted. Ears, nose and feet remained uninvolved. The patient could not be described as of a nervous temperament.

Examination revealed that the terminal phalanx of the left second finger was deformed, the skin was lilac red, and there was a depressed cicatrix. The patient felt as if a wound were being touched. The left third finger was a somewhat darker red and cooler than the others, the terminal phalanges of the second and third fingers were blackish as if they had been dipped in diluted ink, the terminal phalanx of the right middle finger was deformed and pointed, the skin being tough, thick, and wrinkled. The skin at the tip of the second finger was distinctly hardened. Sensation in the hand was everywhere normal, even in the deformed second and third right fingers. Otherwise, examination of the nervous system revealed normal conditions.

Two months later, upon the 19th of February, 1901, the following was noted: The right thumb also was for some time affected. During the examination, local asphyxia periodically appeared, especially in both middle fingers and in the right thumb. The difference in temperature between the individual fingers was very marked; for example, taken with a surface thermometer, in the right little finger it was 93° F., in the right middle finger 62.6° F., in the right thumb 68° F. A second test of the left little finger showed it to be 91.5° F., the tip of the right thumb 73.4° F.

On the 25th of October, 1903, gangrene was no longer present. As the patient stated, the right middle finger remained the focus of the disease. He returned to his occupation of typesetting, and pursued it for a year without any ill effects. In the open air or in a room of low temperature, his fingers became stiff, sensitive to the touch, pale, or blue. When examined they were icy cold. In parts they showed great change, being light red, violet, or extremely pallid, and particularly in the second and third fingers of the right hand, fine brush touches were not felt. The patient reported a remarkable effect that alcohol had upon his fingers: In two minutes after taking a little spirits the activity of the fingers was completely restored.

This patient showed the characteristic symptoms of this remarkable disease. In its well marked forms, it is a *very rare affection*, more frequent in

women than in *men*, and is somewhat more common in infancy. As to the *etiology*, it must be mentioned that occupations carried on in the cold air, or by using cold water, etc., as well as those which expose persons to toxic influences as, for instance, working in lead, are said to be predisposing factors. Trauma may be a cause, especially when it repeatedly affects the fingers (injuries from a circular saw, Brasch; injuries to the fingers of bricklayers, Hess). *Psychical trauma* must also be considered. Dehio's patient was greatly frightened by an attempted rape. The disease has been observed after various *infectious diseases*: Enteric fever, typhus fever, influenza, erysipelas, etc. It has been noticed with particular frequency after an attack of malaria. Symptoms of Raynaud's disease have been observed in the course of chronic infectious diseases, and here both hereditary and acquired syphilis must be considered; from the course it was determined that the affection was actually a manifestation of syphilis. Of course, in such cases we must always seek to ascertain whether or not organic vascular processes, especially gangrene, have produced the symptoms.

Without doubt, the most important of all etiologic factors is the *nervous predisposition* of the individual, whether acquired or congenital.

In my patient (whose history has just been given), as is usually the case, the disease was ushered in by *vasomotor* symptoms. Three different forms may be differentiated: *Local syncope* (regional ischemia, Weiss [1]), local asphyxia (regional cyanosis), and *regional hyperemia* which was not correctly appreciated by Raynaud. The parts attacked by syncope, usually one or several fingers, more rarely the toes, suddenly become white and cold (dead fingers). At the same time there is usually paresthesia, also pain which may increase enormously in severity. Pallor and coldness disappear, the affected part resumes its normal color or becomes intensely blue, even bluish black (local asphyxia), while in other parts under reaction redness and heat appear. These vasomotor symptoms may occur in irregular order, affecting at one time this, at another time that, portion of the implicated area, and may produce an actual play of colors.

With asphyxia, as with syncope, there is usually a decided decrease in temperature; this may be extreme, falling nearly 20° C. below the normal temperature of the affected surface. As stated, this decrease in temperature was observed thermometrically in my patient. We were actually startled when the patient's ice-cold fingers were touched. The affected areas are also often swollen.

The duration of the vasomotor symptoms varies greatly. The individual attack may terminate in a few minutes; it may last for hours or even for days, and may recur several times during the day, etc.

The vasomotor symptoms are followed by *gangrene*, at least in the typical cases. Small vesicles form which rupture and leave ulcers that very slowly heal. At other times the vesicles do not rupture but dry up, or the skin thickens from the onset, to be later sloughed in hard, brown crusts. The slight extent of the gangrene is characteristic. It is usually limited to small areas of a phalanx; very seldom is an entire phalanx sloughed off. As a rule, it affects symmetrical areas, but asymmetry has also been observed,

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[1] M. Weiss, "Ueber symmetrische Gangrän." *Wiener Klinik*, 1882, p. 347.

and has been attributed to certain individual conditions. For example, I saw a case of this kind in which there appeared to be a congenitally weaker development of the arterial vascular system of the affected arm. The seat of the gangrene is usually a finger, a toe, the margin of the ear, more rarely the nose, the tip of the tongue, the nipple, or the lips. The nutrition of the affected areas may also be modified in another manner. These changes belong largely to the realm of *sclerodactylia,* a condition to which I shall revert later. But the parts may become larger, hemorrhages may take place into the skin or into the subcutaneous tissue, changes in the nails are common, whitlow which runs its course with intense pain has often been observed, and this is important in the differential diagnosis.

I saw a patient, aged 32, who was suddenly attacked by ulcers in the fingers, the tips of the fingers became black, the superficial areas and even portions of bone sloughed away, and there was very severe pain; recovery was extremely slow. Since that time pain in the fingers has persisted although there has been no whitlow for seven years. The hands are now deformed; bilaterally a considerable part of the terminal phalanx of the second finger is absent. An X-ray picture shows that the bone also is implicated. In the pulpæ of most of the fingers there are superficial cicatrices, the fingers *in toto* are bluish red, cyanotic, and cold, and in them sensation is somewhat below normal.

Always prominent among the symptoms is *pain,* which in fact occasionally appears to be excruciating; Raynaud says: "I have known persons, otherwise quiet and patient, to howl with pain, their bodies bent and contorted as they sit in bed. These unfortunate subjects appear to pass their entire time in seeking a position which will afford some relief." Sometimes the pains are not severe, and like the very slight objective sensory disturbances, they are usually not circumscribed.

Occasionally *secretory* disturbances, particularly hyperhidrosis, accompany the vasomotor symptoms. Motor phenomena are here very inconspicuous, but no doubt localized motor symptoms, particularly atrophy of a non-degenerative character in the muscles of the hand, do arise in the course of Raynaud's disease; I have reported such an instance.

In one case Raynaud observed a very peculiar ophthalmoscopic picture. The patient called his attention to the fact that while he was able to see during the attack after it passed away there was disturbance of sight. Ophthalmoscopic examination showed that during the time in which the sight was affected there was an abnormal contraction of the central artery of the retina at its origin in the vicinity of the papilla; Weiss, as well as a few later observers, saw a similar condition, but, upon the whole, such findings are rare. Periodic contraction of the arteries has also been noted in other isolated areas. Usually, however, the pulse shows no change; this may be proven graphically.

Interstitial abnormalities are sometimes observed in the secretion of urine. The coincidence of *paroxysmal hemoglobinuria* and Raynaud's disease is peculiar. The relation of these symptom-complexes to each other varies; sometimes the symptoms of Raynaud's disease are prominent and hemoglobinuria is scarcely noticeable, at other times the reverse is true. Here it must be noted that the pathologic picture of paroxysmal hemoglobinuria is in many respects similar to paroxysmal symmetric gangrene. It would lead

us too far from our theme were we to discuss this interesting subject. Paroxysmal *albuminuria* and paroxysmal *melituria* have also been observed. In such cases the diagnosis of Raynaud's gangrene must, of course, be cautiously made in order to prevent confusion with gangrene due to vascular disturbance, particularly when an intermission of the symptoms is observed. A moderate excretion of uric acid has also been noted. English authors have ascribed great etiologic importance to the uric acid diathesis. My experience leads me to assume that gouty persons and members of gouty families show a great tendency to variations of the vasomotor innervations.

The heart and the vascular system usually show no organic changes. In a few cases, besides typical Raynaud symptoms, an organic cardiac affection existed, apparently, independent of these. I saw an instance of this. There are also a few cases reported in which, besides arteriosclerosis, the typical symptoms of Raynaud's disease appeared intermittently. But all of these are exceptions, the diagnosis of which necessitates special care.

As a rule, the *general condition* of the patient is not below par. Disturbances occur gradually, and are usually due to the pain, the processes which cause gangrene, etc.

As has been several times reiterated, the course of the disease is typically intermittent. An attempt has been made to divide it into different stages, but this adds little to our knowledge of the affection. Vasomotor symptoms usually precede the gangrene which appears with an increase of pain. The acme of the disease appears to be reached in the area first affected, but paroxysms may develop in other areas. The disease is often exhausted in a single attack which comprises several paroxysms. This was the case in the two patients whose histories I quoted; the various later disturbances were merely sequelæ; but renewed attacks may appear at shorter or longer intervals. In the subsequent course the paroxysmal type gradually becomes obliterated, and the intensity of the symptoms decreases. There are, however, cases in which the vasomotor symptoms (from the onset) show a chronic and progressive development; certainly the local asphyxia points to such a form. In addition to the other deviations from type we frequently note that the gangrene and the pains become less marked, but, in their place, other trophic disturbances are observed, enlargement of the fingers or other peripheral parts, in which, however, the bones are not involved; in other cases objective sensory disturbances are very conspicuous. I have described such cases as *acroasphyxia chronica*, and have attempted to separate them from the ordinary cases of Raynaud's disease. Other authors (Hirschfeld, Péhu) have observed similar cases, but their number is small.

Unquestionably there are many cases in which only vasomotor disturbances persist, in which gangrene never occurs, or, at least, only in the later course, in which there are slight trophic disturbances, such as thickening or enlargement of the terminal phalanges, and hardening of the skin. Some of these types are the forms which undergo a transition into Nothnagel's *vasomotor neurosis*, some are closely allied to *sclerodactylia*, and perhaps are cases of the last named affection.

The fact that we not infrequently meet with a combination of Raynaud's disease and *diseases of the central nervous system* is especially interesting. Among the latter is tabes dorsalis, particularly gliosis spinalis, syringomyelia

(Schlesinger, Pospelow), also tumor of the spinal cord or of the spinal cord roots. We frequently find Raynaud's symptoms in hemiplegia; on the other hand, I am aware of no positive case of neuritis combined with Raynaud's disease.

Functional disturbances of the nervous system are common; the neurotic diathesis often paves the way for the disease. Among other symptoms we find those of neurasthenia, traumatic neurosis, chorea, epilepsy, and also of hysteria. In many of these cases Raynaud's symptoms are merely the signs of a general neurosis, and lose their substantive character.

These disturbances are most frequent on the part of the nervous system, and may be regarded as an expression of especial sensitiveness of the vasomotor system: Impaired tone of the cutaneous vessels, dermographia, tinnitus aurium, vertigo, a tendency to vomiting, to urticaria and to transitory edema. The combination with migraine and Graves' disease has frequently been noted. Such pathologic pictures have been described by Solis-Cohen [1] and Herz [2] as *vasomotor ataxia.*

Our knowledge of the *pathological anatomy* of the affection is very limited, in spite of the no small number of cases in which necropsy was performed. Changes have been found in the peripheral nerves; to this, however, I attach no causal significance; also changes of the peripheral vascular system, especially of the smaller arteries, which are, however, only of secondary importance.

In regard to the *pathology,* the vasomotor symptoms of syncope, asphyxia, and hyperemia unquestionably depend upon the abnormal irritative, and perhaps also paralytic, symptoms of the vascular nerves, yet we are unable to define all of the individual points. Gangrene is certainly not merely the consequence of vasomotor symptoms; nor can it be referred to changes in the peripheral nerves which are most likely of secondary nature, and the slight organic changes in the central nervous system so far demonstrated cannot be regarded as the cause. Nevertheless I do not doubt that the gangrene in Raynaud's disease is a neuropathic form of gangrene. Upon the whole I regard this malady as a "functional" disease of the vasomotor and trophic tracts and centers. The pathogenesis of the pathologic picture does not appear to be uniform. Congenital loss of resistance and an acquired lability in which cold, intoxication, and infection play a rôle, perhaps also reflex changes in diseases of the peripheral organs (mostly of the vessels, rarely of the nerves) are prerequisites for the development of the affection. Whether the *locus morbi* is always the same has not been demonstrated. I have already stated that, at the present time, it is well not to speak of Raynaud's disease but of Raynaud's symptom-complex.

The *diagnosis* and the differentiation from other diseases must be briefly discussed. If we remember the criteria of the disease previously given, its recognition will usually not be difficult, but its relation to such affections as acroparesthesia, erythromelalgia, scleroderma and angioneurotic edema are so close, the transitional stages between these affections so ill-defined, that discrimination is not always possible, and not always desirable. The differ-

[1] Solis-Cohen, "Vasomotor Ataxia: A Contribution to the subject of Idiosyncrasies." *Am. Journ. Med. Sciences,* CVII, p. 130.

[2] Herz, "Ueber vasomotorische Ataxie," Berlin, 1902.

ential diagnosis from syringomyelia, above all from one of its secondary forms, Morvan's disease, is important. I must emphasize the fact that in the latter the course is a chronic and progressive one, that the whitlow is usually painless, and that motor disturbances and sensory phenomena are more prominent. Some cases of leprosy resemble Raynaud's disease, perhaps also certain forms of beri-beri. When gangrene appears in the course of a cardiac or vascular affection, the differential diagnosis from senile gangrene and gangrene due to endarteritis obliterans also raises the question of the differential diagnosis from Raynaud's disease. The paroxysmal, preceding, vasomotor symptoms, the slight extent and the localization of the gangrene, will usually point to Raynaud's disease. The differential diagnosis from *intermittent claudication* (limping) sometimes necessitates careful consideration. The long persistence of sensory symptoms and the dependence of the disturbances upon the use of the legs are typical of this affection. *Gangrene due to ergot* produces a picture similar to Raynaud's disease. A careful history is of the utmost importance, and this also is true of the differentiation from chilblain.

The disease in itself does not threaten life, but it may become very distressing, and last for years or decades.

The *treatment* must be of two-fold nature: First, prophylactic measures, which consist mainly in the avoidance or decrease of thermic deleterious agents, particularly of the effect of cold. The period of suffering for these patients is winter.

Second, *constitutional treatment* which is directed against the underlying neurotic or, rather, the vasomotor neurotic diathesis. Quinin has been especially advised, in solution (2.0:180 = a tablespoonful three times daily, Herz), as a tincture, or in combination with ergotin, strychnin, or iron. From the vasodilators, amyl nitrite, nitroglycerin or trinitrin, not much is to be expected; occasionally they mitigate an attack, but for this purpose alcohol has the same effect and is a much better remedy; its beneficial effect was praised by one of the patients whose history I have quoted. Hot drinks, as I know from experience in other patients, are likewise beneficial. Adrenal extract is highly recommended by Solis-Cohen; I have not had much experience with it; at all events, it is not harmless, and we should be cautious in its use. Cocain has been advised by Herz (0.1:60 = a teaspoonful three times daily). Derivatives may also be tried. But the greatest care is necessary in making local applications to the diseased part. Raynaud once saw asphyxia of the hands immediately change to gangrene while in a local mustard bath. Treatment by laxatives is better: Laxative waters, Carlsbad salt, Marienbad tablets, etc. Hydriatic procedures are often of advantage. I have repeatedly seen good results from lukewarm hand baths, sometimes with the addition of table salt; Tallermann's hot air apparatus may be tried. Mild massage is also beneficial. Electricity was praised by Raynaud; but a remedy to attack the affection at its root we do not possess. Sometimes amelioration follows the use of the faradic or galvanic hand bath. Herz advises galvanization of the head, the intensity of the current to be gradually increased. In the last few years I have several times seen good results in a form of local hyperemia from bandaging the upper arm with a firm flannel bandage so that distinct venous stasis appeared. This was done daily for a few weeks,

the bandage being kept on for about ten minutes. Courtney reports favorable results after the application of a tourniquet. The *symptomatic treatment* of the pain and gangrene requires no special description.

Erythromelalgia.—In 1872 Weir Mitchell [1] described a pathologic condition characterized by the paroxysmal appearance of *pain, reddening* and *swelling of the feet,* to which he gave the name ERYTHROMELALGIA, "red, painful limbs." Lannois [2] added to our knowledge of this affection by his description in 1880 of this unquestionably very rare affection; up to the present time perhaps 90 to 100 cases in all have been reported. In regard to the *age* and *sex* of those attacked by the disease, nothing noteworthy is reported. As deleterious agents, thermic influences are especially to be considered. The predominant symptom of the disease is peculiar *pain,* a burning and stabbing sensation of great intensity, "as if living fire were under the skin." The pain either sets in with great severity or develops gradually to its height; it is increased by letting the painful member hang down, by heat, and by exertion. Simultaneously with the pain or a little later *redness* and *swelling* appear. The redness is that of active hyperemia, the affected areas are light red to purplish red, the arteries pulsate, the veins are dilated.

The temperature in the diseased parts is increased, being 5° to 8° F. higher than in the normal parts. In contrast with this, during the subsequent course or sometimes with the appearance of cyanosis, the temperature declines.

The *sensitiveness* of the affected areas is *increased,* even the pressure of the bed-clothes annoying the patient; stockings and boots can no longer be worn. Otherwise, sensation is usually normal. The disturbances occur periodically, and are produced by a number of causes: By heat, by a pendant position of the affected portion, and by movements. Most frequently the feet are implicated, sometimes the toes, sometimes the ball of the foot, sometimes the heel. Usually both feet are symmetrically affected, more rarely both hands, or merely one foot or one hand may be involved. Sometimes the disturbances are limited to the region of a single nerve. As accompanying symptoms I must mention disturbances in the secretion of sweat, usually hyperhidrosis. Not rarely trophic disturbances are present, although this was formerly denied. There are usually dystrophic changes which are only slightly conspicuous; hyperplasia of the connective tissue, changes in the nails, and either thickening or atrophy of the skin. Gangrene has also been mentioned. In my opinion the cases in which gangrene appears do not belong to this affection. I believe they should be attributed to endarteritis obliterans or to intermittent claudication. In a few instances, however, true neurotic gangrene was present. These then are the transitional cases of Raynaud's disease. I observed a case in which the symptoms of both neuroses were combined.

The pure cases in which only the local symptoms are present form about one-fourth of all the cases reported. Those which show signs of a general neurotic diathesis may be determined in about the same proportion. The

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[1] Weir Mitchell, "On a Rare Vasomotor Neurosis of the Extremities." *Philadelphia Med. Times,* 1872, p. 81.

[2] Lannois, "Paralysis vasomotrice des extrémités ou erythromelalgie." *Thèse de Paris,* 1880.

cardiac and vascular nervous systems are sometimes most intensely damaged, and we also see the symptom-picture develop upon the base of marked hysteria. It is also noted in organic diseases of the brain: In multiple sclerosis (Collier), in tumors (Schlesinger), in gliosis spinalis (Pospelow), and in tabes dorsalis.

Finally, there are cases in which the symptoms of erythromelalgia are limited to the region of *one or a few definite peripheral nerves* without other symptoms of neuritis being discernible. The regions of the median, the internal plantar, the posterior tibial, and the radial nerves have thus been found diseased.

Pathologic anatomy has furnished no conclusions regarding the nature of the disease, although a few investigations have been reported. Changes have been found in the peripheral nerves of the posterior roots and in the small arteries, but there is no unanimity between the individual reports. The symptoms appear, as a rule, to be the expression of irritation in definite vasomotor (dilator), sensory, and secretory tracts and centers. In one group we may assume that the pathologic focus is situated in the peripheral nerves, and these are the cases in which the symptoms are limited to a circumscribed region of one nerve. In the future we must observe whether this is not, on the contrary, a root distribution; disease of the posterior root appears to be a very plausible theory for these cases. In the second group the assumption of a central nervous genesis appears to be best founded. The areas attacked are apparently of the same physiologic importance as those of the first group: Vasomotor, secretory, and sensory elements. Here then we are dealing with a central (spinal or bulbar) neurosis with vasomotor, trophic, sensory, and secretory symptoms. Whether the posterior gray substance is, as Eulenburg and recently also Lannois and Porot have believed, the point of lesion is still undecided. That a disease of peripheral vessels may produce similar phenomena I cannot deny. In this instance it may be as it is in various forms of muscular atrophy in which the primary muscular and primary neuropathic symptom pictures closely resemble one another. At all events, it appears to me necessary to regard a number of cases of erythromelalgia as nosologic entities.

The *diagnosis* is not difficult if we bear in mind that we are dealing with a combination of vasomotor and sensory symptoms which form the picture of the affection. Vasomotor disturbances alone are not sufficient basis for a diagnosis. From them may result such pathologic pictures as erythrodermia, erythromelia and the like. Nor are merely sensory symptoms sufficient. Here we must differentiate between the more or less substantive topoalgias of the foot (tarsalgia, talalgia, etc.). As a rule, the differentiation between certain vascular processes causes the greatest perplexity. Reflex disturbances of innervation apparently occur in a vessel changed by arteriosclerosis, and this leads to active hyperemia and thence to pain. This furnishes an explanation of the many points of similarity which exist between this affection and intermittent claudication.

The *course* is usually very protracted, but improvement may take place even after a long duration of the affection. In a purely symptomatic sense, we attain most in the treatment by keeping the member in a horizontal position, and by the application of cold in some form. Electrical treatment

should be tried (galvanic local baths). Operative treatment such as neurectomy has been several times proposed, and nerve stretching has been recently performed. The results of these operations are not particularly brilliant; nevertheless, in very stubborn cases it seems that nerve stretching should be at least tried.

Scleroderma.—The symptomatology of Raynaud's disease has made us acquainted with certain nutritive disturbances in the skin of the peripheral parts, which we designate as *sclerodermatic.* There is also a disease in which this trophic disturbance of the skin is the most conspicuous symptom. This disease, SCLERODERMA, is no doubt related to Raynaud's disease, at least to some of its forms, while others deviate so far that a combination of the pathologic pictures does not appear feasible.

Scleroderma appears at all ages, but preferably in the third and fourth decades of life. Women are most frequently affected. In the etiology *thermic causes* are especially to be mentioned: Damp dwellings, working in the wet, a fall into water, etc. Psychical shock occasionally appears to have some influence, especially when it occurs in persons with an acquired or congenital *neurotic diathesis.* Rarely has an organic nervous disease been found. Occasionally it is combined with Graves' disease, Addison's disease, or hemiatrophia facialis.

The first symptom noted in a patient suffering from scleroderma is a change in the skin, which becomes hard, firm, and tense; often there is a marked luster, a glass-like transparency, and occasionally it looks as if lacquered. It gives us the impression of being too tight for the parts included within it, like a too-narrow glove; the small folds of the skin disappear, and if these changes affect the skin of the face it assumes a peculiar mask-like expression. The appearance of such a patient with his immovable features, his narrow pointed nose, his drawn and contracted mouth, eyes which can be closed only with difficulty, and the glistening luster of the skin, is exceedingly characteristic; the picture is impressed upon every one who has once seen it. Occasionally an edematous stage precedes this indurative stage. During this time the affected area of the skin is somewhat swollen and edematous, but there is no pitting upon pressure with the finger. The changes in the skin vary in different cases. They may appear acutely, and increase greatly in intensity and extent in the course of a few days or weeks, or may gradually develop in the course of years, and show a steady progress.

Often a patient will show abnormal *pigmentation,* partly pigment atrophy, partly pigment hypertrophy, in various areas of the body. Pigmentation may become so marked as to suggest a combination with Addison's disease, but usually true Addison's disease does not exist.

The secretion of *sweat* and of the *sebaceous* follicles is disturbed as well as the *growth of the hair*; more significant is the *vasomotility.* We meet with local cyanosis and local syncope as well as with local hyperemia, and a form of scleroderma, namely, sclerodactylia, is marked by the fact that vasomotor symptoms appear early and either persist for a long time, or permanently. Local changes in the temperature of the skin are frequently noted in these forms.

Sensory disturbances play no great rôle. Paresthesia sometimes appears at the onset. It is usually not limited to a certain nerve region but is diffuse.

There are rarely any objective sensory disturbances, even in the parts which are most markedly altered.

Trophic changes are not limited to the skin but affect also other tissues, and in the light of most recent investigations we may state that all the tissues of the body are affected. The changes, as a rule, pass directly from the skin to the muscles, but occasionally the muscles are affected in areas in which cutaneous changes are either slight or wholly absent. I recently observed a case in which the myoscleroses were much more extensive and intense than the dermatoscleroses. Of course this increases the difficulty of differentiating myositis interstitialis, muscular induration. Furthermore, there is atrophy of the *bones* with shortening and thinning (acromicria). I was several times able to determine this in the X-ray picture. If the *joints* are implicated, the differentiation from chronic articular rheumatism becomes very difficult. Only exceptionally are the *mucous membranes* affected. A significant fact in the prognosis is that the internal organs may be affected by sclerotic and atrophic processes, and cachexia often appears in the later stages of the disease; thus scleroderma finally becomes a systemic affection.

The manner in which sclerodermatic changes appear has led to the subdivision of scleroderma into distinct forms. Scleroderma of the entire body is quite rare; in such extreme cases the individual resembles a mummy, a condition Grasset has described by word and picture. In contrast to this *scleroderma diffusa*, which is not always complete, is *scleroderma circumscripta*, in which we recognize the striaform or flake-like arrangement of the sclerodermatous plaques. Under diffuse scleroderma, owing to its special localization, we differentiate *sclerodactylia* which chiefly affects the hands, more rarely the feet. This latter form bears, as I have several times indicated, the closest relation to other vasomotor trophic neuroses. It is certainly not a disease *sui generis*, but besides its localization it has a few individual characteristics which I must mention: The frequent implication of the deep-lying tissues, the tendons, fascia, muscles, joints and bones, the well marked vasomotor and sensory disturbances which often accompany these conditions, the frequent deviation of the trophic disturbances from the true sclerodermatic type so that, besides scleroderma, more or less distributed gangrenous processes also occur. These cases are so closely related to Raynaud's disease that, not rarely, their positive differentiation from it is impossible.

The relations between scleroderma and *hemiatrophic faciei progressiva* are very close. Hutchinson simply classifies the latter as scleroderma localized to the region of the trigeminal nerve. It usually affects young persons, most commonly those between 10 and 20 years of age; we are uncertain as to the cause, but a neurotic predisposition appears to play a rôle. It begins gradually; in a limited area, perhaps in the brow or cheek, there is atrophy of the skin, as well as of the subcutaneous tissue, the bones, and also the muscles. From this starting point atrophy involves gradually the entire half of the face; there is a diminution of the size of all parts. Changes in the pigment, and anomalies in the secretion of sweat, in the secretion of the sebaceous glands, in the temperature of the skin, and in the growth of the hair appear, similar to those of scleroderma. Just as in the latter affection, there are no objective sensory disturbances, but paresthesia and even severe

pain resembling true neuralgia of the fifth nerve are frequently observed in the diseased area. I recently saw a case of scleroderma of the trunk and of the extremities without hemiatrophy combined with neuralgia of the fifth nerve. The combination of scleroderma and hemiatrophy has been reported by various authors, and in the main the opinion appears to be correct that hemiatrophy is only a specially localized form of scleroderma. There are, however, cases of scleroderma in which the sclerodermatous changes are limited to the region of one or a few nerves. Recently the distribution of scleroderma throughout certain root territories has been recognized.

In the majority of cases the *course* of scleroderma is exceedingly chronic. Cases lasting thirty and even forty-eight years have been described. In these chronic cases the progress is not always steady; there are stages of arrest and even of apparent cure; but these are most common when the changes have appeared acutely, especially in children. Sclerodactylia shows the least tendency to recover. Death may be due to cachexia or to the implication of vital organs. More frequently the patients succumb to some intercurrent affection.

I shall not minutely discuss the local and *anatomical* changes of the skin. They are chiefly evident in the corium, but are also found in the subcutaneous cellular tissue; they lead to flattening of the papilla, to an increase of the substance which produces cement, and an increase of the connective tissue trabeculæ. Of other tissues which are implicated, the vessels must be especially mentioned. We find fibrous changes in all of the three coats of the small cutaneous arteries, while in contrast to this the peripheral nerve branches are usually normal. The process in the muscle is designated as myositis interstitialis. Other changes (as in the bones, joints, and internal organs) are usually due to inflammatory hyperplasia of the supporting tissue with subsequent cicatricial atrophy. So far as we know, the central nervous system shows no important anatomical change. The anatomical findings a few times noted are apparently without significance in regard to the nature of the disease, although they are quite necessary in the individual case. Occasionally, as in a case reported by Westphal, these appear merely to indicate a localization of the sclerodermatous process in the brain. At all events we have nothing but hypotheses as to the nature of the disease, and I shall not detail those which have been utilized to explain the affection; my opinion is that the disease has its seat in the sympathetic nerve of the central nervous system and that it is to be regarded as an *angiotrophoneurosis*, as also is Raynaud's disease which is closely allied to it.

The *diagnosis* rarely causes perplexity if the characteristic cutaneous changes can be determined. When these are not so prominent, and there are alterations in the deeper lying structures, especially the joints, bones, and muscles, its recognition is difficult; in this case, as has already been stated, there are transitions into and relations to chronic articular rheumatism, to myositis interstitialis, etc. Among organic nervous affections, the differentiation from Morvan's disease is chiefly to be considered. In scleroderma, or sclerodactylia which is here most in point, the absence of sensory disturbances, of muscular atrophy and paralysis, is characteristic. The differentiation from leprosy is of primary importance. *Treatment* has, in the main, not been successful, but good results have sometimes followed

certain methods. For a time treatment by thyreoidin was employed. This was based upon very uncertain theoretic conclusions, and the result of the treatment has been quite problematical. Adrenal extract has also been unsuccessful. Of drugs used internally salol or the preparations of salicylic acid have occasionally been beneficial. Thiosinamin in a 15 per cent. alcoholic solution has several times been highly praised; as it is generally used to loosen cicatricial tissue, its employment in scleroderma appears rational. Massage is unquestionably of use, as are also hydriatic procedures, such as warm douches, and peat, sulphur and ichthyol baths. Neumann strongly advocates hot air treatment in Tallermann's apparatus. Brocq states that he has seen brilliant results from electrolysis.

Acute Circumscribed Edema.—In 1882 Quincke[1] described a new pathologic picture, the main symptom of which was *transitory edematous swellings* in circumscribed areas. Although some authors had published cases belonging to this category before Quincke's report appeared, yet the attention of physicians was generally directed to this affection solely by Quincke's researches, and these were followed by a number of publications. The disease has been known by various names: Quincke called it "*acute circumscribed cutaneous edema.*" Among other designations I shall mention only that of Schlesinger,[2] the one most often employed of late, "hydrops hypostrophus." I call the disease "acute circumscribed edema." By this I intend to indicate edema, not cutaneous edema, and that the disease occurs in many other portions of the body besides the skin. If we wish to attach an author's name, the affection should be called "Quincke's disease."

It is not a rare affection. With a great number of nervous patients, we may meet it every few months. It generally occurs in youthful persons. From 130 cases I calculated an average age of 25.8 years; after the fortieth year the number of cases decreases rapidly, the aged being almost entirely exempt.

The affection belongs to those which we designate as *family* affections; not rarely it occurs in several members of the same family. Osler has published the ancestral tree of a family in which this disease appeared in five successive generations. Other cases of neurotic predisposition are also mentioned; migraine was often observed in the ascendants. As a factor which immediately produces the disease we occasionally find exogenous intoxication. Alcohol is often mentioned, also carbonic oxid poisoning. In contrast to the closely related *urticaria*, there is no decided influence from the ingestion of certain foods. Occasionally the eating of fish or mushrooms has been reported to be the cause.

The relation of the affection to *nervous diseases* is especially interesting. Transitory swellings have been observed in tumors of the spinal cord, in tabes (especially as an accompaniment of the lightning-like pains and confined to their area), and in gliosis, more commonly in some neuroses, in neurasthenia, and in hysteria, but most often in migraine and in Graves' disease. In all of these instances the edema apparently had only the value of an incidental symptom. Thermic agents were reckoned as the immediate

[1] Quincke, "Ueber acutes umschriebenes Hautödem." *Monatsh. f. prakt. Dermat.*, Juli, 1882.

[2] Schlesinger, "Hydrops hypostrophus." *Münch. medic. Wochenschr.*, 1899, Nr. 35.

cause, especially the effect of cold, and no matter how skeptical we may be, was undoubtedly the etiology in a number of cases. Under these circumstances, it is frequently only the parts of the body not covered by clothing, therefore, the face and hands, which are attacked by edema. I recently saw a woman in whom edema developed after exposure to a severe hail-storm. Only the parts which the patient could not protect were affected. Here, in addition to the influence of cold, local trauma may have played a rôle, as it has occasionally in other diseases. In a closely related affection, designated *epidermolysis bullosa hereditaria*, the causative effect of slight trauma is well known. Anger and excitement also favor the appearance of edema.

The chief symptom of the disease is a *rapidly developed swelling*, which is more or less *sharply defined*, is pale or of *normal color*, in rare cases *somewhat red*, and soon produces a feeling of tension, sometimes with marked itching; the impression of the finger does not cause pitting, or for but a very brief time, and the swelling *declines after a few hours or days*. When it disappears in one part, it reappears in others, and the disease is usually not exhausted in one attack, but there are intermissions followed by a varying number of renewed attacks.

The extent of the swellings may vary. They may be as large as a pea or hazelnut, at other times the size of a plate or even larger. They are situated in the skin and in the subcutaneous connective tissue, but apparently may also be in the deeper parts and in the periosteum. In consistence they are coarse, elastic, somewhat like a contracted muscle, and, as previously mentioned, the pressure of the finger produces no prolonged pitting.

The color of the skin is usually paler than normal, occasionally there is a slight or marked red discoloration. The temperature of the skin usually corresponds to the degree of redness. In rare cases there are extravasations of blood which cause the edema to assume a blue or bluish red color. There may also be severe cutaneous hemorrhages, yet the edema be slight. Such cases, however, should be regarded as *purpura*.

The acute development of the swelling is an important symptom. In a few moments decided edema may appear; as a rule, however, hours elapse before the maximum is attained. The swelling remains at its height for some time, and then disappears without leaving a trace except that, should this area be re-attacked, permanent changes may gradually take place, but are usually not very marked.

Subjective symptoms may be entirely absent. With decided swelling there are, of course, some symptoms such as *stiffness* and *difficulty in moving* the affected parts. Sometimes, however, the edema is accompanied by intense burning and itching, hence the pathologic picture closely resembles ordinary urticaria, especially if, instead of pallor of the skin, we have dark red, burning edema, therefore true urticaria. By transitional stages the two diseases merge into each other.

Edema may appear in different areas; some portions of the body are more commonly implicated than others, but no part is exempt. According to Collins,[1] among 71 cases in 29 the first swelling was in the face, in 22

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[1] Collins, "Angioneurotic Œdema." *Am. Journ. of Med. Sciences*, 1892, CIV, p. 654.

in the extremities, in 3 in the genitalia, in 3 in the back of the neck, and in one behind the ear. There seems to be no preference for the projecting parts of the body, and the edema is rarely symmetrical. The area in which the edema first appears often becomes less resistant to later attacks. These cases with definite localization do not warrant the construction of special pathological forms. *Transitory edema of the eyelid* is undoubtedly a variety of this affection.

Edema may also attack the *mucous membranes*, and most frequently that of the *mouth, pharynx* and *larynx*. Edema of the larynx produces very marked, and sometimes alarming symptoms, such as difficulty in deglutition and dyspnea. In such cases scarification may become necessary; according to recent reports there is no doubt that edema of the larynx so produced may be the immediate cause of death (cases of Collins, Mendel and Sträussler). Fortunately, these are great exceptions. Even edema of the lung was once regarded as a partial phenomenon of acute circumscribed edema (Quincke and Gross).

Of other mucous membranes which are attacked I must mention the *conjunctiva* and the mucous membrane of the *nose* (nervous coryza), etc. Certain intermittent gastrointestinal symptoms the genesis of which is not quite clear are important; for instance, pain, persistent vomiting, meteorism, constipation, exhaustive diarrhea, extreme and burning thirst. The gastric symptoms may be so severe as to closely simulate the picture of a gastric crisis.

Schlesinger observed paroxysmally a swelling of the tendon sheaths. I saw several times, particularly in one case, peculiar painful contractions of certain muscles, among others the biceps; the muscles were very sensitive to pressure, but this condition lasted only a few hours. From the entire course of the case I concluded that the condition was analogous to edema (edematous infiltration of the muscles). Another localization is important, particularly because it forms a new disease: Intermittent articular dropsy (*hydrops articulorum intermittens*). This is a very rare symptom-complex, but is usually seen in nervous individuals. The chief symptom is swelling of one or several joints, almost invariably of one or both knee-joints. The swelling is often painful; there is no fever; the symptoms yield after a short time, but reappear at regular or irregular intervals. Schlesinger first called attention to the fact that this intermittent articular dropsy is merely a variety of hydrops hypostrophus, as is proven by the transitory swellings of the skin in this affection, and by the fact that there is articular swelling in ordinary circumscribed edema.

Certain *cerebral* symptoms must be regarded as indicating the special localization of transitory edema. Lassitude, somnolence, headache, even serious symptoms of cranial pressure, loss of consciousness, slow pulse, and general convulsions have all been observed under these circumstances, and the symptoms were consequently referred to effusions in the meninges which were analogous to cutaneous edema. In some cases polyuria and transitory albuminuria have been observed and, exceptionally, paroxysmal hemoglobinuria. I have reason to believe that the latter occurred in one of my cases.

Sometimes the attacks begin with *general symptoms*: Lassitude, malaise, loss of appetite, slight fever (in one case I found 100.8° F.).

Quincke's disease shows certain relations to, and transitions into, other vasomotor trophic neuroses. I have several times seen the combination of transitory edema and acroparesthesia. In one of my cases there were typical symptoms of Raynaud's disease: Asphyxia and local syncope of the finger, and more rarely of the toes, alternated with transitory edema in all parts of the body. In these patients we occasionally find other evidences of the want of stability of the vasomotor system: Transitory erythema, dermographia, cardiac palpitation, vertigo, rush of blood to the head, and tinnitus aurium; therefore the picture of *vasomotor ataxia* (Solis-Cohen, Herz) which was previously described.

The course of the disease is often exceedingly chronic. Relapses constantly occur, between which there may be free intervals of considerable duration.

The *prognosis* in regard to life is good, but, as already stated, life may be endangered by the laryngeal involvement and sometimes by pulmonary and cerebral symptoms.

As to the *pathogenesis* of the affection, I must state that it is impossible to explain the pale edema merely by the assumption of a change in the circulation of the blood, but that unquestionably another factor is operative. This must be sought either in the fact that a nervous influence upon the capillary cells increases the secretion of lymph or that other nervous influences produce a change in the composition of the walls of the vessels, so that these permit more of the transudate to pass through than under normal circumstances. It must also be borne in mind that the so-called internal symptoms of the affection cannot be attributed to edema of the affected mucous membranes. Therefore acute circumscribed transitory edema is not simply an angioneurosis; on the contrary, besides the undoubted disturbance of the circulation of the blood other factors are active which must be regarded as trophic or secretory disturbances. In transitory edema we are not always dealing with a disease *sui generis*, but it is sometimes merely of symptomatic value, as has already been stated. At all events, the view that Quincke's disease is in its nature a *vasomotor trophic* neurosis appears to me quite justifiable.

The *diagnosis* causes little difficulty inasmuch as we are not dealing with a modification of a similar pathologic picture; I need not discuss this point. The differentiation from hysterical edema occasionally causes some perplexity. The main point is this, that in hysterical edema a multiplicity of swellings is quite unusual. Moreover, in hysterical edema we are most likely to find local sensory symptoms.

Meige described under the name "*trophœdema chronicum hereditarium*" a chronic neuropathic edema which develops gradually, almost invariably affects the lower extremities bilaterally, and shows a marked tendency to appear in families. The dissimilarity to transitory edema is obvious on comparison. With a rare localization of the edema we can of course only make a certain diagnosis if, while under observation, typical cutaneous edema finally appears.

In *treatment* most value is to be attached to the regulation of the diet and to the general management of the neurotic condition. All articles of food which formerly proved injurious must of course be avoided. The regu-

lation of the bowels is most necessary, either by drugs (menthol and camphor are advised as intestinal antiseptics) or by spa treatment in Kissingen, etc. Strychnin, quinin, atropin and arsenic have all been praised for internal medication; I have several times used the latter with success. Electricity, massage, and hydriatic procedures deserve consideration. That tracheotomy is sometimes indicated I need scarcely mention. The gastrointestinal symptoms occasionally necessitate the use of morphin.

OCCUPATION NEUROSES

By R. CASSIRER, Berlin

In another article in this volume I have described a pathologic condition which I designated as *professional neuritis* (occupation paresis or professional paresis). We defined professional paresis as an affection due to continuous and uniform employment giving rise to damage of the peripheral nervous system in which there are also other clinical manifestations of neuritis. Overexertion, which may be primarily regarded as the cause of occupation paresis, may also produce other damage, with which we must concern ourselves somewhat more in detail.

WRITER'S CRAMP

The patient is a man, aged 21, a clerk by occupation. He first appeared for treatment in April, 1905; he came to us because, in the last few months, he was no longer able to write, after having been engaged in writing for the last 5 years. During the last year he had been writing about 10 or 11 hours daily. The disturbance appeared gradually, the writing became uneven, tremulous; the patient was no longer able to write fluently and became tired much sooner than formerly. In addition there were pains upon the inner surface of the hands and upon the back of the hand, and, finally, a spasmodic condition of the thumb appeared. This member was drawn away from the penholder, so that it fell out of the patient's hand. He attempted to continue writing, by holding the penholder between the 3d and 4th fingers.

Finally this had to be given up and since a few days he is no longer able to write at all. The disturbances in the use of the hand are entirely limited to the faculty of writing. In other operations, such as in eating, in carrying objects in the right hand, and even in piano playing there is no difficulty. Questioned in regard to other symptoms he reports that he is easily excited, somewhat nervous, and often suffers from headache. His father suffers from migraine but all the other members of the family (3) are well. He has had a curvature of the vertebral column since his 9th year, which is said not to be hereditary in the family. If we ask the patient to make an attempt at writing, he presses the penholder between the first 3 fingers, at first, almost in a normal manner. Shortly the penholder is held spasmodically, but it is still possible for the patient to write a few words although slowly and with uneven letters. Very soon the thumb or the third finger slips away, interrupting the writing. Each renewed attempt is rendered futile in the same manner. A minute examination of motility and sensation of the hand revealed no change from the normal. The muscles are neither atrophic, paretic, nor painful upon pressure. Nor are the nerve trunks especially tender upon pressure. There is no disturbance in the contact or pain sense. Briefly, the entire objective finding of the right hand as well as the remainder of the body, with the exception of a very pronounced kyphoscoliosis, is entirely negative. In conclusion I shall report the further progress of the case. For 9 months the patient was not permitted to write; his treatment consisted of galvanism, massage and especially resistance gymnastics. There has been some improvement; he is able to write a few lines, the handwriting being fairly good, but he is compelled to write slowly. There can be no thought of resuming his occupation for a long time.

The preceding history illustrates a typical case of *writer's cramp* (*graphospasm, mogigraphia*). The condition consists of spastic contractions in certain muscle groups of the hand, which appear only when certain coördinated movements are attempted, such as writing. Corresponding to this relationship Benedikt chose the term "*coördinative occupation spasm*" for this affection. Writer's cramp is the type of these coördinative occupation spasms. As the spasm is often of secondary importance in the malady and pain besides paralytic conditions appear from the onset the term "*coördination occupation neuroses*" was chosen for the entire group. The designation neurosis states at once, that according to our present knowledge, there is no underlying organic lesion of the nervous system. Charles Bell was the first to describe cases of this kind, Duchenne[1] adding greatly to our knowledge of the malady. The latter author called the condition "*function spasm*" and "*function paralysis*." He states that it is an affection that only appears when definite movements are practiced, being characterized partly by spasmodic, tonic or clonic, painful or painless contractions, partly by tremor and partly by paralysis. Here we encounter the various subvarieties of the malady, the knowledge of which we owe to Benedikt, Berger, Remak and Bernhardt. (Complete report of the literature will be found in Remak,[2] and Bernhardt.[3])

Regarding the various forms in which the affection appears, our case is an example of writer's cramp in the strictest sense, in which actual spastic conditions appeared in separate muscles and muscle groups. As is the case in the majority of instances our patient showed spasmodic flexion in the thumb and index finger rendering writing impossible. Other fingers may also be attacked by the same spasm. Thus the little finger may be drawn into the palm of the hand. An immoderate flexion of the hand may prevent the act of writing. In other cases spasmodic extension of the long extensors of the fingers, and spreading of the fingers, so that the pen slips out of the hand, may prevent writing. The hand may actually be raised from the paper by spasm of the supinator. Duchenne reports 2 cases, where there was a supinator movement in the hand as soon as an attempt was made to write so that the point of the pen was directed upward; the patients were unable to alter the spasmodic position of the hand. I have never seen spasm in the muscles of the shoulder in my cases; these are said to occur, but only in the later stages of the affection.

The spasmodic condition is not always so restricted and distinct as in the case just reported. Very often sensory irritative symptoms are present, either accompanying the spasm or appearing alone.

A police assistant, aged 21, complained of severe pain, which originated in the ball of the small finger, radiating distally and proximally. In addition there were spasms, the little finger being retracted in the hand, which held the pen firmly upon the writing paper. An attempt at writing which the patient made in my presence, proved the correctness of his description. It was impossible for him to write more

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[1] Duchenne, "L'Electrisation localisée," 2. Edition, p. 928.

[2] Remak, "Beschäftigungsneurose." "Eulenburg's Real-Encyklopädie," 1894, III. Aufl., Bd. III, p. 270.

[3] Bernhardt, "Die Erkrankung der peripherischen Nerven." I. Aufl., II. Theil, p. 180.

than a few words. The local condition in this case was also entirely negative. Motion and sensation are quite intact, there is nowhere sensitiveness of the nerves or muscles upon pressure, no thickening or other pathologic condition. The patient was an abnormally large man—nearly 6 feet—with poor constitution, his mother being of a nervous temperament and suffering from headache; in addition the patient complained of general neurasthenic symptoms. For some years he has been compelled to write for eight hours daily in his occupation besides having to do similar work at home.

These pains usually show no exact localization, they do not follow the course of a nerve, but radiate from the point of greatest pain in a diffuse manner both distally and proximally; the pain is present in the fingers, sometimes reaching the shoulder and the back. Pain upon pressure is not present in the majority of cases, but it is not uncommon to find one or more nerves abnormally sensitive upon pressure at one or the other point. I have already insisted upon the fact that, according to Oppenheim, this painfulness upon pressure is quite common in neuropathically predisposed persons, without any local affection being present. Objective, sensory disturbances, according to general experience, are not present; they do not belong to the symptoms of this or any other form of occupation neurosis: this is one of the points by which these clinical pictures differ from occupation neuritis.

Pain may be the only symptom of the disease—then we no longer refer to writer's cramp but to *writer's pain*: but symptomatologic transitional stages between both groups, which apparently have identical pathogenesis, give us the right to speak of "writer's pain." In many persons the pain which appears during writing and which soon prevents a continuance of the act is the only symptom; occasionally there is pain upon pressure over some of the nerve trunks. Remak and Bernhardt have shown that this pure, sensory, neuralgic form of occupation neurosis is even more common in piano players. The pain more often than the spasm is the cause of the disturbance in function. I must add that typewriters, as might be expected, show disturbances which are closely allied to those of piano players; here, also, the pain is the significant factor; but we shall return to this subject.

Vasomotor symptoms are usually not prominent; only in rare instances is there a report of local syncope or local asphyxia. In a case reported by Brissaud, Hallion et Meige,[1] which presented many unusual features, the acrocyanosis was a symptom of coördinative importance. More often the patients complain of acroparesthesia, of tingling and numbness in the finger tips. That in some of the other forms of occupation neurosis nervous symptoms of the kind just mentioned are very prominent and that in these instances the decision whether we are dealing with acroparesthesia, in the restricted sense, or occupation neurosis, is exceedingly difficult will be described later on.

In addition to the spastic and the neuralgic forms of writer's cramp there are also *tremor* and *paralytic varieties*. The tremor form is quite rare.

A court clerk, aged 48, presents a disturbance in writing characterized by tremor, which appears after the patient uses the pen for a short time. The patient states he has a sensation as if he grasped the penholder too firmly, almost spasmodically. The tremor is especially severe when the patient knows that he is being observed; this

[1] Brissaud, Hallion et Meige, "Acrocyanose et crampe des écrivains." *Arch. générale d. medéc.*, 1903, 37.

report requires careful attention. He further states that recently, at night, his fingers become numb. He also suffers from general nervous symptoms. The patient is well nourished and other objective conditions appear to be normal. The character of the handwriting coincides with the tremor: it is zig-zag, but the individual lines are regular, not elongated or interrupted.

As a further and final form of writer's cramp the *paralytic variety* may be mentioned. It is very difficult to separate this form precisely. True paralysis is not present; on the contrary, there is here as in the other form a sensation of fatigue which soon renders writing impossible; in addition to this there is pain; there is no tremor or spasm, "hand and forearm appear as if chilled, the pen rests upon the paper without being moved" (Bernhardt). Occasionally there is a direct impression of an inability to write which produces the difficulty as in the case reported by Brissaud, Hallion et Meige. Meige,[1] in another article, refers to "*aboulie motrice.*" At all events we are here very close to the symptomatologic limits of our theme.

The positive phenomena of the affection are exhausted by what has been stated: spasm of the muscles, in which, as has been very aptly remarked, not merely the muscles that are concerned in writing contract, but also those that should not contract are affected; tremor, paretic sensations of fatigue, and pain, eventually also a certain tenderness upon pressure of the nerves and muscles; in addition it must be stated that all these symptoms appear only while writing, and only during this function. Naturally there are a few exceptions to this last rule. There are other activities in which the finer movements are necessary, such as piano playing, sewing, etc., in which the same symptoms may be present, while coarser movements requiring greater power do not give rise to symptoms. These exceptions are even more frequent in other occupation neuroses than in true writer's cramp.

There are no exceptions to the rule that objective sensory disturbances, such as appear in neuritis, are constantly absent. A qualitative change of electric contractility such as partial DeR is foreign to the picture of graphospasm and in fact to all of the occupation neuroses. Slight quantitative changes of contractility are said to be present in rare instances, both as an increase and decrease of contractility, as well as an inversion of the normal formula, so that the anodal contraction is stronger than the cathodal. But this may be noted in the small muscles of the hand even under other conditions, for instance, when they are cold or cyanotic. The size of the muscles remains unchanged.

In addition to writer's cramp all three of the patients whose history has been given showed other general nervous symptoms which, to a greater or lesser extent, correspond to neurasthenia. This coincides with the general experience that the majority of cases of writer's cramp develop from neurasthenia. This neuropathic disposition may be congenital or acquired. A hereditary predisposition shows itself in the family appearance of the occupation neuroses. Gallard reported the cases of a notary, his mother and sister having also suffered from writer's cramp. One of my patients, a lad aged 15, attending school, had the sensory form of writer's cramp; his father, a piano teacher, had piano player's cramp.

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[1] Meige, "Cramps fontionelles et professionelles." *Traité de médeo.*, X, p. 344.

Among the deleterious factors that may affect the patient, all that give rise to neurasthenia may be present: primarily over-exertion must be mentioned. The work of a copyist is poorly paid, and as Seeligmüller aptly remarks, *not authors but copyists are attacked by writer's cramp.* The poor remuneration requires prolonged work, "over-time" and even in addition to this, very frequently, it does not even provide sufficient food; thus *over-exertion and under-nutrition* act together in the same individual. *Night work,* which is so common in these cases, is of especial import. In addition to writing there are also other occupations such as mental over-exertion, in preparing for examination, which are often of consequence in these conditions. After the malady has once developed, the fear of the social consequences (which are often quite serious) are a constant aggravating factor both to the writer's cramp and to the neurasthenia. The presence of other neuroses has also been noted: epilepsy, hysteria, tic convulsif, hemicrania, neuralgia. I saw a combination of graphospasm and *meralgia paræsthetica.* Oppenheim mentions an association with tabes and with poliomyelitis. It is, therefore, possible that organic diseases of the nervous system may prepare the ground for the development of the neurosis.

On the other hand chronic intoxication, first alcoholism and then nicotinism, etc., may be important factors.

Certain local changes appear to be of **etiologic importance** in some cases. Bernhardt reports the case of a policeman, aged 45, who for a long time had been occupied with writing; after having his right hand run over, he began to present tremor in the thumb and index finger while writing. In other cases injuries to the shoulder, the hand, the fingers, were the cause of the appearance of the affection. I have a patient with general well-developed neurasthenic symptoms, in whom a markedly developed Dupuytren's contracture was present in addition to the tremor form of writer's cramp. Indurations and thickening of the tendon sheaths have been regarded as the cause, as well as periosteal inflammation (periostitis of the external condyle of the humerus). Any of the changes may predispose to the development of the affection but they are hardly likely to develop it.

The neurosis develops gradually under the influence of writing as an occupation upon the prepared groundwork. In some persons the regular work of writing 6–8 hours is sufficient, in others the frequent over-time has the injurious influence; upon the whole only a minority of those occupied with penmanship are affected. The malady is not very common. There can be no doubt that the style of pen and penholder may be of some influence in the causation of the affection; of most unfavorable import is the marked right angle position of the finger which fixes the pen. Writing teachers may do much to prevent the development of the malady in teaching their pupils a free position in holding the pen, avoiding too thin penholders, preventing the use of too pointed pens. The posture while seated during writing is also of importance.

Although the development of graphospasm, upon the whole, is gradual, nevertheless in connection with exertion, psychical shock, etc., acute exacerbations may occur. Oppenheim reports a case in which the affection appeared in immediate connection with attendance upon a spiritualistic meeting.

The **diagnosis** presents no difficulties. Naturally difficulties in writing may occur under all possible varieties and circumstances; every localized paralysis, every spasm, every tremor, every change in sensation hinders writing; but the cause may be determined from the individual symptoms and careful examination, at the onset of an organic affection, will reveal the reason for the changes. In these instances, not alone the writing but every other function, in which delicate movements are necessary, will be disturbed. The differentiation from occupation neuritis will hardly present difficulties; in the other professional neuroses (*milker's spasm* and similar conditions) a careful examination for sensory and motor symptoms of absence of function and for qualitative electric changes will be sufficient for a correct diagnosis.

There is occasionally some doubt in deciding the question as to whether the writer's cramp originated from the neurasthenia or whether the condition is one of neurasthenia with the prominence of local symptoms. Thus a simple neurasthenic or hysterical tremor may cause difficulty in writing; the tremor may attack the right hand, which has borne the brunt of the exertion alone or most markedly, and finally the difficulty may be more marked in writing than in any of the other finer movements. A decision is all the more difficult in these cases for the reason that there are very intimate relations between these conditions. It must be stated that these transitional affections are even more common in the other varieties of occupation neurosis, such as the neurosis of typewriters, piano players, telegraph operators, etc.

Which of the varieties of writer's cramp is present is very easy to determine from the symptomatic criteria that have been described. In regard to the correct treatment it is necessary to determine not merely that the affection is one of writer's cramp, but to discover which muscles reveal immoderate tension, where pressure points are situated, and so forth.

The **prognosis** was formerly always regarded as unfavorable. Recent authors are, upon the whole, less pessimistic; from my experience I coincide with this latter view; I have frequently seen, especially in cases that were not too severe, great improvement even after work had been resumed. In very severe cases prolonged writing and especially that required by occupation is impossible, or if after a prolonged interval writing is resumed this must speedily be given up. Among those who, in order to resume writing, learn to write with the left hand, a large proportion suffer later on from similar conditions in the left hand. A better remedy for those who, on account of their social position, find it necessary to continue writing is to learn to use the typewriter. It appears that the disturbances that arise under these circumstances are, upon the whole, milder or at least not so tenacious as in writing with the pen; but the fact that our experience in this field is still limited indicates that we must be cautious in giving an opinion of this kind.

TYPIST'S CRAMP

In accordance with an entirely different muscular activity in writing upon a typewriter, the disturbances are necessarily of a different nature and resemble those that we meet with in *piano player's* and *telegrapher's cramp.*

In these forms, as it appears, the spastic phenomena are of a more secondary nature, the sensory-paralytic features dominating the scene.

Lady, aged 38, for many years has been using the typewriter. For a year she has been suffering from pain in the right hand, which radiates from the middle finger to the elbow; in addition there is an intense feeling of weakness in the right arm and often a sensation as if the fingers were stiff without there being actual spasmodic conditions. Her hands are very cold and, occasionally in the autumn, actual numbness is present in the fingers. While formerly—as is done by most persons—she used the right hand in writing, she now employs the left. In other uses to which the hand is put—carrying an umbrella, sewing—there is frequently a sensation of fatigue. She is easily excited, nervous and readily frightened. Examination revealed that at the tips of the first and second fingers pin pricks and contact with the brush were not distinctly perceived. But the skin at these points is also somewhat thicker. It is worthy of note that in writing short-hand and employing the pen there is but slight difficulty, but she never continues this for any length of time.

I have seen quite a number of cases of this kind. They always occurred in the female sex; I emphasize this because formerly (see Bernhardt, *l. c.* p. 187) importance was attached to the fact that writer's cramp appeared almost exclusively in men; this is naturally due only to social conditions. The persons affected are usually pale, anemic, underfed individuals with the usual symptoms of general nervousness: headache, poor sleep, hyper-irritability, and mental depression. These persons have, as a rule, been occupied in writing many hours daily for a long number of years (8 hours and longer upon the typewriter); in addition some of them, as the patient just referred to, have written with the pen, employing short-hand.

The affection commonly begins with pain in the finger tips; at other times there is paresthesia, a feeling of numbness, tingling, formication and cold. Later the pain radiates to the shoulder and the back of the neck. The limitation of function is especially marked if in addition to the paresthesia and the pain there is fatigue which compels the patient to cease his activity after a short time. The patient often complains of spasmodic sensations, but I have never met with actual spastic conditions; nevertheless it cannot be doubted that these may occur.

Objective sensory disturbances are sometimes present, such as a decrease of sensation in the finger tips. It must, however, be remembered that the skin is somewhat thicker at these points.

Occasionally there are abnormal vasomotor symptoms, such as local syncope in the fingers. In these cases the differentiation from acroparesthesia is exceedingly difficult. Where the vasomotor symptoms appear in immediate connection with the occupation and disappear when the occupation ceases, they may be regarded as a symptom of the occupation neurosis; there are, however, cases in which there can be no positive decision.

As the right hand is the member most in use, the symptoms are first noted there; as a rule, this member is the more skillful and better able to stand the strain. But the left hand is by no means exempt.

It has already been stated that very frequently in this variety there is great fatigue in the performance of other activities: pain and numbness of the hands in dressing, arranging the hair, carrying an umbrella, in sewing, in piano playing, etc.

There is great resemblance between writer's cramp and another occupation neurosis, *telegrapher's cramp*. This was first described by Onimus in 1874. Cronbach [1] a few years ago published a comprehensive study of the malady. I have seen a few cases of this kind. The affection usually occurs in men. Among the sensory symptoms there are pains of various kinds, dull, stabbing, pulling, boring. Further, there are sensations of cold, tingling, formication, hypersensitiveness and numbness, feelings of swelling and stiffness. Among the motor symptoms there are pareses of some of the fingers, fatigue of the entire hand, of the forearm as well as of the entire member. Spastic symptoms are not prominent. Cronbach has reported a case in which the extensor tendons of the fingers revealed contractions. He emphasized the occurrence, in his cases, of vasomotor (cold, heat) and of secretory disturbances (abnormal sweating, abnormal dryness). He also noted pain and numbness of the hands in his cases even when the patient was not employed in his occupation. Among the objective symptoms there are pressure points along the nerves, the importance of which I have already considered. In some instances there were also pressure points in some of the muscles; in a case of Bernhardt's, in the sheaths of the tendons. In some instances in addition to telegrapher's cramp there was also writer's cramp, or this appeared later. Cronbach was able to demonstrate that not merely Morse's but also Hughes's apparatus is capable of developing the neurosis; hence, it follows that the advice to patients to give up the Morse apparatus and employ the Hughes (as was formerly done) is absolutely valueless.

Another form of occupation neurosis is *piano player's cramp*. Here the activity of the muscles is similar to the previously mentioned forms, but the technic is very much more delicate and much more difficult, requiring a very much longer practice of a purely mechanical nature. Professionals are most commonly attacked, apparently oftenest during their student days. In addition to intense and extensive activity they present the evidences of somatic and psychic symptoms. The clinical symptoms need not be described in detail. Among them are pains which are most prominent in the forearm, thence radiating upward and downward, these being the most important. The localization may vary, on the one hand, according to the technic employed, and, on the other hand, according to individual circumstances (small hand, incomplete stretching of the fingers, insufficient power of individual muscle groups, etc.). Fatigue, which appears with abnormal rapidity, causes prolonged pauses; gradually fatigue, present even at the onset, renders playing impossible. In addition there are spasmodic conditions (extension of some of the fingers). Pain upon pressure is especially marked in those muscles which are implicated in the function.

CRAMPS CAUSED BY PLAYING OTHER MUSICAL INSTRUMENTS

The picture of the occupation neurosis in *violinists, cellists*, etc., is the same. In these the spasm may affect the hand that uses the bow or the left hand which comes in contact with the strings.

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[1] Cronbach, "Die Beschäftigungsneurose der Telegraphisten." *Archiv für Psychiatrie*, XXXVII, p. 243.

A cellist, aged 23, complained of pain in the last 3 fingers of the left hand, the pain having started in the middle finger. The pain resembled pin pricks and was most marked in the tips of the fingers; gradually a sensation of numbness and tingling appeared. The patient believed that there was some loss of sensation in the tips of the fingers but this could not be demonstrated by objective examination. He complained of abnormal dryness in these fingers, in spite of the fact that there appeared to be an unusual amount of sweating. The difficulties were entirely limited to his cello playing.

Remak has reported tenacious hyperesthesia of the *pulpa* of the left index finger in violin players; in a patient of Oppenheim's there was paresthesia upon every attempt to play the violin, which prevented the continuance of the playing, the patient having no other difficulty. Remak reported a case of *flute player's cramp* in an aged musician, the patient showing spastic phenomena in the left thumb and little finger.

Thus the professional use of any instrument may give rise to similar symptoms, which need not necessarily affect the upper extremity.

Oppenheim has reported an occupation spasm in the muscles of the lips in a *cornet player*; as soon as the patient attempted to play, spasm appeared in the orbicularis oris and the patient was unable to produce a tone. Whether spasm or paralysis was present could not be determined. Strümpell reported an occupation spasm of the tongue in a *clarinet player*; similar conditions have been reported in *trumpet players* by Düms, Kalmus and Stadler.

CRAMPS OF THE LOWER EXTREMITIES

Meige reports a case of *harp player's cramp,* due to using the pedal of the instrument. *Occupation spasms of the lower extremity* are otherwise quite rare; a patient of Aldrich's was compelled to use the right foot daily for a number of years upon a pedal which worked a drum and kettledrum.

Rigidity and stiffness, as well as fatigue and pain, appeared in the muscles thus employed. In workmen who are compelled to use pedals, lathe workers, knife grinders, sewing machine workers, similar conditions may develop. These cases must be carefully examined for the symptoms of occupation neuritis. In *professional dancers,* such occupation neuroses, affecting the lower extremity, are also found. I have often noted a pain in the heel which presented all of the signs of an occupation neurosis. In a man who was compelled to stand for several hours daily in the same position, there was pain in the heel, which was not present under any other circumstances, such as prolonged walking or mountain climbing; in shop girls I have observed similar conditions. No local changes could be observed. Discontinuing the occupation was always of favorable influence. Bernhardt[1] has reported similar cases.

OTHER FORMS OF OCCUPATION NEUROSIS

As many forms of occupation neurosis are met with as there are occupations. I must mention some of the most common.

A patient, a jeweler, who was compelled to hold a tool firmly in his hand,

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[1] Bernhardt, *l. c.*, p. 466.

suffered from numbness in the little finger and in the ball of the member; there were no spastic symptoms, but, on the other hand, there was slight tremor. Meige observed the same symptoms in an engraver; Stephan reports occupation neurosis in diamond cutters. The same symptoms are common in persons who fold paper as an occupation. One of my patients who held the paper firmly with the left hand while he used the folder with the right hand complained of pain in the left elbow joint, thence radiating to the neck and region of the ear; in the right hand there was paresthesia. As he formerly suffered from *neuralgia paraesthetica* this is interesting in a pathogenetic sense. In paper sorters I have seen paresthesia; others have reported this in addition to spastic symptoms in money counters.

Occupation neuroses have been reported in watchmakers. T. Cohn reported spasm of the orbicularis in a watchmaker; Oppenheim reports spasm of accommodation; similar conditions have been noted after the prolonged use of the microscope. A drill-ocular muscle spasm (spasmodic position of the eyes to the right upon rotation of the head to the right) is reported by Franjen. Oppenheim regards the *nystagmus of miners* as belonging to this category.

The *occupation spasms of seamstresses, tailors* and *cobblers* affect the small muscles of the hand. Here sometimes the question of *tetany* may arise. The objective signs of the latter affection, especially Trousseau's sign, and the characteristic increase of electric contractility, will permit a differentiation.

Oppenheim has reported 2 cases of *barber's spasm.* This consisted of spasmodic tension of the muscles of the hand and fingers, occurring in barbers upon any attempt at using the razor in shaving.

Painful and spasmodic conditions appear in the muscles of the upper arm in connection with the labor of *lock-smiths, black-smiths, tin-smiths* and *carpenters.* Duchenne reports the case of a tailor whose arm was rotated inward by contraction of the subscapularis; a fencing master had the rapier rotated inward, at the same time presenting extension of the forearm.

In the course of this article I have several times pointed out that it is necessary to discriminate between professional pareses and professional neuroses. There are, however, cases in which, in addition to spasmodic and neuralgic symptoms, atrophic paresis is added. It has been known for a long time that in milkers spasmodic symptoms appear (*milker's cramp*): there is painful spasm in the flexors of the hand and fingers upon any attempt at milking, sometimes associated with vasomotor, irritative symptoms. In the cases reported by Remak, Stephan, Bernhardt, and myself, there were, however, in addition, degenerative-neuritic symptoms. Remak insists upon the great importance of this fact in the consideration of some of the varieties of occupation spasm, as it lends support to the view that they originate from peripheral, irritative conditions and are maintained by these. Similar symptoms have been observed in *cigar-makers. Wood-sawer's cramp* also belongs to the occupation spasms.

B. Fränkel reports a professional neurosis of the voice (*mogiphonia*). In analogy with writer's cramp he differentiates three varieties, spastic, tremor, and painful fatigue. The voice refuses to act in the professional employment; the laryngoscopic examination reveals normal conditions.

PATHOGENESIS

The question of PATHOGENESIS has just been touched upon. A few words more are necessary: it is very questionable whether this is the same for all of the varieties. It has already been stated that peripheral causes may be at fault; but this is merely the case in the minority of instances. On the other hand, it is sometimes the impression of "not being able" in addition to disagreeable peripheral sensations which are the causes of the affection. Between these extremes there are transitional forms; but I shall not discuss this very difficult subject; the causes which have been given by Benedikt, of which there are numerous adherents, at the very least are not applicable to every case.

TREATMENT

A successful TREATMENT of all forms of occupation neurosis depends upon complete rest for a long time. This requirement, unfortunately, very often meets with insurmountable difficulties. The chances of lasting results are greatly decreased by this fact; but, nevertheless, treatment must be attempted. Different forms of the affection require a varied management. The cases in which the neurosis depends upon a general, neuropathic predisposition—and we have seen how often this is the case—require observation of this fact in the therapy: general exhaustion as well as irritability is to be combated by hydriatic measures, tonics, and a change of climate; we cannot enter upon a detailed description of these measures here.

In writer's cramp—and also in other neuroses with proper modification—it is necessary to note whether the manner in which the pen is held, the nature of the pen and penholder, are responsible for the origin of the malady. I have mentioned that stiffness in holding the pen is injurious; the same is true of very thin penholders and very pointed pens. All this is to be avoided or altered if necessary. In addition, definite exercises are necessary; these are very advantageous. Zabludowski [1] has given explicit advice on this subject as well as Konindjy,[2] who speaks of a "*rééducation de l'écriture.*" Zabludowski begins by having the patient make circular figures in the air, such as are employed in large initial letters. In milder cases exercises in writing may be begun at once upon paper, in which, at first, an apparatus, Nussbaum's bracelet or Zabludowski's penholder carrier, may be employed. This apparatus prevents the fingers from assuming the faulty position. Writing exercises, with the inverted hand, so that the volar surface of the hand is placed upward, is also advised; this gives the hand an entirely new base of support. These writing exercises should be practiced 3 times daily, the duration of each being 5 minutes. Konindjy emphasizes the necessity of writing slowly, many pauses being introduced, so that the pen may be dipped frequently into the ink. The movements of the arm necessary for this purpose causes the contraction of other muscles than those required in writing. In very severe cases, in which the pen can no longer be used, it is sometimes

[1] Zabludowski, "Ueber Schreiber- und Pianistenkrampf." *Volkmann's Samml. klin. Vortr.*, 1901.

[2] Konindjy, "La crampe professionelle." *Nouv. Icon. d. l. Salpêtr.*, 1905, p. 521.

possible to have these exercises practiced with a lead pencil, progress being attained in this manner.

Massage of the entire arm and hand may be used in addition to the exercises. This treatment is especially advisable in cases in which there are inflammatory changes. Zabludowski advises energetic vibration massage. I have attained excellent results with vibration massage, gradually increasing the intensity, special muscles in the arm and hand being treated separately. I have used this treatment in combination with resistance movements; this method is especially indicated in the occupation neurosis of typewriters. The same apparatus may be employed for resistance movements.

Electricity may be used according to general principles. The galvanic current is most often applied, the anode being placed upon the brachial plexus or upon the elective points of the different nerves of the arm. The strength of the current should be 4–6 ma. Galvanization of the cervical cord has also been advised. Most authors (Gowers, Bernhardt, Oppenheim and others) report but slight favorable results from electricity. The faradic current is applied when there is great muscular debility: strong faradic currents should, however, never be used. Faradic hand-baths have been employed with advantage (Laquer) as well as the static machine, arc light, and the incandescent light (Laquer).

Where local changes are present these should be relieved if possible without surgical intervention. Cutting the flexor and extensor tendons of the fingers, which was formerly practiced (Dieffenbach) is now quite properly rejected.

In those instances in which it was impossible for the patient to write with the previously mentioned apparatus, an attempt was occasionally made to use the left hand for the purpose; in some cases this has been successful for a number of years. Often, however, difficulties soon appear in the left hand which render writing impossible. The use of the typewriting machine is now urged. That this remedy may also fail in some cases was to be expected; in a case of Simpson's, writer's cramp also originated from the use of the type machine.

It cannot be doubted that suggestion plays an important rôle in the therapy of writer's cramp and the other occupation neuroses. That its influence will vary in the different forms is obvious.

The treatment of the other varieties of occupation neurosis may be deduced from what has been advised for writer's cramp. I have already called attention to some of the modifications.

Patience and perseverance on the part of the physician and patient and certain favorable circumstances in the social position of the sufferer are of the greatest advantage in procuring favorable results in the treatment of these maladies.

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